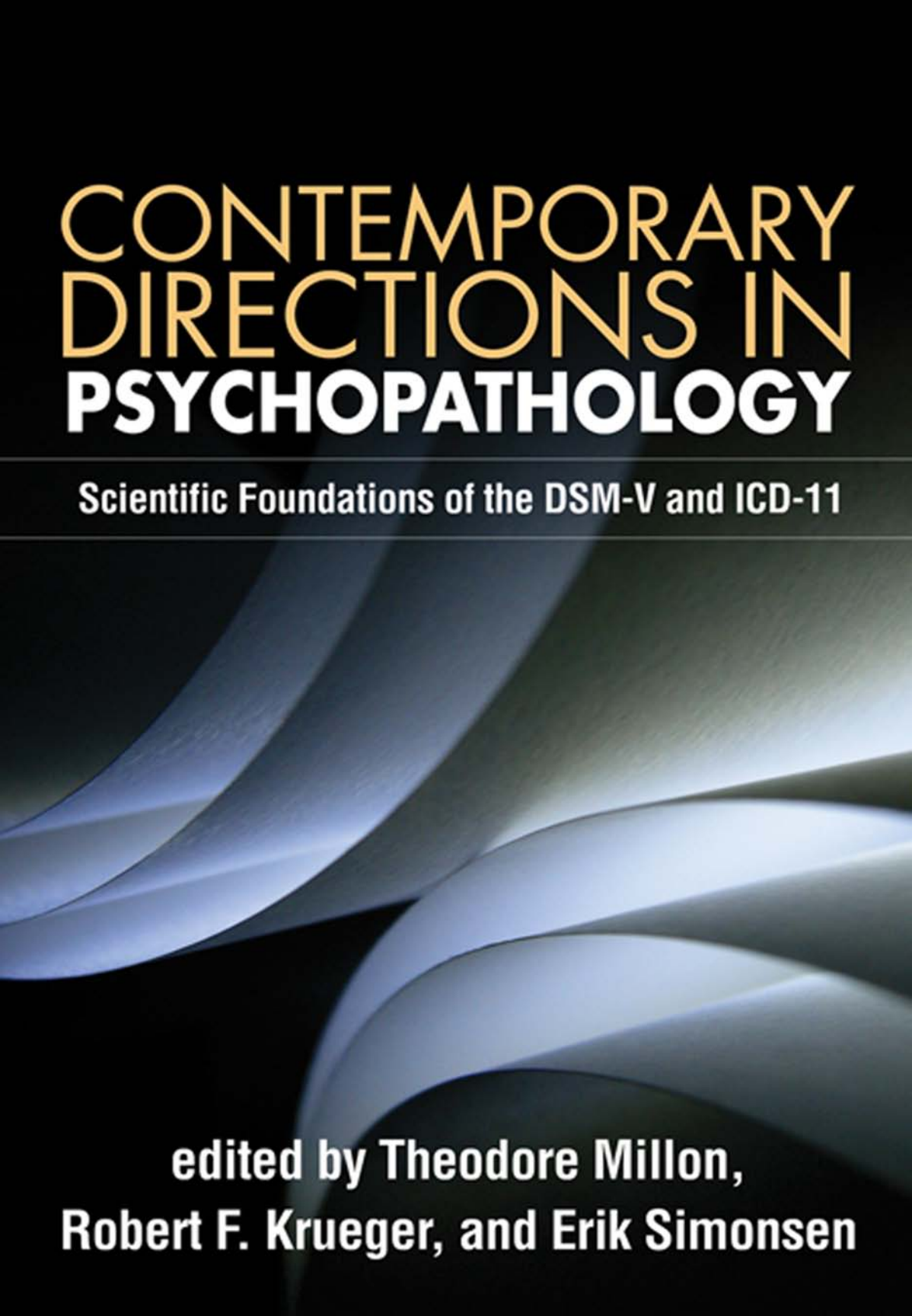
CONTEMPORARY DIRECTIONS IN PSYCHOPATHOLOGY

Scientific Foundations of the DSM-V and ICD-11



edited by Theodore Millon,
Robert F. Krueger, and Erik Simonsen

CONTEMPORARY DIRECTIONS IN PSYCHOPATHOLOGY

CONTEMPORARY DIRECTIONS IN **PSYCHOPATHOLOGY**

Scientific Foundations of the DSM-V and ICD-11

edited by
Theodore Millon
Robert F. Krueger Erik Simonsen

THE GUILFORD PRESS
New York London

A Division of Guilford Publications, Inc.
72 Spring Street, New York, NY 10012
www.guilford.com

Printed in the United States of America

This book is printed on acid-free paper.

Last digit is print number: 9 8 7 6 5 4 3 2 1

Library of Congress Cataloging-in-Publication Data

Contemporary directions in psychopathology : scientific foundations of the DSM-V and ICD-11 / edited by Theodore Millon, Robert F. Krueger, Erik Simonsen.
p. cm.
Includes bibliographical references and index.
ISBN 978-1-60623-532-4 (hbk.)
1. Psychology, Pathological. 2. Mental illness—Etiology. 3. Mental illness—Diagnosis. 4. Mental illness—Classification. I. Millon, Theodore. II. Krueger, Robert F. III. Simonsen, Erik, 1949–
RC454.C647 2010
616.89—dc22

2009028186

About the Editors

Theodore Millon, PhD, DSc, is Dean and Scientific Director of the Institute for Advanced Studies in Personology and Psychopathology. He was Founding Editor of the *Journal of Personality Disorders* and inaugural president of the International Society for the Study of Personality Disorders (ISSPD). Dr. Millon has held full professorial appointments at Harvard Medical School, the University of Illinois, and the University of Miami. A prolific author, he has written or edited more than 30 books on theory, assessment, and therapy, as well as more than 200 articles and book chapters. Dr. Millon is the recipient of the 2008 American Psychological Foundation Gold Medal for Life Achievement in the Application of Psychology.

Robert F. Krueger, PhD, is Professor of Psychology and Psychiatry at Washington University in St. Louis. His major research interests lie at the intersection of personality, psychopathology, psychometrics, and behavioral genetics. Dr. Krueger has received a number of awards, including the American Psychological Association's Distinguished Scientific Award for Early Career Contribution and the American Psychological Foundation's Theodore Millon Award for midcareer contributions to personality psychology.

Erik Simonsen, MD, is Director of Psychiatric Research in Region Zealand, Denmark; Director of the Institute of Personality Theory and Psychopathology; and Associate Research Professor at the University of Copenhagen. He has published widely on personality disorders, first-episode psychosis, personality assessment, outcome of psychotherapy, and psychiatric classification. Dr. Simonsen is past president of the ISSPD and a recipient of the ISSPD Award. He has also served as president of the Section on Personality Disorders of the World Psychiatric Association.

Contributors

Renato D. Alarcón, MD, MPH, Mood Disorders Unit and Department of Psychiatry, Mayo Clinic College of Medicine, Rochester, Minnesota

Steven R. H. Beach, PhD, Institute for Behavioral Research, University of Georgia, Athens, Georgia

Edward M. Bernat, PhD, Department of Psychology, Florida State University, Tallahassee, Florida

Roger K. Blashfield, PhD, Department of Psychology, Auburn University, Auburn, Alabama

Sidney J. Blatt, PhD, Department of Psychiatry, Yale School of Medicine, New Haven, Connecticut

Bekh Bradley, PhD, Trauma Recovery Program, Atlanta Veterans Affairs Medical Center, and Department of Psychiatry and Behavioral Sciences, Emory University, Atlanta, Georgia

Jessica Combs, BA, Department of Psychology, University of Kentucky, Lexington, Kentucky

Rina Dutta, MRCPsych, Institute of Psychiatry, Kings College London, London, United Kingdom

Nicholas R. Eaton, MA, Department of Psychology, Washington University in St. Louis, St. Louis, Missouri

Michael B. First, MD, Department of Psychiatry, Columbia University, New York, New York

Elizabeth Flanagan, PhD, Department of Psychiatry, Yale School of Medicine, New Haven, Connecticut

Joseph P. Gone, PhD, Department of Psychology, University of Michigan, Ann Arbor, Michigan

Seth Grossman, PsyD, Counseling and Psychological Services Center, Florida International University, Miami, Florida

William M. Grove, PhD, Department of Psychology, University of Minnesota, Minneapolis, Minnesota

Richard E. Heyman, PhD, Department of Psychology, Stony Brook University, State University of New York, Stony Brook, New York

Nadine J. Kaslow, PhD, Department of Psychiatry, Emory University School of Medicine, Atlanta, Georgia

Jared Keeley, MS, Department of Psychology, Auburn University, Auburn, Alabama

Kenneth S. Kendler, MD, Virginia Institute for Psychiatric and Behavioral Genetics, Virginia Commonwealth University, Richmond, Virginia

Laurence J. Kirmayer, MD, Division of Social and Transcultural Psychiatry, McGill University, and Institute of Community and Family Psychiatry, Jewish General Hospital, Montreal, Quebec, Canada

Helena Chmura Kraemer, PhD, Department of Psychiatry and Behavioral Sciences, Stanford University, Stanford, California; Department of Psychiatry, University of Pittsburgh, Pittsburgh, Pennsylvania

Robert F. Krueger, PhD, Departments of Psychology and Psychiatry, Washington University in St. Louis, St. Louis, Missouri

Mark F. Lenzenweger, PhD, Department of Psychology, State University of New York at Binghamton, Binghamton, New York

Mark R. Lukowitsky, MA, Department of Psychology, Pennsylvania State University, University Park, Pennsylvania

Patrick Luyten, PhD, Department of Psychology, University of Leuven, Leuven, Belgium

Mario Maj, MD, PhD, Department of Psychiatry, University of Naples, Naples, Italy

Paul E. Meehl, PhD (deceased), Department of Psychology, University of Minnesota, Minneapolis, Minnesota

Theodore Millon, PhD, DSc, Institute for Advanced Studies in Personology and Psychopathology, Port Jervis, New York

Robin M. Murray, MD, Institute of Psychiatry, Kings College London, London, United Kingdom

Kile M. Ortigo, MA, Department of Psychology, Emory University, Atlanta, Georgia

Joel Paris, MD, Department of Psychiatry, McGill University, and Institute of Community and Family Psychiatry, Jewish General Hospital, Montreal, Quebec, Canada

Christopher J. Patrick, PhD, Department of Psychology, Florida State University, Tallahassee, Florida

Aaron L. Pincus, PhD, Department of Psychology, Pennsylvania State University, University Park, Pennsylvania

Kristin Raley, MS, Department of Psychology, Auburn University, Auburn, Alabama

David Reiss, MD, Yale Child Study Center, Yale University, New Haven, Connecticut

Bryna Siegel, PhD, Department of Psychiatry, University of California, San Francisco, San Francisco, California

Erik Simonsen, MD, Psychiatric Research Unit, Region Zealand, and Department of Neuroscience and Pharmacology, University of Copenhagen, Copenhagen, Denmark

Andrew E. Skodol, MD, Sunbelt Collaborative and Department of Psychiatry, University of Arizona College of Medicine, Tucson, Arizona

Gregory T. Smith, PhD, Department of Psychology, University of Kentucky, Lexington, Kentucky

Susan C. South, PhD, Department of Psychological Sciences, Purdue University, West Lafayette, Indiana

Michael H. Stone, MD, Department of Psychiatry, Columbia University, and private practice, New York, New York

Robert Tringone, PhD, Counseling Center, St. John's University, Jamaica, New York

Scott I. Vrieze, BA, Department of Psychology, University of Minnesota, Minneapolis, Minnesota

Jerome C. Wakefield, PhD, DSW, Silver School of Social Work and Department of Psychiatry, New York University, New York, New York

Marianne Z. Wamboldt, MD, Department of Psychiatry, University of Colorado Denver School of Medicine, Aurora, Colorado

Drew Westen, PhD, Departments of Psychology and Psychiatry and Behavioral Sciences, Emory University, Atlanta, Georgia

Aidan G. C. Wright, MS, Department of Psychology, Pennsylvania State University, University Park, Pennsylvania

Peter Zachar, PhD, Department of Psychology, Auburn University at Montgomery, Montgomery, Alabama

Preface

The present book is similar in concept to a well-received volume that one of us (Theodore Millon) edited with Gerald L. Klerman of Harvard University in 1986; it is, however, an almost entirely new book, with only one chapter carried over from the earlier work. Gerry and I were colleagues at the Stanley Cobb Psychiatric Laboratories of Massachusetts General Hospital, as well as active participants in the development of DSM-III. Our aim in the earlier volume was to describe substantive and innovative advances since the publication of DSM-III in 1980, and to emphasize themes we believed should be considered in the forthcoming DSM-IV. We, the present editors, intend to do the same in this volume for DSM-IV(-TR) and for the forthcoming DSM-V and ICD-11.

Numerous changes in the character of psychopathology have begun taking place in the past several decades. Slow though progress has been, there are inexorable signs that the study of mental disorders has advanced beyond its earlier history as an oracular craft. No longer dependent on the intuitive artistry of brilliant clinicians and theoreticians who formulated dazzling but often unfalsifiable insights, psychopathology has acquired a solid footing in the empirical methodologies and quantitative techniques that characterize mature sciences. Although the term "psychopathology" was used in the past as synonymous with "descriptive symptomatology," it can now be justly employed to represent "the science of abnormal behavior and mental disorders." Its methods of study comfortably encompass both clinical *and* experimental procedures.

Among the many indices of continuing progress is the construction of psychometrically sound diagnostic tools that wed the quantitative and statistical precision typifying rigorous empirical disciplines with the salient and dynamic qualities characterizing the concerns of a clinical profession. Contributing to this precision is the introduction of comprehensive and comparable diagnostic criteria for each mental disorder—an advance that not only enhances the clarity of clinical communication, but strengthens the reliability of research, contributing thereby to the collection of reciprocal and cumulative data. Similarly, sophisticated multivari-

ate statistical methods now provide quantitative grounds for analyzing symptom patterns and constructing an orderly taxonomy.

Theoretical formulations have also begun to take on a more logical and orderly structure. Whereas earlier propositions were often presented in haphazard form, with circular derivations and ambiguous or conflicting empirical consequences, contemporary theorists began to specify explicit criteria for their concepts, as well as to spell out objective procedures and methods for testing their hypotheses. Moreover, theorists have become less doctrinaire in their positions than formerly; that is, they no longer act and write as religious disciples of "theological purity." A true "ecumenism" has emerged—an open-mindedness and sharing of views that are much more characteristic of disciplines with secure foundations. Thus erstwhile analysts have shed their former dogmatisms and have begun to incorporate findings such as those in the neurosciences and social psychology; similarly, once-diehard behaviorists have jettisoned their earlier biases and have integrated cognitive processes into their principles. On many levels and from several perspectives, the signs indicate consistently that psychopathology is becoming a full-fledged science.

It is our intent in this book to draw attention to innovations that constitute continuations of these directions. The volume is not intended to be a comprehensive textbook, but many of its chapters provide thoughtful pedagogic reviews and heuristic recommendations that may prove useful to the forthcoming DSM-V and ICD-11. In this latter regard, we very much favor current efforts to construct further rapprochements between the American Psychiatric Association's DSM and the World Health Organization's ICD. Work on the new editions of both manuals is well underway, and we believe that a successful accommodation will come from the combination of careful theoretical and conceptual analyses, and the parallel acquisition of empirical data from well-designed research. This work not only reflects the current state of psychopathology as a science, but should help identify the issues and methods that can foster this important reconciliation.

As noted, all but one of the 30 chapters in this volume is new. Only Paul E. Meehl's classic chapter on "diagnostic taxa" is a repeat from the earlier book; it is one that Mark F. Lenzenweger reflects on and thoroughly reviews. Notable in this edition are several chapters that bring to the forefront the role played by social context and culture in the roots of numerous mental disorders. The book begins with an extensive historical survey that leads up to contemporary thinking. Here are traced the contributions of theorists and researchers from ancient times (e.g., Zang Zhongjing, Alcmaeon, Aretaeus), the many fruitful ideas of 19th-century clinicians (e.g., Esquirol, Griesinger, Kahlbaum), and the work of more modern scholars (e.g., Kraepelin, Freud, Beck).

Classification matters are explored deeply in chapters that deal with philosophical issues underlying construct validity, syndromal comorbidity, and the clinical utility of categories versus dimensions. Innovative proposals are presented on such topics as the neuroscientific foundations of psychopathology and the use of evolutionary principles in articulating the development and composition of psychopathology.

It is our hope that this volume will contribute further to the long and fruitful collaboration between the disciplines of psychology and psychiatry. Psychopathology needs "all the help it can get" if it is to fulfill its promise as a science. The best minds are not to be found in one school of thought or in one mental health profession. Different perspectives not only contribute to "rounding out" important areas of content and technique, but help spark fresh insights and ideas. We three editors have found collaborative work to be both stimulating and rewarding; we hope that this book will serve not only as a model of cooperation between our fields, but as an invitation to biochemists, epidemiologists, psychometricians, geneticists, sociologists, and professionals in other disciplines to join us in similar enterprises.

In closing, we should like to express our appreciation to the book's contributors. Among them are numerous distinguished scholars, as well as well-respected and promising young investigators from psychology, psychiatry, philosophy, and sociology. In addition to being extremely pleased with the high quality and original thought that went into each chapter, we should note that both authorial and editorial royalties for this volume will be turned over to the World Health Organization, to provide it with additional resources to facilitate and expedite the development of ICD-11.

Theodore Millon
Robert F. Krueger
Erik Simonsen

Contents

PART I

HISTORICAL AND CULTURAL PERSPECTIVES

CHAPTER 1

A Précis of Psychopathological History

THEODORE MILLON
ERIK SIMONSEN

Before we and our contributors undertake a systematic analysis of current trends in psychopathology in this text, it may be useful to introduce the subjects with reference to its historical origins and evolution. Efforts to understand and resolve the problems that researchers studying mental disorders continue to face can be traced through many centuries in which solutions have taken unanticipated turns and have become enmeshed in obscure beliefs and entangled alliances, most of which have unfolded without the care and watchful eye of modern scientific thoughts and methods. Psychopathology remains today a relatively young science. We find that many techniques and theories of our time have long histories that connect current thinking to preexisting beliefs and systems of thought, many of which are intertwined in chance associations, primitive customs, and quasi-tribal quests. The path to the present is anything but a simple and straight line; it has come to its current state through an involvement in values and customs of which we may be only partly aware. Many are the product of historical accidents and erroneous beliefs that occurred centuries ago, when mysticism and charlatanism flourished.

The traditions of psychopathology today are not themselves tight systems of thought in the strict sense of scientific theories; they certainly are neither closed nor completed constructions of ideas that have been worked out in their final details. Rather, they are products of obscure lines of historical development—movements often subject to the confusions and misunderstandings of our remote past, when a disaffection with complexities typified life. Nevertheless, interest in ourselves, in our foibles as well as our achievements, has always been central to our human curiosity. The origins of interest in the workings of psychopathology were connected in their earliest form to studies of astronomy and spiritual unknowns. Even before any record of human thought had been drafted in written form, we humans were asking fundamental questions, such as why we behave, think, act, and feel as we do. Although primitive in their ideas, ancient people were always open to the tragic sources in their lives. Earliest answers, however, were invariably associated with metaphysical spirits and magical spells. Only slowly were more sophisticated and scientific ideas formulated.

It was not until the 6th century B.C. that the actions, thoughts, and feelings of humans

were attributed to natural forces—that is, to sources found within ourselves. Philosophers and scientists began to speculate intelligently about a wide range of psychological processes; many of their ideas turned out to be remarkably farsighted. Unfortunately, much of this early imaginative and empirical work was forgotten through the centuries. Time and again, it was then slowly stumbled upon and rediscovered by careful or serendipitous efforts. For example, John Locke in the 17th century described a clinical procedure for overcoming unusual fears; the procedure he described is not very different from the systematic desensitization method developed this past century by Joseph Wolpe. Similarly, Gustav Fechner, founder of psychophysics in the mid-19th century, recognized that the human brain is divided into two parallel hemispheres that are linked by a thin band of connecting fibers (what we now term the corpus callosum). According to Fechner's speculations, if the brain was subdivided, it would create two independent realms of consciousness—a speculation confirmed and elaborated in the latter part of this past century by Roger Sperry, in what has been referred to as "split-brain research."

Every historical period was dominated by certain beliefs that ultimately won out over previously existing conceptions while retaining elements of the old. As the study of mental science progressed, different and frequently insular traditions evolved to answer questions posed by earlier philosophers, physicians, and psychologists. Separate disciplines with specialized training procedures developed. Today divergent professional groups are involved in the study of the mind (e.g., the neuroscientifically oriented psychiatrists, with a clear-eyed focus on biological and physiological processes; the psychoanalytic psychiatrists, with an austere yet sensitive attention to unconscious or intrapsychic processes; the personological psychologists, with the tools and techniques for appraising, measuring, and integrating the mind; and the academic psychologists, with a penchant for empirically investigating the basic processes of behavior and cognition). Each group has studied the complex questions generated by mental disorders with a different focus and emphasis. Yet the central issues remain the same. By tracing the history of each of these and other conceptual traditions, we will learn how different modes of thought today have their roots in chance events, cultural ideologies, and accidental discoveries, as well as in brilliant and creative innovations.

From today's perspective, it seems likely that future developments in the field will reflect recent efforts to encompass and integrate biological, psychological, and sociocultural approaches. No longer will any single and restricted point of view be prominent; each approach will enrich all others as one component of a synergistic whole. Integrating the disparate parts of a clinical science—theory, nosology, diagnosis, and treatment—is the latest phase in the great chain of history that exhibits an evolution in mental science professions from ancient times to the new millennium. Intervening developments (both those that have been successful and those that have not) were genuine efforts to understand more fully who we are and why we behave the way we do. The challenge to know who we are is unending, owing to the complexity of human functioning. New concepts come to the fore each decade, and questions regarding established principles are constantly raised. Perhaps in this new century we will bridge the varied aspects of our poignant yet scientific understanding of psychopathology, as well as bring the diverse traditions of the past together to form a single, overarching synthesis.

Ancient History

Primitive humans and ancient civilizations alike viewed the unusual and strange within a magical and mythological frame of reference. Behavior that could not be understood was thought to be controlled by animistic spirits. Although both good and evil spirits were conjectured, the bizarre and often frightening behavior of persons with mental disorders led to a prevailing belief that demon spirits must inhabit them. The possession of evil spirits was viewed as a punishment for failing to obey the teachings of the gods and priests. Fears that demons might spread to afflict others often led to cruel and barbaric tortures. These primitive "therapies" of shock, starvation, and surgery have parallels in recent history, although the ancients based them on the more grossly naive conception of demonology.

What has been called the sacred approach in primitive times may be differentiated into three phases, according to Roccatagliata (1973): "animistic," "mythological," and "demonological." These divergent paradigms shared one point of view: that psychopathology was the expression of transcendent magical action brought about by external forces. The animistic model was based on pre-logical and emotional reasoning derived from the deep connection between primitive beings and the mysterious forces of nature. From this viewpoint, events happened because the world was peopled by animated entities driven by obscure and ineffable forces that acted upon human minds and souls. The second phase, that characterized by mythological beliefs, transformed the animistic conception so that indistinct and indefinable forces were materialized into myths. Every fact of life was imbued with the powers of a particular entity; every symptom of disorder was thought to be caused by a deity who could, if appropriately implored, benevolently cure it. In the third, or demonological phase, the transcendent mythological deities were placed into a formal theological system such as the Judeo-Christian. In line with this latter phase, two competing forces struggled for superiority: one creative and positive, represented by a good father or God; the other destructive and negative, represented by the willful negation of good in the form of demonic forces of evil. These three conceptions followed each other historically, but they did overlap, with elements of one appearing in the others at times.

Many aspects of prehistoric life could not be understood; magic and supernatural concepts helped early humans make sense out of the unfathomable and unpredictable. Weighted with life's painful realities and burdensome responsibilities, these beliefs gave an order and a pseudo-logic to fears of the unknown—a repository of unfalsifiable assumptions in which the supernatural filled in answers for that which could not be understood. Ultimately, supernaturalism became the dominant world view in which the perplexing experiences of life could be objectified and comprehended. Priests and wizards became powerful, capitalizing on the fears and peculiarities of the populace to undo spells, "heal" those with physical illnesses, and "purify" those with mental distress. Within this worldview, eccentric or irrational individuals were assuredly touched by spirits who possessed superhuman powers to induce psychic pathology. Almost all groups permitted healing to fall into the hands of priests and magicians—a situation that still exists today in some societies. Living in a world populated with imaginary beings, these spiritual forces could often calm the worst human anxieties and expunge the ever-present terrors of life. Despite extensive archeological analyses, however, our knowledge of primitive times is no more than fragmentary. Nevertheless, we may assume that primitive humans saw a world populated with spirits that were essentially illusions created by their own state of anguish and perplexity.

India, Babylonia, and China

Many contributions of the early Hindus are associated with the name of ***Susruta***, who lived 100 years before Hippocrates. His works followed the traditional beliefs of his day regarding possible demonic possession. However, Susruta suggested that the passions and strong emotions of those mental disorders might also bring about certain physical ailments calling for psychological help (Bhugra, 1992). Anticipating the significance of temperament or innate dispositions, Hindu medicine proposed that three such inclinations existed: wise and enlightened goodness, with its seat in the brain; impetuous passions, the sources of the pleasure and pain qualities, with their seat in the chest; and the blind crudity of ignorance, the basis of more animalistic instincts, its seat located in the abdomen.

A concern with mental health has long been a part of Indian cultures, which evolved various ways of attempting to understand and negotiate mental disorder and psychological problems. Indians have long been involved in constructing explanatory techniques. In the first formal system of medicine in India, *Ayurveda* (*The Book of Life*), physical and mental illnesses were not clearly demarcated. *Caraka Samhita* dealt with medical diagnoses and management possibly dating from 600 B.C. and was the foremost text of the ancient Indian medical system. *Caraka* defined *ayu* (life) as a state consisting of *shareera* (body), *indriya* (sens-

es), *satva* (psyche), and *atma* (soul). Soul could not be destroyed, and it underwent reincarnation. The mind was responsible for cognition, and it directed the senses, controlled the self, reasoned, and deliberated. The equilibrium between the self and mind was viewed as paramount to good health. *Caraka* used the general term *doshas* for the body fluids or humors, *vata*, *pitta*, and *kapha*. The theory of *doshas* may have developed independently of the Greek humoral theory, or possibly the Hindu system may have traveled to Greece. Types of food were thought to influence the mind, personality characteristics, and the interactions among the three *doshas*. Different personality types were described in detail as leading to mental illness, through either unwholesome diet or moral transgressions. In the Hindu system, mental disorders were seen as largely metaphysical, but different appearances of mental disorders (like *unmada*, insanity) were described as resulting from heredity, imbalanced *doshas*, temperament, inappropriate diet, and metapsychological factors. *Caraka* also contained many descriptions of possession states regarded as arising from supernatural agents—a belief that is still apparent in many parts of highly religious Indian society. Religious connotations and references to spiritual enlightenments were only challenged in the early 19th century by the emerging Western-science-based medicine introduced by British rulers. In India, colonial medical institutions became brick-and-mortar symbols of Western intellectual and moral power, with European doctors even being taken as the sole excuse for empire. Indian magical practices and religious customs have been marginalized to some extent, but a variety of shamans—whose therapeutic efforts combine classical Indian alchemy, medicine, magic, and astrology with beliefs and practices from folk and popular traditions—are still present.

In the Middle East was the ancient civilization of Babylonia; it was not only a vast geographical expanse, but the foundation of philosophical thought for most nations in the Mediterranean region. In fact, many of the traditions discussed among the Greeks and Romans can be traced to ideas generated initially in the Babylonian empire. Babylonians were oriented toward astronomical events; superstitions regarding the stars produced many gods, a result largely of their intellectual leaders' fertile imaginations. Help from the gods was often sought through magical rites, incantations, prayers, and the special powers of those who were physicians or priests. The Babylonians assigned a demon to each disease; insanity, for example was caused by the demon Idta. Each was to be exorcised through special medicines (primarily herbs and plants), confessions, and other methods to help restore a balance between conflicting supernatural forces. As the Babylonians saw it, invariable tensions existed among the different gods—but, more importantly, between a more or less rational, as opposed to a superstitious, explanation of psychic ailments.

The first medical book in China, *Neijing* (*The Canon of Internal Medicine*), was compiled between 300 B.C. and 100 B.C. Organic syndromes, like epileptic seizures (*dian*) and delirium-like states, were also described, but with no clear distinction from the concepts of insanity and psychosis (*kuang*). The primary causes of psychiatric illness were suggested to be vicious air, abnormal weather, and emotional stress. The famous doctor ***Zang Zhongjing***, the Hippocrates of China, introduced other concepts and syndromes, such as febrile delirium, globus hystericus, and puerperal psychosis, in his *Jinkuiyaolue* (*A Sketchbook in a Golden Box*). Chinese medicine has tended to explain pathology change by means of philosophical concepts, and this framework has undergone little change. It includes the notions of the complementary *yin* and *yang*; the five elements, gold, wood, water, fire, and earth; and the principle of *Tao* (i.e., the way), which has been considered as the ultimate regulator of the universe and the most desirable state of well-being and longevity achieved by integrating the individual self into the realm of nature. These ontological principles were described in *The Yellow Emperor's Classic of Internal Medicine* some 20 centuries ago (Liu, 1981). Different personality types were portrayed as resulting from combinations of the five elements (e.g., the fiery type, the earthy type, the golden type, and the watery type). Phenomena occurring inside human beings were understood in terms of phenomena occurring outside in nature. Chinese medicine later became organ-oriented; that is, every visceral organ was believed to have charge of

a specific function. The heart was thought to house the mind, the liver to control the spiritual soul, the lung the animal soul, the spleen ideas and intelligence, and the kidney vitality and will. No attention was paid to the brain! For a long time psychiatric symptoms were interspersed with those of physical disease. The mind–body dichotomy was not a central theme. Mood disturbances and psychiatric symptoms attributed to menstrual irregularities tended to be expressed in somatic terms. In ***Chin-Yue's*** *Medical Book*, the Chinese word for "depression" literally meant "stagnation," implying obstruction of vital air circulation in the body. Case vignettes of patients with "deceiving sickness" (i.e., hysterical neurosis) were presented in the same book explaining symptom formation in people trapped in very difficult situations. In a similar way, sexual impotence was explained by excessive worry. In summary, psychiatric concepts of mental illness in China have undergone basically the same sequence as in the West: supernatural, natural, somatic, and psychological stages. However, Chinese medicine has been relatively less influenced by religious thoughts compared than early European medicine was; patients in Europe in the Middle Ages were declared by priests to be bewitched and were punished. Acupuncture, traditional Chinese medicine, folk herbs, and psychotherapy have been the most commonly used treatment approaches in China.

Egypt, Greece, and Rome

In Egypt, as in other early civilizations, there is evidence that the heart was thought to be the center of mental activity. Egyptians also had difficulty in separating prevailing supernatural beliefs from beliefs about things that could be observed and modified in nature. Astronomical phenomena were the primary objects of worship. "Natural" qualities were usually turned aside in favor of the mystical powers of the gods. Over the course of a century or two, Egyptian philosophers and physicians began studying the brain, ultimately recognizing it as the primary source of mental activity. Egyptians recognized that emotional disorders could be described in line with ideas proposed by the Greeks. Thus the set of disturbances the Greeks termed "hysteria" (using their word for "uterus") was caused, as the Egyptians saw it, by a wandering uterus that had drifted from its normal resting location; the task of the physician was to bring the uterus back to its normal setting. This explanation for hysteria continued until the late Middle Ages.

In the earliest periods of Greek civilization, insanity was considered a divine punishment, a sign of guilt for minor or major transgressions. Therapy sought to combat madness by various expiatory rites that removed impurities, the causes of psychic disorders. Priests mediated an ill person's prayers to the gods so as to assure his or her cure. Thus, with divine help, the person's heart could be purified of its evil. Albeit slowly, Greek scholars realized that little of a rational nature characterized their way of thinking about mental pathology. To them, external but unseen agents could no longer serve as a logical basis for a genuine understanding of mentally troublesome phenomena. A fundamental shift began to take place, not merely in the manner in which different types of mental disorders might be described, but in the basis for thinking about ways to alter these aberrant behaviors. In order to "treat" mental disorders, the Greeks began to recognize the necessity of understanding how and why mental disorders were expressed in the natural world; only then could they successfully deal therapeutically with the tangible symptoms of everyday mental life. Instead of leaving the treatment of mental disorders to the supernatural and mystical, they began to develop a more concretely oriented perspective. This transition was led by a number of imaginative thinkers in the 5th and 6th centuries B.C.

A central intellectual effort of Greek philosophers was the desire to reduce the vastness of the universe to its fundamental elements. Most proposed that complexities could be degraded to one element—be it water, air, or fire. Their task was to identify the unit of which all aspects of the universe were composed. Among the first philosopher-scientists to tackle this task was ***Thales*** (652–588 B.C.). What little we know of Thales comes largely from the writings of later Greek philosophers, notably Aristotle, Plato, and the historian Herodotus. This nimble-witted Greek proposed that the fundamental unit of the universe was a tangible and identifiable substance, water.

Though Thales was not the prime forerunner of a modern understanding of mental processes, he was a radical thinker who redirected attention away from mysticism, recognizing that psychic disorders were natural events that should be approached from a scientific perspective. As a pivotal figure in his time, he ushered in an alternative to earlier supernatural beliefs. Equally significant was Thales's view that efforts should be made to uncover underlying principles on which overt phenomena were based. Oriented toward finding these principles in physical studies and "geometric proportions," he turned to "magnetic" phenomena, convinced that the essential element of all life was its animating properties. To Thales, action and movement, based on balanced or disarrayed magnetic forces, was what distinguished human frailty. In this belief, he further derogated the view that external supernatural forces intruded on the psyche; rather, the source of pathology was inherent within persons themselves.

Paralleling the views of Thales, ***Pythagoras*** (582–510 B.C.) reasserted the importance of identifying the underlying scientific principles that might account for all forms of behavior. He differed from Thales in that he retrogressively preferred to use ethics and religion as the basis for deriving his scientific principles. More progressively, however, he was the first philosopher to claim that the brain was the organ of the human intellect, as well as the source of mental disturbances. He adopted an early notion of biological humors (i.e., naturally occurring bodily liquids), as well as positing the concept of emotional temperament to aid in decoding the origins of aberrant passions and behavior. The mathematical principles of balance and ratio served to account for variations in human characterological styles (e.g., degrees of moisture or dryness, the proportion of cold or hot, etc.). Balances and imbalances among humoral fundamentals would account for whether health or disease was present. Possessing a deep regard for his "universal principles," Pythagoras applied his ideas to numerous human, ethical, and religious phenomena. Though he believed in immortality and the transmigration of souls, this did not deter him from making a serious effort to articulate the inner "equilibrium" of human anatomy and health.

Pythagoras considered mental life as reflecting a harmony between antithetical forces: good–bad, love–hate, singular–plural, limited–unlimited, and so on. Life was regulated according to his conception of opposing rhythmic movements (e.g., sleep–wakefulness, inspiring–expiring). Mental disorders reflected a disequilibrium of these basic harmonies, producing symptoms of psychic impairment. To him, the soul could rise or descend from and to the body. The more the soul was healthy, in balance, and without psychic symptoms, the more it resembled solar energy. Pythagoras spoke of the soul as composed of three parts: reason, which reflected truth; intelligence, which synthesized sensory perceptions; and impulse, which derived from bodily energies. The rational part of the soul was centered in the brain; the irrational one, in the heart. Incidentally, Pythagoras coined the term "philosophy" by putting together the words *philo*, meaning "love," and *sophia*, meaning "wisdom."

Ostensibly through his father, Apollo, ***Aesculapius*** (ca. 550 B.C.) gained his understanding of the nature of mental disorders through the divination of dreams, which he then transmitted to his sons, Machaon and Podaleirius. A series of followers, called Aesculapians, established long-enduring "medical temples" and a distinguished cult. It is unclear historically whether Aesculapius actually existed or whether his ideas should properly have been attributed to Pythagoras. As the Aesculapian cult spread throughout the Greek empire, numerous temples were erected in the main cities of the Mediterranean basin, including Rome in 300 B.C.

What may be best known about Aesculapiad temples today is the *symbol* of medical knowledge they employed: a serpent wrapped around a rod. Medicine gradually evolved into a branch of philosophy in the 6th and 7th centuries B.C. No one of that early period achieved the mythic stature of Aesculapius, however—the presumed founder of temple-based hospitals designed to execute the healing traditions in which he believed, notably a rest from life's stressors and opportunities for positive mental growth. Located in peaceful and attractive settings, these temples were established to encourage patients to believe that there were good reasons to want to recover. Included

among the temples' treatment techniques were a balanced diet, a daily massage, quiet sleep, priestly suggestions, and warm baths, all of which were thought to comfort and soothe patients.

Also of value during this early period was the work of ***Alcmaeon*** (557–491 B.C.), possibly a son or favorite student of Pythagoras, carried out in the 5th century B.C. Alcmaeon became a philosopher-physiologist who asserted that the central nervous system was the physical source of mental activity, and that cerebral metabolism was based on the stability of "the humoral fluxes"; if these fluxes were imbalanced or unstable, they would create shifts in cerebral tissue functioning, leading then to various mental disorders. Metabolic fluxes were caused by a disequilibrium between the nervous system's qualities of dry–moist and hot–cold.

Most notable were Alcmaeon's efforts to track the sensory nerves as they ascended to the brain. He articulated, as perhaps no one else before him had done, the structural anatomy of the body through methods of careful dissection. No less significant was his conviction that the brain, rather than the heart, was the organ of thought. As Aesculapius reportedly did, he also anticipated the work of Empedocles and Hippocrates, in that he believed that health called for a balance among the essential components of life—coolness versus warmth, wetness versus dryness, and so on. The notion of fundamental elements in balance became a central theme in the work of Aesculapius and Alcmaeon; it also served to guide the views of their disciples. Alcmaeon's "biological model" based on the concept of metabolic harmony, called "isonomy," took the place of Greek's early mythological theology and was an extension of the growing secular and democratic spirit of Greek's 6th-century B.C. culture.

Empedocles (495–435 B.C.) adopted the homeostatic model generated in the work of Pythagoras, Aesculapius, and Alcmaeon. Most significant was his proposal that the basic elements of life (fire, earth, air, and water) interacted with two other "principles" (love vs. strife). Empedocles stressed that a balance among the four elements could be complicated by the fact that they might combine in either a complementary or a counteractive way. Love and strife represented human expressions of more elementary magnetic processes such as attraction and repulsion. All of the elements/humors could be combined, but Empedocles wondered what the consequences would be if they were organized in different ways. He set out to weave the several threads of his theory and concluded that the force of attraction (love) would be likely to bring forth a harmonic unity, whereas repulsion (strife) would set the stage for a personal breakdown or social disintegration.

To Empedocles, blood was a perfect representation of an equal mix of water, earth, air, and fire. He therefore suggested that persons with problematic temperaments and mental disorders would exhibit imbalances within their blood. Among his other contributions, Empedocles posited a rudimentary model of an evolutionary theory, anticipating Darwin's by 2,000 years. As he phrased it, "creatures that survive are those whose blood elements are accidentally compounded in a suitable way," whereas a problematic compounding will produce "creatures that will perish and die." To him, nature created a wide variety of healthful and perishing blood configurations—that is, different ways in which the four elements combined.

Some philosophers disagreed with the notion that the universe was composed of a simple and permanent element. ***Heraclitus*** (530–470 B.C.), for example, proposed that all nature was made up of fire. He asserted, however, that the universe was composed of no lasting substance—nothing stable, solid, or enduring. All real and tangible things would inevitably vanish, change their form, even become their very opposites.

In a similar manner, ***Anaxagoras*** (500–428 B.C.) asserted that a reduction to the basic elements could not explain the universe. He differed from Heraclitus in that he did not believe the universe lacked an enduring substance. He asserted that an endless number of qualitatively different elements existed, and that the organization or arrangement of these diverse elements was central to the structure of the universe. Anaxagoras's belief that the character of these constituents could not be explained except through the action of human thought was novel—a view similar to one asserted many centuries later by the phenomenologists and the gestaltists, who claimed that the structure of objective matter was largely in the interpretive eye of the perceiver.

Later the philosopher ***Democritus*** (460–362 B.C.), following ***Leucippus*** (ca. 445 B.C.), proposed that the universe was made of variously shaped atoms—small particles of matter in constant motion, differing in size and form, but always moving and combining into the many complex components that comprise the universe as we know it. This innovative speculation endures to the present time. Extending the theme proposed a century earlier by Anaxagoras, Democritus stressed the view that all truths were relative and subjective. As noted, he asserted that matter was composed of numerous invisible particles called atoms. Each atom was composed of different shapes that combined and were linked in numerous ways; again, although this idea was based on pure speculation, it was highly innovative and is regarded as essentially correct to this day. The physical thesis of contemporary times known as the Heisenberg principle also has its origins in Democritus's speculation.

A contemporary of Democritus, born the same year, became the great philosopher-physician who set the groundwork for sophisticated clinical medicine for the ensuing centuries. The fertility of this wondrous period of Grecian thought cannot be overestimated, ranging from the brilliant ideas of Democritus and Aristotle to the creative foundations of scientific medicine by Hippocrates.

Hippocrates (460–367 B.C.; see Figure 1.1) was born on the island of Cos, the center of an ancient medical school. He was the son of an Aesculapian priest, from whom he acquired his first medical lessons and whose philosophy he would follow in his own future therapeutic efforts. In the work of Hippocrates—the inheritor of his father's tradition and the humoral concepts of Pythagoras and Empedocles—mental disorders progressed from the magical and mythical realm, and the demonological and superstitious therapeutic approaches of an earlier era, to one of careful clinical observation and inductive theorizing. He synthesized the practical and sympathetic elements of the Aesculapian cult with the more "biological" proposals of Pythagoras, blending these elements to elevate mental processes and disequilibria into a clinical science.

Thus in the 5th century B.C., truly radical advances were made to supplant the superstitions of temple medicine. The astuteness and prodigious work of Hippocrates highlighted the naturalistic view that the source of all disorders, mental and physical alike, should be sought within the patient and not within spiritual phenomena. For example, the introductory notes to the Hippocratic book on epilepsy state:

> It seems to me to be no more divine and no more sacred than other diseases, but like other affections, it springs from natural causes. . . . Those who first connected this illness with demons and described it as sacred seem to me no different from the conjurers, purificators, mountebanks and charlatans of our day. Such persons are merely concealing, under the cloak of godliness, their perplexity and their inability to afford any assistance. . . . It is not a god which injures the body, but disease.

As a number of his progenitors had done, Hippocrates emphasized that the brain was the primary center of thought, intelligence, and emotions. It is only from within the brain, he asserted, that pleasures and joys and laughter arise, as well as sorrows, griefs, and tears. It is, he went on to say, this very same source that makes us mad or delirious, inspires us with dread and fear, and brings sleeplessness, inopportune mistakes, aimless anxieties, absentmindedness, and other acts contrary to the person's habitual ways. All of these stem from the brain when it is not

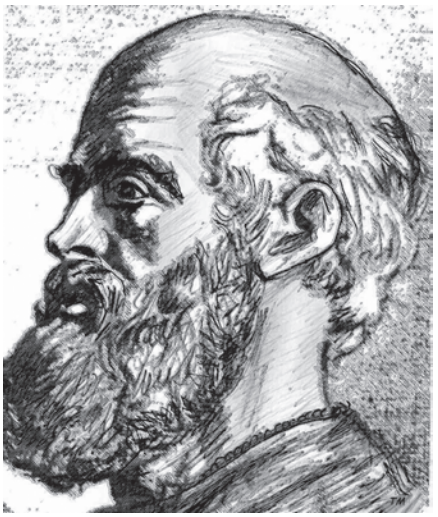


FIGURE 1.1. Hippocrates.

healthy (i.e., as when an imbalance exists between hot and cold or moist and dry).

Hippocrates's approach was essentially empirical, despite the growing eminence of philosophical thought that characterized his time. He was a practical biologist stressing the role of bodily humors and focusing on the use of physical treatments (notably diet, massage, music, and remedies promoting sleep and rest) rather than philosophical ones. Central to the medical practices of Hippocrates and his followers was the crucial role given keen observation and fact gathering. Contrary to the work of Plato, who relied on abstract hypotheses and so-called self-evident truths, Hippocrates focused his attention on observable symptoms, their treatments, and their eventual outcomes. In this regard, Hippocrates modeled Aristotle's empirical orientation, emphasizing facts rather than abstractions.

As were a number of his forebears, Hippocrates was convinced that dreams could serve as indicators of health or illness. Mental pathology stemmed from a disparity between the content of dreams and that which existed in reality. Dream symbolism, as regarded by Hippocrates, led him to anticipate later hypotheses concerning the operation of "unconscious forces."

Hippocrates also established the tradition of carefully recording personal case history, detailing the course and outcome of the disorders he observed. These histories provide surprisingly accurate descriptions of such varied disorders as depression, phobias, convulsions, and migraine. With his associates at the Cos College of Medicine in Athens, he provided a logic for differentiating among various mental ailments—not only those we now label the DSM-IV-TR Axis I syndromes, but also the Axis II personality types, the latter of which were construed as abnormalities of temperament. Temperament was associated with the four-humors model, which transformed earth, fire, water, and air into their parallel bodily elements. Individuals were characterized in terms of which one of the four elements predominated. Among other clinical syndromes differentiated were delirium, phobia, hysteria, and mania. Lacking precise observations of bodily structure, and prevented by taboo from performing dissections, Hippocratic physicians proposed hypothetical explanations of disease. They adhered closely, however, to the first nonsupernatural schema that specified temperament dimensions in accord with the doctrine of bodily humors. Interestingly, history has come full circle, in that much of contemporary psychiatry continues to seek answers with reference to inner biochemical and endocrinological processes.

Hippocrates identified four basic temperaments: the "choleric," the "melancholic," the "sanguine," and the "phlegmatic." These corresponded, respectively, to excesses in yellow bile, black bile, blood, and phlegm. As elaborated by a Roman, Galen, centuries later, the choleric temperament was associated with a tendency toward irascibility; the sanguine temperament prompted an individual toward optimism; the melancholic temperament was characterized by an inclination toward sadness; and the phlegmatic temperament was conceived as an apathetic disposition. Although the doctrine of humors has long been abandoned, giving way to studies on topics such as neurohormone chemistry, its archaic terminology still persists in contemporary expressions such as persons being "sanguine" or "good-humored."

Hippocrates and his Cos associates were among the first to stress the need for a relationship between diagnosis and treatment. The mere description of a clinical disturbance was not sufficient for them, unless it provided a clear indication of the course that therapy should follow. Indeed, Hippocrates anticipated that much effort may be wasted in specifying diagnosis, unless followed by a consideration of its utility for therapeutic decisions. Although naive in conception and execution, Hippocrates's approach to therapy followed logically from his view that disorders were of natural origin. To supplant the prevalent practices of exorcism and punishment, he recommended such varied prescriptions as exercise, tranquility, diet, venesection or bloodletting where necessary, and even marriage. Systematically (in a contemporaneous sense), Hippocrates and his colleagues devised a series of therapeutic regimens that they believed would reestablish the humoral balance thought to underlie most diseases; they also employed surgical techniques such as trephining to relieve purported pressure on the brain.

Several themes relevant to the mind and its difficulties characterize ***Plato***'s (429–347

B.C.) work: (1) Powerful emotional forces could come to the foreground and overwhelm the everyday behavior typifying a person's life; (2) conflicts could exist between different components of the psyche (e.g., the personal discord that often arises between an individual's rational side—that which is desired—and the surge of emotional feelings); and (3) mental disorders did not result from simple ignorance, but from irrational superstitions and erroneous beliefs. To Plato, all humans were partly animal-like; hence all humans acted irrationally at times—some more, some less. He found evidence for these conclusions in dreams, where bizarre events invariably occur and unnatural connections among thoughts and images are dominant.

Not to be overlooked was his contention that therapeutic efforts could modify any and all forms of mental illness. For Plato, the use of educational procedures could dispel ignorance and uncover "truth" through the application of fundamental principles. No less important with regard to therapy was Plato's use of a dialectical model to change a patient's cognitions and belief systems. In this regard, Plato's philosophy provided a methodology for engaging in therapy, essentially the application of rational discussions to modify faulty cognitions (shades of contemporary cognitive therapies!).

Plato had many distinguished students, the most eminent of whom was ***Aristotle*** (384–322 B.C.). Though he was Plato's student for over 20 years, Aristotle turned sharply away from Plato and toward matters more realistic and tangible than abstract and idealistic. Some would say that Aristotle provided history's first integrated and systematic accounts not only of psychological matters, but of astronomy, physics, zoology, and politics. The last of the great philosophers of the 4th century B.C., Aristotle was more scientist than philosopher. He gave special attention to the need for experimental verification and the use of sensory-based observable data; in fact, he was the first of the major philosophers to take an inductive and empirical approach in his writings. He was interested in the concrete observables of experience as registered through the senses. Although he admired the abstract rationalism of Plato, he was much more disposed to deal with the tangible world than with high-order abstractions or broad principles. He believed that data should be grounded in empirical observables in order to minimize the risk of subjective misinterpretations. Despite these reservations, Aristotle believed that thought transcended the sensory realm. As he saw it, imagination could create thoughts of a higher order of abstraction than could sensations themselves.

Yet not all matters were successfully brought within Aristotle's purview. Despite growing evidence that the brain was the center of thought and emotion, Aristotle retained the erroneous belief that the heart served as the seat of these psychological experiences. He made keen and significant observations, however, in recognizing the psychological significance of cognitive processes, dreams, and emotional catharses. For example, it was Aristotle who said that events, objects, and people were linked by their relative similarity or their relative difference from one another. To Aristotle, things became "associated" if they occurred together; in this, he was clearly a forerunner of the associationist school of the 18th and 19th centuries. Aristotle viewed dreams as afterimages of the activities of the preceding day. Although he recognized that dreams might fulfill a biological function, he judged the content of dreams to be ideal gauges of potential pathology. He had a specific interest in how physical diagnoses could be deduced from dream content.

Aristotle's scope was exceptionally broad and inventive. It was he who wrote most perceptively of the intellectual and motivational features of the mind from the viewpoint of a natural scientist. Thus, in what might be termed a psychobiological theory, he outlined the basics of human perception and rational thought, stressing the importance and validity of sense impressions as the source for an objective form of experimental study. Along the same lines, Aristotle articulated a series of proposals concerning the nature of learning—a model based on the principles of association and reinforced by what we have come to term the "pleasure principle." Similarly, he emphasized the importance of early experience and education in the acquisition of skills, and the role of habit and practice in the formation of psychological attitudes. To him, the processes of development were key themes in understanding human behavior.

When Aristotle left Athens in the year 322 B.C., following the death of Alexander the Great, he arranged to have his associate

Theophrastus (371–286 B.C.) succeed him as head of the Lyceum. Shortly thereafter, Aristotle, alone and despondent over the turn of political events in Athens, died in exile. Theophrastus was only a decade younger than Aristotle and had come to Athens to study with Plato. He and Aristotle had been friends, joined together in their travels and shared in their study of nature. Theophrastus remained head of the Lyceum for some 30 years. Perhaps most significant was the attention Theophrastus paid to the study of botany, establishing him as the true founder of that science, just as Aristotle's works established the field of zoology.

A prolific and sophisticated thinker, Theophrastus wrote no less than 220 treatises on a variety of different topics. Although this diversity of work was substantial, he became best known for a secondary aspect of his career, the writing of personality sketches he called "characters." Each of these portrayals emphasized one or another psychological trait, providing a vignette of various personality "types" (e.g., individuals who were flatterers, garrulous, penurious, tactless, boorish, surly, etc.).

Whether these portrayals were penetrating or poignant, Theophrastus (as well as later novelists) was free to write about his subjects without the constraints of psychological or scientific caution. Such lively and spirited characterizations most assuredly captured the interest of many, but they could also often mislead their readers about the true complexities of natural personality patterns.

Although the beginning and ending of the Roman period cannot be sharply demarcated, it basically spanned a 12-century period from the 7th century B.C. to the 5th century A.D., when the last of the major Roman emperors was deposed. As a formal organization, the Roman Republic dated from the 5th century B.C. to the 3rd century A.D.

The more cultured classes of Rome were determined to eliminate magic and superstition as elements in considering psychic processes. A mechanistic conception of mental disorders came to the foreground; it was fundamentally materialistic and opposed to all transcendental mythologies, which were regarded as superstitious beliefs that originated from fear and ignorance. Mental disorders were caused not by the action of mysterious forces, nor by biohumoral movements or conflicts, but by the periodic enlargement or excessive tightening of the pores in the brain. In this corpuscular hypothesis, a derivative of the atomistic notions of Democritus of Greece, the task of the mental healer was to confirm and normalize the diameter of the pores. Persons with certain mental illnesses were seen as apathetic, fearful, and in a depressed mood, by what was called a *laxum* state. Those with other disorders presented an excited, delirious, and aggressive appearance; they were in a *strictum* state. If both sets of these symptoms co-occurred, there was a *mixtum* state.

A follower of the vitalist school of thought that adopted the concept of *pneuma*, the natural or animal spirit, as the physical embodiment of the soul, ***Aretaeus*** (30–90 A.D.) was little known in his time and was rarely quoted by fellow Roman scholars. This was probably owing to the fact that his works were written in the Ionic dialect rather than in Latin or Greek. Furthermore, his vitalistic philosophy, based on the fluidity of the soul's nature, and adopted by Galen a century later, rivaled the more atomistic or solidistic corpuscular theory of his contemporary Roman thinkers. Scarcely familiar with the Greek language and its medical philosophies, Aretaeus was a born clinician who was retained as a physician for the ruling Roman classes.

According to Aretaeus, the vicissitudes of the soul served as the basis of psychic disturbances. Specifically, the interconnecting linkages among "solid organs, the humours, and the pneuma" generated all forms of mental aberration. For example, anger and rage stirred the yellow bile, thereby warming the pneuma, increasing brain temperature, and resulting in irritability and excitability. Conversely, fear and oppression stirred black bile, augmenting its concentration in the blood, and thus leading to a cold pneuma and consequent melancholy.

Disturbances of consciousness usually resulted from the sudden diminishing of the strength of the pneuma around the heart. Aretaeus's descriptions of epilepsy were notably impressive. He spoke of its premonitory symptoms, such as vertigo and nausea, the perception of sparks and colors, and the perception of harsh noises or nauseating smells. Aretaeus also described the origins and characteristics of fanaticism; he formulated a primitive psychosomatic hypoth-

esis in stating that emotions could produce problematic effects on humoral metabolism, noting that "the black bile may be stirred by dismay and immoderate anger." Similarly, he formulated what we speak of as cyclothymia in describing the alternation of depression with phases of mania. He stated, "Some patients after being melancholic have fits of mania . . . so that mania is like a variety of melancholy." In discussing the intermittent character of mania, he recognized its several variants, speaking of one type as arising in subjects "whose personality is characterized by gayness, activity, superficiality, and childishness." Other types of mania were more expansive in which the patient "feels great and inspired. Still others become insensitive . . . and spend their lives like brutes."

Perceptive observations by Aretaeus strengthened the notion of mental disorders as exaggerated normal processes. He asserted that a direct connection existed between an individual's normal characteristics of personality and the expression of the symptoms the individual displayed when afflicted. His insightful differentiation of disorders according to symptom constellations (i.e., syndromes) was a striking achievement for his day.

Although Hippocrates may have been the first to provide a medical description of depression, it was Aretaeus who presented a complete and modern portrayal of the disorder. Moreover, Aretaeus proposed that melancholia was best attributed to psychological causes (i.e., that it had nothing to do with bile or other bodily humors). As noted, he may have been the first to recognize the covariation between manic behaviors and depressive moods, antedating the views of many clinical observers in the 16th and 17th centuries.

Aretaeus was also a major contributor to the humanistic school of thought in early Rome. Most notably, he introduced long-term follow-up studies of patients. He tracked their lifetime course, their periodic disease manifestations, and their return to a more normal pattern of behavior; in this regard, he anticipated the authoritative writings of Emil Kraepelin, who recognized the course of an illness as a key factor in discriminating a specific disorder from others of comparable appearance. He seriously studied the sequence and descriptive characteristics of his patients, contending that a clear demarcation could be made between the basic personality disposition of a patient and the form in which a symptomatic and transient disorder manifested itself periodically.

No less important was Aretaeus's specification of the premorbid conditions of patients; he viewed these conditions as forms of vulnerability or susceptibility to several clinical syndromes. As Aretaeus phrased this, he found that persons disposed to mania are characteristically "irritable, violent, easily given to joy, and have a spirit for pleasantry or childish things." By contrast, those prone to depression and melancholia were seen as characteristically "gloomy and sad often realistic yet prone to unhappiness." In this manner, Aretaeus elaborated those essentially normal traits that make an individual susceptible to a clinical state. As Zilboorg and Henry (1941) have noted, the melancholia of Aretaeus is still observed in our time, although under different psychiatric labels. Owing to his observations of patients over extended periods of time, Aretaeus proposed a series of predictions about the general outcomes of different mental conditions. More than other physicians of his day, Aretaeus not only described psychological conditions with keen sensitivity and humane understanding, but (in a spirit more akin to recent scientific work) sought to compare various clinical syndromes and illuminate ways in which they could be differentiated.

Claudius Galenus (Galen) (131–201; see Figure 1.2) was the last major contributor to adopt a psychological perspective in Rome. He preserved much of the earlier medical knowledge, yet generated significant new themes of his own. Galen lived more than 600 years after the birth of Hippocrates. A Greek subject of the Roman Empire, he was born in Asia Minor about 131 A.D. During his mature years, numerous radical political and cultural changes took place in Rome. Galen and his medical associates set out to synthesize primitive conceptions of disease with then-modern methods of curing the sick. Following the ideas of Hippocrates, he stressed the importance of observation and the systematic evaluation of medical procedures, arguing against untested primitive and philosophical hypotheses in favor of those based on empirical test. As a follower

FIGURE 1.2. Galen.

of Aristotle as well as Hippocrates, Galen emphasized the data of experience, rather than logical hypotheses that were devoid of factual evidence. Unfortunately, he doubted that environmental and psychological factors could affect the course of human disease. Although Galen avoided philosophical themes concerning the nature of illness, he nevertheless proposed a principle termed *spiritus anima*, in which he asserted that humans possessed an extraphysical life-giving force; this thesis was based on his efforts to distinguish organic from inorganic matter.

Galen's conception of psychic pathology was based on the physiology of the central nervous system. He viewed clinical symptoms as signs of dysfunctional neurological structures and characterized mental diseases as "a concourse of symptoms," among which a specifically pathognomonic one could be isolated. According to his organic-functional approach, mental symptoms originated from the pathogenic action of a toxic, humoral, vaporous, febrile, or emotional factor that affected the brain physically and then altered certain of its psychic functions. Consonant with the beliefs of his time, Galen believed that the activities of the mind were prompted by animal spirits that carried out both voluntary and involuntary actions. These animalistic spirits (*pneuma*) were divided by Galen into two groups: those that controlled sensory perceptions and motility, whose damaging effects would cause neurological symptoms; and those that had more directive functions, such as coordinating and organizing imagination, reason, and memory. To him, most psychiatric symptomatology stemmed from alterations of the second group of functions.

In describing catatonic psychosis, Galen suggested a paralysis of the animal spirits in which the imaginative faculty was "blocked or incomplete." As far as the syndrome of hysteria was concerned, he disagreed strongly with Hippocrates's uterocentric view. Galen asserted that hysteria, on the basis of his own clinical examinations, could not be a disease that reflected the uterus "wandering agitated in the body." As he saw it, hysterical symptoms were provoked by the toxic action of vapors that formed in the normal uterus and vagina; it arose from the stagnation of semen, owing to a lack of sufficient sexual intercourse. The disease therefore signified a lack of sexual hygiene.

Galen's stature grew over the next millennium—so much so that his views were thought to be sacrosanct. His writings were summarized and commented on by many lesser physicians, most of whom were recognized as being wrong-headed; indeed, their books were often referred to as "wretched treatises." Some of these post-Galen compilations were not based on his work at all, but dishonestly carried his name for its ability to promote the sale of untenable or alien ideas. Although many of his notions were diluted by the passage of time or refuted by empirical knowledge, his vast contributions must be considered significant, in that no other figure in history exercised so extended an influence on the course of medicine.

Later in Roman history, there emerged an organized theology known as Christianity, including faith healing, magic, and superstition. The doctrine of the early Christian church became the dominant approach to thought, medicine, and mental healing in the Western world until the 17th century. Most of the populace remained illiterate during this period. Education was religious, otherwise inchoate, and of dubious value. The idea of a scientific basis for understanding mental disorders barely appeared on the scene. Faith was the all-powerful guide.

During the first two to three centuries A.D., a distinction was made between psy-

chologically normal individuals who doubted the dogma of the church's ideology, and those whose "peculiar" beliefs arose not out of opposition, but out of a mental affliction. Nevertheless, both groups were considered guilty of heresy and subjected to punishment. In a similarly irrational twist, others' implausible or nonsensical behavior ostensibly demonstrated their fervent adherence to church authorities and their dogma. Such persons were venerated. It was not long thereafter that the works of Aristotle and other Greek philosophers were condemned.

Christianity in the 3rd century led physicians to assume a moralistic and judgmental approach to psychic pathology. Unable to escape the growing spirit of superstitiousness, they proposed that mental cases were definitely the products of mystical events that could not be understood in the natural world. More seriously, they adopted the ancient belief that demons often appeared under the guise of confused humans, and that it was the job of physicians to identify and to "eliminate" them. In this and other similar matters, they laid the groundwork for a return to the age of supernaturalism and superstitions; they were nevertheless thought well of until the close of the 17th century.

Aurelius Augustine (354–430) was a key figure in the transition from early Roman thought to the Middle Ages. Better known as St. Augustine of Hippo, we can see in his writings an effort to synthesize the Greek and the new Christian perspectives on mental maladies. Perhaps the most influential philosopher of his time, Augustine set the foundation and tone of Christian intellectual life for centuries to come. To him, all knowledge was based on the belief that only God could provide the ultimate truth, and that to know God was the ultimate goal. To think otherwise, as Augustine averred, would not only be vain, but would assuredly lead to error and corruption. Individuals, as children of God, would in their faith begin to understand the very nature of life, and thereby would be able to lead a life of grace and honor.

The Early Muslim World

Three major medical figures from the Muslim world of the Middle East around the end of the first millennium A.D. are worthy of note: Rhazes, Unhammad, and Avicenna. Each proposed helpful ideas that came to represent a fresh and innovative point of view concerning mental illness.

Rhazes (860–930) lived during the late 9th and early 10th centuries and wrote textbooks dealing with a wide variety of medical, psychological, philosophical, and religious subjects. In contrast to the predominant religious orientation of Baghdad, Rhazes strongly argued against the notion of a demonological concept of disease and the use of arbitrary authority to determine what is scientific and what is not. He attacked the superstitious religious beliefs of his contemporaries and was strongly in favor of developing a rational schema for understanding all disorders. Empirically oriented, he nonetheless subscribed to the theory of the four elements originally developed by Empedocles and Hippocrates.

Unhammad (870–925) was a contemporary of Rhazes who provided intelligent descriptions of various mental diseases. The observations he compiled of his patients resulted in a nosology that was the most complete classification of mental disorders in its day. Unhammad described nine major categories of mental disorders, which, as he saw it, included 30 different diseases. Among the categories was an excellent description of anxious and ruminative states of doubt, which correspond in our thinking today with compulsions and obsessions. Other categories of mental disease were judged by Unhammad to be degenerative in their nature; a few were associated with the involutional period of a man's life. The term used by the Greeks for mania was borrowed to describe states of abnormal excitement. Another category, most closely associated with grandiose and paranoid delusions, manifested itself, according to Unhammad, in the mind's tendency to magnify all matters of personal significance, often leading to actions that proved outrageous to society.

A most significant and influential philosopher and physician of the Muslim world was ***Avicenna*** (980–1037), often referred to as the "Galen of Islam," largely as a consequence of his vast and encyclopedic work called the *Canon of Medicine*. The *Canon* became *the* medical textbook chosen throughout European universities from the 10th through

the 15th centuries. However, Avicenna was not regarded as a highly original writer, but rather as a systematizer who encompassed all knowledge from the past that related to medical events. Similar to Galen, Avicenna noted the important connection between intense emotions and various medical and physiological states, although he fully accepted Hippocrates's humoral explanations of temperament and mental disorder. To his credit as a sophisticated scholar of the brain, Avicenna speculated that intellectual dysfunctions were in large part the results of deficits in the brain's middle ventricle, and asserted that common sense and reasoning were mediated by the frontal areas of the brain.

The Middle Ages

The enlightened ideas of Hippocrates were submerged for centuries after the death of Galen and the fall of the Roman Empire. During the thousand years of the so-called Dark Ages, superstition, demonology, and exorcism returned in full force and were further intensified by sorcery and witch burning. With few dissenting voices during this period, the naturalism of the Greco-Roman period was all but condemned or distorted by notions of magic. Only in the Middle East did the humane and naturalistic aspects of Hippocratic thought remain free of the primitivism and demonology that overcame Europe.

Signs for detecting demonic possession became increasingly indiscriminate in the Christian world. During epidemics of famine and pestilence, thousands wandered aimlessly until their haggard appearance and confusion justified the fear that they were cursed. The prevalent turmoil, the fear of one's own contamination, and the frenetic desire to prove one's spiritual purity led widespread segments of the populace to use these destitute and ill roamers as convenient scapegoats.

As the terrifying uncertainties of medieval life persisted, fear led to wild mysticism and mass pathology. Entire societies were swept up simultaneously. Epidemic manias of raving, jumping, drinking, and wild dancing were first noted in the 10th century. Referred to as "tarantism" in Italy, these epidemic manias spread throughout other parts of Europe, where they were known as St. Vitus's Dance.

During the early Middle Ages, before later catastrophes of pestilence and famine, few people with mental illnesses were totally destitute. Monasteries served as the chief refuge for such individuals, providing prayer, incantation, holy water, relic touching, and mild exorcism as prescriptions for cure. As the turmoil of natural calamity grew more severe, mental disorders were equated increasingly with sin and Satanic influence. Significant advances were made in agriculture, technology, and architecture during the Middle Ages, but the interplay between changing theological beliefs and naturalistic catastrophe speeded acceptance of the belief that "madness" and "depravity" were the devil's work. At first, it was believed that the devil had seized mentally ill individuals against their will, and such individuals were treated with established exorcistic practices. Soon, however, the afflicted were considered willing followers of Satan; classed now as witches, they were flogged, starved, and burned.

Among the major tenets of this medieval mythology was a belief that an international conspiracy, based on Satanic forces, was bent on destroying all forms of Christianity. The agents of this widespread conspiracy were witches, who not only worshipped Satan at secret meetings, but attempted to desecrate Christian symbols and beliefs, as well as to engage in murder, cannibalism, and sexual orgies. The ideas of a demonic and Satanic conspiracy existed first and foremost in the imagination of the religious leaders of the day. It was Pope Gregory IX who established the Inquisition in 1233 to root out witches, heretics, and all other agents of Satan, who he asserted were setting out rapidly to destroy the clerical and political orders of the Church. Those with an administrative status possessed the legal right to judge which aspects of Satanic witchcraft would be deemed demonic. It was not only higher-order religious leaders who conveyed this dogma; the common people took these belief systems to heart, as well. From the 15th through the 17th century, demonic possession and exorcism became common phenomena among the masses. In the postmedieval period, both Catholics and Protestants believed that

witches, fueled by Satanic forces, would send demons to possess those judged to be undesirable. It was the task of religious authorities to coerce those possessed by demons to admit that they were witches. These individuals could justly be arrested and tortured, especially if they "confessed" to their involvement in these nonexistent Satanic conspiracies. "Witch finders" soon became prominent guardians of the faith, prompted by religious authorities who sought to undo the political powers of their ostensible "enemies."

Encouraged by the 1484 *Summis Desiderentes Affectibus*, in which Pope Innocent VIII exhorted the clergy to use all means for detecting and eliminating witchcraft, two inquisitional Dominicans, Heinrich Kramer and Johann Sprenger, issued a notorious manual titled *Malleus Malefacarum* (*The Witches' Hammer*). Published between 1487 and 1489, this "divinely inspired" text set out to prove the existence of witchcraft, to describe methods of identification, and to specify the procedures of examination and legal sentencing.

Malleus Malefacarum reflected the spirit of its time, even though it was published in the early stages of the Renaissance and at the threshold of the Reformation. Here the conflict between paganism and Christianity, between magic and a monotheistic outlook, had not ceased to be a burning issue (in more than one sense of the word). As the ancient idols and deities were torn down from their pedestals, demons nevertheless retained their grip on the minds of the ordinary people. Idols and deities were relegated to the role of fallen angels, but devils and evil demons continued to reside in the human unconscious, and belief in them continued to be widely embraced.

With torture recommended as a means of obtaining confession, and with feelings of guilt and hopeless damnation characteristic of many of the afflicted, the inevitable consequence for most persons with mental illnesses was strangulation, beheading, or burning at the stake. Unredeemed by good sense and wise judgment, this barbaric epidemic swept Protestant and Catholic countries alike, including several American colonies. Although the last execution of a witch occurred in 1782, the bewildering notion that mentally ill individuals were in league with the devil persisted in popular thought well into the 19th century.

It was in the 15th century that the medieval period began to be gradually transformed into what we view today as the modern world. Slowly but persistently, the importance of human emotions and strivings became a significant element to guide intellectual thought, ultimately replacing the medieval belief that the revelation of deeper human truths were beyond human capabilities. Psychological processes became increasingly humanized; opportunities to study human beings as biological rather than purely spiritual organisms permitted these processes to be considered as aspects of natural rather than metaphysical science. Christianity had begun to lose its spirit and vitality; although the supernatural world still existed in human minds, it had lost much of its power, increasingly ruled by static and rigid belief systems and symbols.

Desiderius Erasmus (1465–1536) was a sincere churchman who asserted a new humanism. He attacked the formalism and the corruption of the church, which he judged as sterile and possessed of rituals that were divested of their purpose and humanism. As Robinson (1976) has noted, his "psychology" was both practical and wise, expressed with verve and clarity in Erasmus's *Colloquies*. Here he pricked vanity, exorcised exorcism, lamented superstition gleefully, and guided individuals to their duty to adhere to the simple and humane lessons of Christ's life, rather than to behaviors that would obscure or deceive his worthiness. In his essays and letters, Erasmus, neither scientist nor formal philosopher, addressed the everyday world, seeking to expose its vanities, follies, charlatans, and warmongers. His was the attitude of a Renaissance humanist, with a fine mind and a sympathetic heart. So, too, was the humanistic outlook of the Spanish Jew ***Juan Louis Vives*** (1492–1540), who contributed fundamentally to educational reforms and evinced a passionate concern for the welfare of those with mental illnesses, who were routinely incarcerated and maligned.

The Renaissance and Beyond

Gradually, the horrors of the Inquisition were left behind. In the 16th century, the

work of intelligent and humanistic thinkers slowly awakened humankind from its long slumber. Zilboorg and Henry (1941) have written that for several centuries, philosophers had repeatedly stated that human beings should be studied and not their souls; these scholars slowly convinced themselves by listening to their own voices. These scholars were not physicians, because physicians had turned their attention to the new anatomy and physiology of animals and cadavers, rather than the emotions and natural states of living humans.

The waning of medieval supernaturalism and the advent of the liberating Renaissance era had numerous effects upon the emergence of psychological thought. The Renaissance broke the hold of medieval dogma upon the mind of early clinicians. It also opened up new nonphilosophical pathways for purely psychological ventures and inquiries into the general character of human nature, as well as the substantive nature of mental disorders.

The Theories of Paracelsus

Paracelsus (1493–1541) would have been an extraordinary person in any historic age, but, given his time, he looms as a strange if not rare blend of the mysticism of the past with the practicality of his day. Paracelsus's actual name was Theophrastus Bombastus Von Hohenheim. Perhaps in anticipation that he would be a courageous and intrusive battler all of his life, he shortened his name to simply Paracelsus, even if its selection was rather pretentious. Specifically, he adopted this name to suggest that his views were superior to those of Celsus, the chief medical authority of ancient Rome. Others have suggested that he gave himself this name in order to show that he was surpassing the encyclopedists and medical methodists of his time—in other words, that he intended to blaze a new trail by his adventurous approach to mental disorders. Like most thinkers of his day, Paracelsus was a believer in divination from the stars and in the healing powers of such preparations as powdered Egyptian mummy. As such, he was both an astrologer and an alchemist.

For some of his disciples, Paracelsus was the towering medical figure of the Renaissance period, comparable to such other contemporary luminaries as Leonardo da Vinci, Copernicus, and Shakespeare. To most historians of today, he is regarded as an imaginative adventurer, if not a charlatan, and most are inclined to view his contributions to scientific medicine as modest at best. Among his works were efforts to test the effects of various chemical agents to treat several medical conditions (attempts not unlike the activities of pharmaceutical firms today). Although he made no lasting discoveries, he was an inventive and creative pioneer. Nevertheless, the whimsies he had proposed were consigned largely to the rubbish heap. Despite a number of sound insights, he dissipated much of his energy combating colleagues who did whatever they could to make his life unbearable.

When Paracelsus interrupted his mystical flights of fantasy to deal with his medical opportunities, he spoke in a voice akin to that of a seeker of scientific truth, despite his rebellious defiance of ancient traditions and scholastic dogma. Most notably, he denounced the cruelties of the Inquisition, stating that "there are more superstitions in the Roman Church than in all these poor women and presumed witches." In his rejection of the views of the clergy regarding the sources of mental disorders, Paracelsus (1567/1941) wrote:

> In nature there are not only diseases which afflict our body and our health, but many others which deprive us of sound reason, and these are the most serious. While speaking about the natural diseases and observing to what extent and how seriously they afflict various parts of our body, we must not forget to explain the origin of the diseases which deprive man of reason, as we know from experience that they develop out of man's disposition. The present-day clergy of Europe attribute such diseases to ghostly beings and threefold spirits; we are not inclined to believe them.

Paracelsus was the first physician to lay out a systematic classification of disorders that abandoned the habit of categorizing disorders by beginning with the head, then working down step by step to the feet. His mental health classification was outlined in a treatise titled "On the Diseases Which Deprive Men of Health and Reason." Here whole groups of persons with mental disorders were identified, notably *lunatici*, *insani*,

vesani, and *melancholici*. *Lunatici* suffered from disorders stemming from their reactions to the phases of the moon. *Insani* suffered from disorders identifiable at birth and clearly derived from family heritage. *Vesani* were poisoned or contaminated by food or drink. *Melancholici*, by virtue of their temperament, lost their ability to reason accurately. In addition to these four forms of mental illness, Paracelsus identified others as *obsessi*—that is, obsessed by the devil. In this latter formulation, Paracelsus was dissenting from the dogmatic view of earlier centuries in which a devil obsession lay at the heart of all mental disorders. As he perceived it, numerous sources of mental dysfunction existed, only one of which could be traced to demonic preoccupations; he saw the other disorders as problems of defective thought processes, rather than as consequences of supernatural powers.

Physiognomy and Phrenology before, during, and after the Renaissance

"Physiognomy," the art of interpreting people's psychological characteristics from aspects of their physical characteristics, was present in ancient times, reaching its peak of study in the 2nd century A.D. Advocates of physiognomy assumed that inner traits of people are expressed in their outer physical features, especially the face. The great thinkers of Greece made formal efforts to systematically interpret physiognomic characteristics—for example, in Pythagoras's 6th-century B.C. writings, and later in Aristotle's *Analytica Priora* (Tredennick, 1967) and *Historia Animalium*, where he wrote: "Persons who have a large forehead are sluggish, those who have a small one are fickle; those who have a broad one are excitable, those who have a bulging one, quick tempered" (Peck, 1965, I, VIII, 891b, p. 39).

Physiognomica (Hertt, 1936)—also attributed to Aristotle, but more likely written by his followers—examined parallels between the physiques of men and animals, to compare different ethnic groups, and to investigate the relationship between bodily characteristics and temperamental dispositions. Among the useful signs recorded were the movements, shapes, and colors of the face; the growth of hair; the smoothness of skin; the condition of the flesh; and the general structure of the body. Sluggish movements denoted a soft disposition, quick ones a fervent temperament; a deep voice denoted courage, a high one signified cowardice. The writers were wise enough to note that it would be foolish to base a judgment on any one of these signs. Centuries later, ***Leonardo da Vinci*** (1452–1519) made similar physiognomic proposals in his *Treatise on Painting*, in which he explored relationships between emotional states and overt facial expressions.

In the 16th century, ***Giovanni Battista della Porta*** (1535–1615) published a book titled *De Humana Physiognomia* (1586), derived from Aristotle's writings, which included many drawings designed to show similarities between humans and animals. For example, a person who looked leonine ostensibly possessed the courage, strength, and will of a lion. This book proposed the theory that every person's head resembled a specific animal's head, thereby suggesting that the person possessed the same personal characteristics as that animal. Another work by Porta, *Natural Magick* (1558/1957), outlined similar speculations by a number of his contemporary colleagues. No less speculative was Porta's *Phytognomonica* (1588), in which he addressed matters of vegetable physiognomy—that is, the art of determining the inner nature of plants on the basis of their exterior appearance.

In his five-volume work, *Les Charactères des Passions* (1640), eminent French physician ***Marin Cureau de la Chambre*** (1594–1669) wrote:

> the resemblance Man has with other Creatures . . . teacheth us that those who have any part like to those of beasts, have also their inclinations . . . that men who have anything of a feminine beauty, are naturally effeminate; and that those women who have any touch of a manly beauty, participate also of manly inclinations.

Burdened with the prejudices of his day, de la Chambre (1640) was nevertheless a highly insightful physiognomist, addressing in detail the significance to be found "in the motions of the eyes, the inflection of the voice, the color of the lips," and so on. Unfortunately, he could not help but draw

upon astrological influences, speculating on the power, especially of the moon upon the brain, "causing it to increase or decrease in volume upon whether the moon is waxing or waning."

A distinguished philosopher and jurist, ***Christian Thomasius*** (1655–1728) helped inaugurate the period of German enlightenment, founded the University of Halle, and asserted that philosophy should concern itself with practical matters of everyday life. A prolific author, Thomasius wrote only briefly on physiognomy, drafting an essay entitled *Recent Proposals for a New Science for Obtaining a Knowledge of Other Men's Minds* (1692). Basing his ideas on the work of de la Chambre, Thomasius recommended that observation can be most useful when obtained through personal conversation with one's subject; he also cautioned that observers must distinguish between genuine and affected emotions.

A theorist of physiognomy in the late 18th century, ***Johannes Kaspar Lavater*** (1741–1801), asserted unequivocally the existence of a relationship between fixed aspects of the body's surface and a person's character. In his well-received book *Essays on Physiognomy* (1789), published in four lavish volumes, Lavater claimed that physiognomy was truly a science because it offered law-like regularities and depended on empirical observation. In characterizing the trait of obstinacy, Lavater wrote:

> The higher the forehead, and the less the remainder of the countenance, the more knotty the concave forehead, the deeper sunken the eye, the less excavation there is between the forehead and the nose, the more closed the mouth, the broader the chin, the more perpendicular the long profile of the countenance—the more unyielding the obstinacy: the harsher the character.

Though similar in many respects to classical approaches in physiognomy, a new "scientific" model known as "phrenology" emerged in the late 18th century. Both approaches drew inferences about character and personality from external bodily features—physiognomy from facial structure and expression, phrenology from external formations of the skull. Their underlying assumptions, however, were quite different. Physiognomists believed that a person's inner feelings and characteristics were expressed in facial features, voice, and so on. Phrenologists made no assumptions as to the external expression of varied dispositions. Their two fundamental assumptions were unusual for their era: First, that different mental functions were located in different regions of the brain; and, second, that the skull's external topography reflected the magnitude of these functions. This was the first "scientific" effort made to analyze the underlying brain structure from which character and personality might be derived.

Despite its discredited side, phrenology, as ***Franz Joseph Gall*** (1758–1828) proposed it, was an honest and serious attempt to construct a neurological substrate in the brain to undergird a science of character depiction. Although numerous writers such as Vesalius, Willis, and Stensen in medieval times (Millon, 2004) had speculated and explored brain structures as the center of mental functioning, Gall took this view in an original direction. Most early characterologists conceived the brain as a locale where the immaterial soul might influence bodily activities. Gall asserted not only that the brain *was* the mind in an explicitly material sense, but that different regions subserved different dispositions.

Gall identified 27 different "organs" in the brain that undergirded separate psychological tendencies. Through "reading" the skull (usually by running one's hands over the head), one could identify different enlarged organs. Gall went to prisons and lunatic asylums to read skulls and collect data on correlations between protuberances in certain locations and personality traits.

Gall referred to his research on brain physiology as "organology" and "crainoscopy," but the term "phrenology," which his younger associate Johann Spurzheim coined, came to be its popular designation. As noted, the rationale that Gall presented for measuring contour variations of the skull was not illogical. In fact, his work signified an important advance over the naive and subjective studies of physiognomy of his time, in that he sought to employ objective and quantitative methods to deduce the inner structure of the brain. He concluded, quite reasonably, that both the intensity and character of thoughts and emotions would correlate with varia-

tions in the size and shape of the brain or its encasement, the cranium. That this gross expression of personality proved invalid is not surprising when we think of the exceedingly complex structure of neuroanatomy.

Views from the Later Renaissance to the 17th Century

A chronically depressed clergyman and reclusive scholar, ***Robert Burton*** (1576–1640), wrote a single major work of extraordinary insight and sensitivity in 1621, titled *The Anatomy of Melancholy*. Burton's introspective accounts of his moods contained a wealth of impressive clinical analyses. He also sought to record the behavior and emotions of others, recognizing patterns similar to his own moodiness and eccentricity. This great volume of work, despite rambling irrelevancies and inaccuracies, makes fascinating reading today, as may be judged from the following excerpt:

> It is most absurd and ridiculous for any mortal man to look for a perpetual tenure of happiness in this life. Nothing so prosperous and pleasant, but it hath some bitterness in it, some complaining, some grudging; it is all a mixed passion, and like a chequer table, black and white men, families, cities, have their falls and wanes; now trines, sextiles, then quartiles and oppositions. We are not here as those angels, celestial powers and bodies, sun and moon, to finish our course without all offence, with such constancy, to continue for so many ages: but subject to infirmities, miseries, interrupted, tossed and tumbled up and down, carried about with every small blast, often molested and disquieted upon each slender occasion, uncertain, brittle, and so is all that we trust unto. (Burton, 1621, p. 261)

Although his perspective was limited, Burton did establish a classification system, one that differentiated melancholy from madness—a distinction akin to our differentiation of neuroses from psychoses. He outlined the following general categories: (1) diseases emanating from the body; (2) diseases of the head (primarily the brain); (3) madness (mania); and (4) melancholy, for which Burton further distinguished melancholy of the head, the body, or the bowels, and identified the major sources of melancholy (e.g., excessive love, excessive study, intense preoccupation with religious themes).

Burton's introspective awareness of his own personal sadness and depression led him to recognize the sources of his own melancholy. He recognized guilt as a major element, despite his exemplary lifestyle. Other causes of melancholy included bodily deterioration and old age; bad diets; sexual excesses; idleness; solitariness; and an overpreoccupation with imagination, fears, shame, and malice. Burton clearly stated that melancholy could be engendered by a wide range of human frailties and life circumstances.

Among the many topics that Burton included in his book on melancholy, he touched on a variety of mental aberrations that we recognize today as obsessions and compulsions. Thus he wrote of an individual

> who dared not to go over a bridge, come near a pool, rock, steep hill, lie in a chamber where cross-beams were, for fear he'd be tempted to hang, drown, or precipitate himself. In a silent auditorium, as at a sermon, he was afraid he shall speak aloud at unawares, something indecent, unfit to be said. (1621, p. 253)

Burton anticipated what ultimately became the core of modern psychotherapy—that is, engaging a patient in a dialogue with a trusted and sympathetic outsider. But because he was not part of the medical establishment, his proposals had little effect on the course of mental health study of his time, despite the brilliance of his book.

A man of great intensity and imagination, ***Thomas Willis*** (1621–1675) was the originator of the term "neurology"; he also generated the term "psychology" to designate the study of the so-called "corporeal soul." Arguably the most significant founder of what came to be referred to as "biological psychiatry," he considered most ailments to be disorders of nerve transmission, rather than diseases of the blood vessels. He is perhaps best known by the circuit of arteries located at the base of the brain, known today as the "circle of Willis."

In 1664, Willis published a major book on the history of the brain sciences, entitled *Cerebri Anatome*. It was a work of considerable scope and insight, and was for many decades thereafter without equal in the field. The title suggested that the book was limited to anatomy. However, Willis, a thoroughly educated Oxford physician, concerned himself not only with brain functions, but with

their behavioral consequences. Willis proposed also that vital and involuntary systems existed in the brain that were mediated not by the higher centers of the brain, but by the "cerebellum." The detailed articulation of the functional segments of the brain, grounded in comparative anatomic precision, was enriched by his clinical observations. Drawing ideas from existing theories, his work, both speculative and empirical, stimulated many another neuroanatomist.

In his clinical work, Willis (1664/1978) reported his observation of a sequence in which "young persons who, lively and spirited, and at times even brilliant in their childhood, passed into obtuseness and hebetude during adolescence." Thus Willis anticipated by two centuries an idea more fully developed by Benjamin Morel, who termed this behavioral course "dementia praecox." To his credit, Willis rejected the idea of a "wandering womb" that ostensibly led to the syndrome of hysteria. In his view, the brain functioned as the center of all mental disturbances, and the various nerves emanating from the brain served to connect this overarching organ to the rest of the body. Willis, like most others of his time, spoke of processes generated by "animal spirits"—that is, the soul, which somehow or other could be sucked out of the brain. Also of note was Willis's observation that melancholia and mania frequently coexisted within the same person, who would shift erratically from an excited state to one of depression. This observation contributed to what we now refer to as bipolar disorder and/or manic–depressive psychosis.

Willis's clinical observations were uncontaminated by formal theories. His accurate inferences were based on repeated observations of patients over time—that is, on the long-term course of their difficulties. Included in Willis's classification system were some 14 categories, of which several were primarily neurological. His system, published in *De Anima Brutorum* (1672/1971), specified three major impairments: morosis, mania, and melancholia (each encompassing several subcategories). It also encompassed a number of neurological disorders, such as headache, insomnia, and vertigo.

Thomas Sydenham (1624–1689), a colleague of the philosopher John Locke, held strongly to the view that hypotheses should be set aside in favor of closely observing all forms of natural phenomena, such as various medical diseases. As he put it, too many writers had saddled fairly distinct diseases with excessive features that stemmed from their overblown interpretations. Sydenham did not trust books, believing only what he could see and learn from his own bedside observations (Comrie, 1922). Locke preached that all reliable knowledge came from observation. In his work, Sydenham came to typify the 17th-century empiricist emphasis in England.

Especially informative were Sydenham's contributions to the description of hysteria. His observations of hysterical patients enabled him to recognize the variations of conversion symptoms among patients with paralysis and pain, as well as to speculate on the operation of intense but unconscious emotions. The precision of his descriptions of hysterical phenomena was so comprehensive that little can be added today to what he said over three centuries ago. He recognized that hysteria was among the most common of chronic diseases, and observed that men exhibited the symptom complex no less than women. He averred that hysterical symptoms could simulate almost all forms of truly organic diseases; for example, he noted that a paralysis of the body might be caused by stroke, but could also be found in a hysterical hemiplegia "from some violent commotion of the mind." He spoke of hysterical convulsions that resembled epileptic attacks, psychogenic palpitations of the heart, and hysterical pain that could be mistaken for kidney stones; he also suggested that differential diagnosis between real biological diseases and those generated by the mind could only be made if the patient's psychological state could be thoroughly known. He was among the most successful in illustrating that emotions can generate and simulate physical disorders. In his efforts to formulate a syndromal pattern for numerous disorders, he extended the range of his observations to include not only the patient's dispositions, emotions, and defenses, but the family context within which they arose. In this way, he sought to determine the overall pathogenesis of certain syndromes, largely through the use of both physical and psychological phenomena. What was most informative was Sydenham's recognition that a syndromal picture rarely developed from a single pathogenic agent, be it a humoral imbalance or a systemic disturbance of the

body. In fact, to Sydenham, multiple influences operated simultaneously on a patient, each of which took a somewhat different turn and produced a somewhat different appearance in the same disease process. He strongly believed in syndrome complexes rather than in a distinct or singular expression of a disorder. As a consequence, all physicians were eventually trained to consider a wide range of elements, which together play a partial role in generating disease. However, Sydenham believed that hypotheses and philosophical systems should be set aside to ensure that pathological phenomena were observed with reliability and accuracy.

Particularly notable was Sydenham's belief in nature's own healing processes. These natural remedies of the body would not invariably solve a problem because they were often delayed or displaced. Included among the healing processes of nature, according to Sydenham, were a variety of well-established "excretions, eruptions, and fevers." Sydenham's speculations were based on comprehensive observations, which comprise the most modern methods for investigating mental illness and diagnosing specific clinical syndromes. He also emphasized the importance of identifying the antecedent emotional factors that may lead to the development of mental disorders. Insightfully, he observed the interplay between personal emotions and social pressures.

Born in Germany, ***Georg Ernst Stahl*** (1660–1734) wrote his doctoral dissertation in his early 20s. In it, he expressed the view that the then-prevalent theory of animal spirits was essentially incorrect, and that the various processes of the mind stemmed from a life-giving force, to which he applied the term "soul." However, Stahl's soul was not the supernatural phenomenon that characterized ancient and medieval thinking; it represented the source of energy of all living organisms, both human and animal.

Stahl's life force was not notably different from Freud's conception of the libido. It was the sum total of the nonmaterial side of humans and animals, which, together with nature, had the power to effect desired cures. Hence Stahl's "soul," which in many ways is equivalent to our "psyche," was able to perform a variety of functions that could either bring on or stave off various diseases.

Many present-day scholars consider Stahl the originator of the distinction between organic and functional mental disorders. To him, mental disorders were the result of neither physical, mechanical, nor supernatural forces, but were in fact essentially psychogenic. Stahl was appalled by the sharp demarcation of body and mind. Not only did he judge this dichotomy to be unjustified, in that it hindered a fundamental understanding of disease unity, but it was especially problematic in understanding the complexity of forces involved in mental diseases. He advocated a synthesis of physical and mental phenomena.

The 18th and 19th Centuries

As clinics and hospitals began to record case histories and detail observations, physicians could identify syndromal groupings (i.e., clusters of symptoms) and classify them into disease entities. The success with which botanical taxonomists had systematized their field by the 18th century provided additional impetus to the trend toward categorizing symptom clusters into a formal psychiatric taxonomy or nosology.

A second major trend within biological medicine—the view that mental disorders might result from organic pathology—can be traced to the early writings of Hippocrates, Aretaeus, and Galen. With the advent of valid anatomical, physiological, and biochemical knowledge in the early 18th century, and the discovery in the 19th century of the roles played by bacteria and viruses, the disease concept of modern medicine (including the view of mental illness as a disease) was firmly established. Efforts at developing somatic (e.g., electrical, chemical, surgical) treatment methods followed naturally. Although these three stages—diagnostic classification, biological causation, and somatic treatment—rarely proceeded in a smooth or even logical fashion, they characterized progress in psychopathology and continue today to guide neuroscientists who follow the medical and biological tradition.

These scientific and medical activities, however, presuppose a classification system (i.e., a taxonomy) that is not only logical but valid. Unfortunately, physicians classified diseases long before they understood their true nature. Such nosologies have persisted because of widespread or authoritative use; however, they rested most often

upon unfounded speculations or, at best, judicious but essentially superficial observations. Criticism of premature nosological schemes is justified, given the frequent slavish adherence to them. On the other hand, there is no reason to overlook the *potential* value of a taxonomy, or to abolish a sound classification system that may serve many important purposes. With the waning role of supernaturalism and the advent of liberating thought during the Renaissance, several enlightened thinkers of the 16th and 17th centuries began to explore ideas related to a realistic classification of mental disorders, as reported above.

Perhaps the leading taxonomist of the 18th century was ***François Boissier de Sauvages*** (1706–1767). He had completed his dissertation at the age of 20, defending the teachings of the faculty at his medical school, Montpelier. Conservative in mind but clearly distant from the demonological prejudices that were prevalent in most academic circles of his day, he thought that all mental diseases were located in distinct anatomical regions. Moreover, he believed that the "will" had much to do not only with the generation of mental aberrations, but also with their ultimate treatment. He also believed that physicians had a responsibility to shape or guide individual behaviors; otherwise, there would be no social compact or personal justice.

de Sauvages followed Linnaeus in seeking to create an encyclopedic framework for the many categories of mental disorder. He outlined 10 classes, 295 genera, and 24,000 species, spending the better part of his life immersing himself in the large body of medical knowledge that had accumulated from early times. His urge to catalog the bewildering and scattered array of human disorders can be viewed as an effort to surmount the rather spotty and supernatural beliefs that typified earlier thought.

Besides being a physician, de Sauvages was a botanist. Most of his colleagues spent their time arranging plants and animals in a clearly articulated and "evolutionary" system; the latter was a new departure that did not achieve its fullest impact until the work of Darwin a century later. The details of de Sauvages's presentations were first published in a small book, *Treatise de Nouvelles Classes de Maladies* (1731). Included in his broad classification were such illnesses as fevers, inflammations, spasms, breathing disturbances, weaknesses, pains, and dementias. Dementias, which comprised the bulk of mental diseases in this book, were organized into four types: those of extracerebral origin, disturbances of the instinctual and emotional life, disturbances of the intellectual life, and irregular eccentricities and follies.

de Sauvages completed the three-volume *Nosologie Methodique* (1771) late in life, and it was published several years after his death. In this work, de Sauvages made available to others the complete model he had constructed; this model was used as an orderly classification for decades, if not centuries, to come. In this comprehensive volume, de Sauvages organized all forms of mental illness. For example, he grouped the syndrome of melancholia into numerous species (e.g., religious, imaginary, extravagant, vagabonding, enthusiastic, and, sorrowful).

In the late 1770s and early 1780s, a distinguished physician and professor at the University of Edinburgh, ***William Cullen*** (1710–1790), became a most influential nosologist; he drew upon the work of de Sauvages, but extended the Linnean themes even more comprehensively. The Frenchman Philippe Pinel, who played a well-publicized role in the movement toward humane mental treatment, used Cullen's nosology as the basis for his "scientific" teachings. In contrast to most of his colleagues, Cullen became a popular educator because he refused to lecture in esoteric Latin and spoke in the vernacular.

In his first major work, the four-volume *First Lines of the Practice of Physick* (1777), Cullen made an effort to categorize all the then-known diseases (both psychological and physical) in line with the symptoms they displayed, the methods by which diagnoses were generated, and the therapy that might best be applied. Cullen was a notable pioneer of neuropathology and, in keeping with his orientation, believed that most pathological conditions of the mind should be attributed to diseases of the brain. Despite this orientation, he recognized that life experiences often influenced the character in which these biologically grounded diseases were expressed. Cullen proposed the term "neuroses" to represent neurologically based diseases. Most etiologically obscure mental illnesses were labeled neuroses, ostensibly to represent diseases of nerves that were inflamed and

irritable. As he perceived it, neuroses were affections of sense or motion that stemmed from a disharmony of the nervous system. Into the general category of neuroses, Cullen subcategorized four variants: those representing a diminution of voluntary motion, those representing a diminution of involuntary activity, those representing disturbances in the regular motions of the muscles or muscle fibers, and disorders of judgment.

Along with Cullen, ***Robert Whytt*** (1714–1766) played a large role in providing Scottish physicians of his day with a classification system of "neurotic" individuals. Cullen and Whytt proposed somewhat different schemas of mental disorders, although each adhered to a physiological grounding for these disturbances. Whytt attended to the less severe mental conditions of his time, categorizing them into three broad syndromes: hysteria, hypochondriasis, and nervous exhaustion—the last of which was subsequently referred to by ***George Beard*** (1839–1883) as neurasthenia. This classification does not deviate much from our current diagnostic manual, although Whytt's ideas were not based on detailed psychological observations. Whytt's basic theory was similar to Cullen's: He posited that disturbed motility within the nervous system produced nervous disorders. The selection of the term "neuroses" made good sense, as both Cullen and Whytt assumed that different sensibilities of the nerves could be the foundation upon which certain problematic behaviors might be based. This belief continued for at least another century, anticipating ideas that were explored in greater depth first by Charcot, and later by Janet and Freud.

John Haslam (1766–1844), a British psychiatrist, is perhaps best known for the diligence and astuteness of his clinical observations. As Zilboorg and Henry (1941) noted, "Through the sheer effort of keen observation of minute, seemingly unrelated details . . . and orderly arrangements of these details . . . a coherent clinical picture of the disease came to the fore" (p. 303). More careful than his predecessors, Haslam provided the first clinical description of various forms of paralysis, most notably general paresis. Alert to the epidemic of venereal disease that spread across Europe in the early 19th century, he wrote:

> A course of debauchery long persisted would probably terminate in paralysis . . . frequently induces derangement of mind. Paralytic affections are a much more frequent cause of insanity than has been commonly supposed, and they are also a very common effect of madness; more maniacs die of hemiplegia and apoplexy than from any other disease. (1809, p. 209)

Also of great significance was Haslam's recognition that states of excitement and depression alternated in the same individual—an observation recorded by Aretaeus 17 centuries earlier. Importantly, it also recognized the significance of the course of a disease as a factor in classifying mental syndromes, thereby laying the groundwork for Kraepelin's central rationale for his nosological model almost a century later. In his 1809 book, *Observations on Madness and Melancholy*, Haslam described a number of cases that would subsequently be classified as dementia praecox or schizophrenia. In the following year, he published an innovative text, *Illustrations of Madness*, which presented a detailed examination of an individual with diverse paranoid features.

No less significant was Haslam's sophistication in matters of nomenclature and semantics. In his 1809 text he wrote:

> Madness is therefore not a complex idea, as has been supposed, but a complex term for all the forms and varieties of this disease. Our language has been enriched with other terms expressive of this affliction. . . . Instead of endeavoring to discover an infallible definition of madness, which I believe will be found impossible, [I will] attempt to comprise, in a few words, the wide range and mutable character of a Proteus disorder. (1809, pp. 5–6)

Note should be made in this chronological sequence of the important contributions of ***Jean Esquirol*** (1772–1840), the great humanistic reformer and associate of Philippe Pinel. Among Esquirol's diagnostic proposals was the attention he gave to a patient's dispositions and deficits of affect and impulse in his concept of *lypemanie*, by which he meant a deficiency in the capacity to feel or desire—a feature seen in patients whom many would speak of today as depressed. Esquirol grouped the several variants of mental disorder into five broad classification syndromes: *lypemanie, monomanie, manie*,

dementia, and imbecility/idiocy—a series of distinctions utilized in France for over a century. Esquirol also made significant contributions to the clarification of delusions and hallucinations. He wrote:

> In hallucinations there is no more sensation or perception than in dreaming or somnambulism, when no external object is stimulating the senses. . . . In fact, [a] hallucination is a cerebral or psychological phenomenon that takes place independently from the senses. The pretended sensations of the hallucinated are images and ideas reproduced by memory, improved by the imagination, and personified by habit. (1838, pp. 191–192)

His description of a hallucination as essentially a variant of a delusion differentiated it from simple sensory errors such as illusions and brought it into the realm of the patient's personality dysfunctions.

Also notable were the contributions of ***Jean-Pierre Falret*** (1794–1870), another humane reformer and student of Esquirol, who articulated notions similar to his mentor's regarding delusions. He specified several factors instrumental in their formation—notably, the state of the brain, the character of the patient, the circumstances surrounding the time the delusion began, and concurrent internal and external sensations. He expressed his conception of delusions as follows:

> Delusions may reflect the most intimate preoccupations and emotions of the individual. Indeed, the features of delusions may help us recognize what aspects of the subject's organization are suffering the most. Practitioners should give attention to relationships between delusions and the character of the subject. (1862, p. 357)

Falret also contributed an early and insightful series of papers that further detailed the variable character of mania and melancholy, which he called *forme circulaire de maladie mentale*, consisting of periods of excitation followed by longer periods of weakness. Presenting this theme as a facet of his 1851 lectures at the Salpêtrière Hospital, he subsequently elaborated these views in a book published in 1854; similar ideas were proposed almost concurrently by Jules Baillarger.

A series of novel classifications also gained prominence in Germany. They were based on a threefold distinction among the "faculties of the mind" (volition, intellection, and emotion), as well as a number of "morbid" processes (e.g., exaltation and depression). Among the early promoters of this schema was ***Johann Christian Heinroth*** (1773–1843)—perhaps the first physician to occupy a chair in psychiatry, that at Leipzig University in 1811. He subdivided one of the major categories of mental disorder, *vesania*, into several orders, genera, and species. Designing a complex matrix combining the major faculties on one dimension with the morbid processes on the other, he proposed a classification system comprising subtypes that became the basis of several variations throughout Germany and England in the ensuing century. Heinroth also developed a theory of mind with a tripartite structure. The basic or undergirding layer was characterized by the animalistic instinctual qualities of human beings; the intermediary layer reflected consciousness, including both intelligence and self-awareness; and, finally, a superior layer consisted of what we would call conscience. Presaging ideas proposed later by Freud, Heinroth also proposed the notion of conflict when two layers became opposing forces—such as the instinctual impulses of sin on the one hand, and the conscience's sense of moral correctness on the other.

Especially insightful was Heinroth's (1818) recognition of the significance of the patient's affect, or passions. He specified these insights in the following passage:

> The origin of the false notions in patients are erroneously attributed to the intellect. The intellect is not at fault; it is the disposition which is seized by some depressing passion, and then has to follow it, and since this passion becomes the dominating element, the intellect is forced by the disposition to retain certain ideas and concepts. But it is not these ideas or concepts which determine the nature of the disease.

Heinroth recognized a deep connection between the human qualities of mind and the more fundamental vegetative or animal passions that are fundamental to mental disorders, notably those of melancholy and rage. Heinroth also conceived of a term akin to what today we call "psychosomatics," in

which he took exception to Descartes's contention of a dualism between mind and body. In his view, health reflected harmony between these two components when they acted as a singular entity. He not only recognized a unity between mind and body, but considered that each person was composed of the same elements that made up the rest of nature.

Heinroth traced the term "paranoia" some 2,000 years back in the medical literature. The word had disappeared from the medical lexicon in the 2nd century B.C. and was not revived until Heinroth, following the structure of Kantian psychology, employed the term in 1818 to represent a variety of disorders. He termed disturbances of the intellect "paranoia"; he called disturbances of feeling "paranoia ecstasia." He also proposed the parallel concepts of *Wahnsinn* and *Verrucktheit* (the latter term is still in use as a label for paranoia in modern-day Germany). Griesinger, to be discussed shortly, picked up the term *Wahnsinn* in 1845 to signify pathological thought processes and applied it to cases of expansive and grandiose delusions. In 1863, Kahlbaum, also discussed later in this chapter, suggested that paranoia be the exclusive label for delusional states.

British alienist ***James Cowles Prichard*** (1786–1848), credited by many as the first to formulate the concept of "moral insanity," was in fact preceded in this realization by several theorists; nevertheless, he was the first to label it as such and to give it wide readership in English-speaking nations. Although he accepted Pinel's notion of *manie sans délire*, he dissented from Pinel's morally neutral attitude toward these disorders and became the major exponent of the view that these behaviors signified a reprehensible defect in character that deserved social condemnation. He also broadened the scope of the original syndrome by including under the label "moral insanity" a wide range of previously diverse mental and emotional conditions. All of these patients ostensibly shared a common defect in the power to guide themselves in accord with "natural feelings"—that is, a spontaneous and intrinsic sense of rightness, goodness, and responsibility. In Prichard's opinion, those afflicted by this disease were swayed, despite their ability to intellectually understand the choices before them, by overpowering "affections" that compelled them to engage in socially repugnant behaviors.

A major figure in extending the ideas of Esquirol and Falret at the Salpêtrière Hospital, ***Felix Voisin*** (1794–1872) was also a strong adherent of the phrenological speculations of Gall. His particular expertise was related to the linkage between the brain and the sexual organs; he stressed the importance of the nervous system as causally involved in generating various disorders of sexual desire. Placing special attention on the pathologies of nymphomania and satyriasis, especially as they were related to hysteria, Voisin articulated a progression in these disorders from their early stages to their more severe forms, contributing to the idea that disease *course* was central to clinical diagnostics.

In his major work, *The Analysis of Human Understanding* (1851), Voisin specified three major faculties of human functioning: moral, intellectual, and animal. This division predated and paralleled Freud's subsequent formulation of the mind's structure of superego, ego, and id. Also notable was Voisin's contribution to the moral treatment of persons with mental retardation at the Bicêtre Hospital. Influenced by Prichard, Voisin delved briefly in his later years into the problems of criminal and forensic pathology, speaking of criminals as products of lower-class origins and of their inevitable moral degeneration—a theme addressed elsewhere by Cesar Lombroso and Benedict Morel.

Another contributor to French thinking of the day was ***Paul Briquet*** (1796–1881), who focused primarily on problems of hysteria and their ostensive connection to female maladies. In his extensive monograph, *Traite Clinique et Therapeutique a l'Hystérie* (1859), he took exception to the notion posited by Plato and Hippocrates that hysteria was a consequence of sexual incontinence. Briquet specified with great clarity the multiple, exaggerated gastrointestinal, sexual, and other complaints that typified the symptoms presented by his "hysterical" patients. Such symptoms are labeled "somatization disorder" in official nosologies today, as well as occasionally referred to as "Briquet's syndrome." He recorded, in contrast to prior beliefs, that married women were no more inclined to hysteria than were unmarried women; that numerous cases appeared before puberty; and, most significantly, that an

active sexual life was no assurance that one would not develop such symptoms.

Going beyond the assumptions of many of his contemporaries, Briquet rejected the view that men could not develop symptoms of hysteria. He also pointed to numerous psychological influences that often contributed to the symptomatological expression of the disorder, noting painful emotional states (such as sadness and fear) as elements in precipitating the syndrome. Moreover, he speculated on a variety of untoward developmental and life experiences as playing a pathogenic role (e.g., parental mistreatment, spousal abuse, unfavorable employment circumstances or business failures). Recognizing that only a small subset of those subjected to these psychosocial experiences developed the hysterical syndrome, Briquet proposed the concept of "predispositions" as pathogenic factors. Aware that life circumstances often troubled his patients, he suggested that many would benefit from speaking to an empathic counselor or physician who might serve as a confidant. Briquet showed great sensitivity in going beyond the crude medications of the day to employ a psychotherapeutic approach to his patients' difficulties.

Ernst von Feuchtersleben (1806–1849) may have been the first Austrian psychiatrist to gain a distinguished status in European circles during the mid-19th century. His one major publication, *The Principles of Medical Psychology*, published in 1847, probably had a significant influence on Freud and his many disciples in Vienna. A strong critic of those who supported the Cartesian mind–body dichotomy, Feuchtersleben (like many in the 20th-century psychosomatic movement) considered the mind and the body to be a unitary phenomenon, essentially indivisible. An exponent of the role of personality qualities in the life of mental patients, Feuchtersleben wrote with great sensitivity on the psychic sources of mental disorders. In describing those inclined to the development of depressive diseases, Feuchtersleben said:

> Here the senses, memory, and reaction give way, the nervous vitality languishes at its root, and the vitality of the blood, deprived of this stimulant, is languid in all its functions. Hence the slow and often difficult respiration, and proneness to sighing. . . . When they are chronic, they deeply affect vegetative life, and the body wastes away. (1847, p. 135)

Moreover, in what may have been the first purely psychological description of what is now referred to as histrionic personality disorder, Feuchtersleben depicted women disposed to hysterical symptoms as being sexually heightened, selfish, and "over-privileged with satiety and boredom." Attributing these traits to the unfortunate nature of female education, he wrote: "It combines everything that can heighten sensibility, weaken spontaneity, give a preponderance to the sexual sphere, and sanction the feelings and impulse that relate to it" (1847, p. 111). Chauvinistic as this judgment may be regarded today, Feuchtersleben at least recognized and was sensitive to the limitations Victorian society placed upon women in his time. Moreover, he asserted the important role that psychological factors could play in helping patients understand the origins of their difficulties. He also espoused a hopeful therapeutic attitude and recommended opportunities for patients to acquire a second education in life.

As noted earlier in this chapter, the great English neurologist Thomas Willis (1664/1978) reported having observed a pathological sequence in which "young persons who, lively and spirited, and at times even brilliant in their childhood, passed into obtuseness and hebetude during adolescence." Better known historically, however, are the texts of Belgian psychiatrist ***Benedict-Augustin Morel*** (1809–1873), who described the case of a 14-year-old boy who had been a cheerful and good student, but who progressively lost his intellectual capacities and increasingly became melancholy and withdrawn. Morel considered such cases to be irremediable and ascribed the deterioration to an arrest in brain development that stemmed from hereditary causes. He named the illness "dementia praecox" (*demence precoce*), to signify his observation that a degenerative process began at an early age and progressed rapidly.

After Morel became chief physician at St. Yon Asylum in 1856, he continued to lecture and write on the inevitable sequence of deterioration, which he considered to be an inexorable course in all mental disorders.

He judged this "incessant progression" of degeneration to be human destiny. Speaking of those subjected to hereditary mental disorders, he wrote:

> The degenerate human being, if he is abandoned to himself, falls into a progressive degradation. He becomes . . . not only incapable of forming part of the chain of transmission of progress in human society, he is the greatest obstacle to this progress through his contact with the healthy portion of the population. (1857, p. 46)

Although his work secured him a niche in the history of psychiatry, Morel's views contributed to the pessimistic attitude regarding mental illness that was then pervasive in the European public at large—a view that unfortunately gained a horrendous following a century later in Nazi Germany.

In 1854, ***Jules Baillarger*** (1809–1892) and Jean-Pierre Falret summarized the results of their independent work with depressed and suicidal persons. They reported that a large proportion of these patients showed a course of extended depression, broken intermittently by periods of irritability, anger, elation, and normality. The terms *la folie circulaire* (Falret, 1854) and *folie à double forme* (Baillarger, 1853) were applied to signify this syndrome's contrasting and variable character. Baillarger contributed to a wide range of psychopathological conditions beyond the syndrome known today as bipolar disorder, notably in his ideas on hallucinations and delusions, neurohistology, epilepsy, and general paralyses. With regard to delusions, he sought to describe the perceptual basis of this disorder by stating that delusions were based on false interpretations of normal sensations, whereas illusions were distortions at the sensory rather than the ideational level. Similarly, he explored the question of whether hallucinations were sensory or psychological phenomena. He proposed two types: psychosensory hallucinations, which stemmed from the interaction of both sensory and imaginal distortions, and psychological hallucinations, which were independent of any sensory involvement.

Although born and educated in Germany, ***Richard von Krafft-Ebing*** (1810–1874) became a close follower of Morel, whose concept of degeneration struck a resonant chord in his work and practice at the Illenau Asylum in Baden. Krafft-Ebing was convinced that the Morelian process of degeneration was the primary cause not only of mental disorders, but also of criminality and sexual pathology. He wrote: "Madness, when it finally breaks out, represents only the last link in the psychopathic chain of constitutional heredity, or degenerate heredity" (1879, p. 439). Moving to Graz, Austria, he became a professor of psychiatry at the university there and the director of its provincial asylum. In his major work, *Lehrbuch der Psychiatrie* (1879), he referred to the problem of progressive sexual degeneration as follows: "It is specially frequent for sexual functioning to be . . . abnormally strong, manifesting itself explosively and seeking satisfaction impulsively, or abnormally early, stirring already in early childhood and leading to masturbation" (p. 424). By the mid-1880s, Krafft-Ebing assumed the chair at the University of Vienna and wrote his most famous book, entitled *Psychopathia Sexualis* (1882/1937), in which he spoke of the pervasive pathology of all variants of sexual activity (i.e., those differing from the approved and "proper" behavior of Victorian times).

The label "masochism" was proposed by Krafft-Ebing as a new concept in his catalog of sexual perversions. In a manner similar to the creation of "sadism" from the name of the Marquis de Sade, the "masochism" label was created from the name of a well-known writer of the time, Leopold von Sacher-Masoch. In Sacher-Masoch's novel *Venus in Furs* (1870), the hero suffers torture, subjugation, and verbal abuse from a female tormentor. Krafft-Ebing asserted that flagellation and physical punishment were necessary elements in the perversion, but were less significant than a personal relationship that included enslavement, passivity, and psychological serfdom. Hence, from its first formulations, the concept of masochism (although centrally sexual in nature) included the need to experience suffering, not just physical pain.

The growth of knowledge in anatomy and physiology in the mid-18th century strengthened the trend toward organically oriented disease classifications. ***Wilhelm Griesinger*** (1817–1868; see Figure 1.3), a young German psychiatrist with little direct patient experience, asserted the disease concept in his

FIGURE 1.3. Wilhelm Griesinger.

classic text *Mental Pathology and Therapeutics*, published in 1845 when he was barely 28 years of age. His statement "Mental diseases are brain diseases" shaped the course of German systematic psychiatry for the next 40 years. Griesinger's contention that classifications should be formed on the basis of underlying brain lesions was not weakened by the fact that no relationship had yet been established between brain pathology and mental disorders. In fact, Griesinger's own system of categories—depression, exaltation, and weakness—did not parallel his views regarding the importance of brain pathology. Nevertheless, he convinced succeeding generations of German neurologists, led by Thomas Meynart and Carl Wernicke, that brain diseases would be found to underlie all mental disturbances.

Griesinger was born in Stuttgart, Germany, and completed his medical studies in Zurich and Tübingen. There he learned to view medicine as a science based on the direct observation of patient experiences and behaviors rather than on historical speculations and philosophy. He began his formal career in psychiatry at the Winnenthal Asylum in Stuttgart. Assuming that he had gathered sufficient expertise in a 3-year span, he penned his classic 1845 text. To him, the study of mental illness was integral to the study of general medicine. He conceived of mental disorders as chronically progressive, like most medical diseases. Thus he regarded depression as beginning with a minor level of cerebral irritation, leading next to a chronic and irreversible degeneration, and ending ultimately in pervasive dementia—a path of deterioration that became a central theme of Kraepelin's belief that the course of a mental disorder was its most crucial characteristic.

It was not until 1861 that Griesinger revised his 1845 text, following which he returned to his work in psychiatry at the University of Berlin. Here he both lectured and practiced at its Charité Clinic, where he divided his patients into those with routine nervous diseases and those with nervous diseases that also exhibited psychiatric symptoms. He also initiated and assumed the editorship of a new journal, the *Archives for Psychiatry and Nervous Diseases*. In its first volume, Griesinger wrote:

> Psychiatry has undergone a transformation in its relationship to the rest of medicine. . . . This transformation rests principally on the realization that patients with so-called mental diseases are really individuals with diseases of the nerves and the brain. . . . Psychiatry . . . must become an integral part of general medicine and accessible to all medical circles. (1868, p. 12)

Although the work of Griesinger and his followers regarding the role of the brain in mental disorders soon dominated continental psychiatry, a different emphasis regarding the basis of classification was developing concurrently. Jean Esquirol, Pinel's distinguished associate, had often referred to the importance of age of onset, variable chronicity, and deteriorating course in understanding pathology. This idea was included as a formal part of classification in 1856 when German psychiatrist ***Karl Ludwig Kahlbaum*** (1828–1899) extended Esquirol's idea by developing a classification system in which disorders were grouped according to their course and outcome. It became the major alternative system to the one Griesinger proposed. Kraepelin, noting his indebtedness to Kahlbaum's contributions, stated that "identical or remarkably similar symptoms can accompany wholly dissimilar diseases while their inner nature can be revealed only through their progress and termination" (Kraepelin, 1920, p. 116).

Kahlbaum wrote of how useless attempts had been to group disorders on the basis of the similarity of their overt symptomatology, as if such superficial symptom collections would themselves expose something essential concerning the underlying diseases. He commented as follows:

> It is futile to search for the anatomy of melancholy or mania, because each of these forms occurs under the most varied relationships and combinations with other states, and they are just as little the expression of an inner pathological process as the complex of symptoms we call fever. (1874, p. 2)

Kahlbaum turned his attention in 1857 to psychoses that were typical of young adolescents, focusing on the sudden emergence of mental disorientation and rapid disintegration—a pattern not unlike that described by Morel a decade or two earlier. Similarly, reading the work of Falret and of Jules Baillarger, he also directed his attention to the problems of patients whose mood disorders appeared to follow a sequential course from mania to depression and back.

In a series of monographs and books published between 1863 and 1874, Kahlbaum not only established the importance of including longitudinal factors in psychiatric diagnosis, but described newly observed disorders that he labeled "hebephrenia" and "catatonia," as well as coining the modern terms "symptom complex" and "cyclothymia." Kahlbaum, together with his disciple Ewald Hecker, introduced the term "hebephrenia" to represent conditions that began in adolescence, usually starting with a quick succession of erratic moods, followed by a rapid enfeeblement of all functions, and finally progressing to an unalterable psychic decline. The label "catatonia" was introduced to represent "tension insanity" in cases where the patient displayed no reactivity to sensory impressions, lacked "self-will," and sat mute and physically immobile. These symptoms ostensibly reflected deterioration in brain structure.

It was Kahlbaum also who, in 1882, clearly imprinted current thinking on the fixed covariation of mania and melancholia, known today as bipolar disorder. Although he regarded them as facets of a single disease, which he termed "dysthymia" (following a label introduced two decades earlier by Carl Flemming), the disease actually manifested itself in different ways at different times—occasionally euphoric, occasionally melancholic, and occasionally excitable or angry. It was the primacy of the former two emotions that rigidified future conceptions of the syndrome and redirected thinking away from its more typical affective instability and unpredictability. He termed a milder variant of the illness, notable for its frequent periods of normality, "cyclothymia." A more severe and chronic form of the same pattern was designated by Kahlbaum as *vesania typica circularis*.

Henry Maudsley (1835–1918) was admitted to London's University College at age 15; here he proved to be a brilliant student, completing his medical degree at age 21. Unsure about his future and lacking the means to follow an early interest in surgery, he entered the East India Company's service, spending the better part of a year as medical officer at the Wakefield Mental Asylum. Owing to his high intelligence and vigorous appearance, at age 23 he was appointed medical superintendent of the Cheadle Royal Hospital, despite a total lack of administrative experience or formal psychiatric training. Shortly thereafter, he became superintendent of the newly opened Manchester Royal Lunatic Hospital. His fame grew throughout England, and at age 27 he became the editor of the country's major psychiatric publication, the *Journal of Mental Science*. He was appointed to a professorship at the University College Hospital in 1870.

In his major text, *Physiology and Pathology of Mind* (1876), Maudsley attempted to redirect the philosophical inclinations typical of British clinicians and sought to anchor the subject more solidly within the biological sciences. He vigorously asserted that mind and body comprised a unified organism, "each part of which stirs the furthest components, [and] which then acts upon the rest and is then reacted on by it. . . . Emotions affect every part of the body and [are] rooted in the unity of organic life." He also wrote, consistent with comparable views expressed by Griesinger in Germany, that "mental disorders are neither more nor less than nervous diseases in which mental symptoms predominate" (1876, p. 41). Despite this view, Maudsley had asserted earlier

> that there is no boundary line between sanity and insanity; and the slightly exaggerated feeling which renders a man "peculiar" in the world differs only in degree from that which places hundreds in an asylum. . . . Where hereditary predisposition exists, a cause so slight as to be inappreciable to observers is often efficient to produce the disease. (1860, p. 14)

The Japanese first met with Europeans in the middle of the 16th century, but they were highly ambivalent toward European influences and remained isolated until the latter half of the 19th century. Western medicine (including Western psychiatry in Britain and Germany) was introduced, and Japanese psychiatry became strongly organically oriented. The writings of Maudsley, who viewed insanity as a bodily disease, and of Griesinger, whose approach has been described as "psychiatry without psychology," were among the most important influences. ***Shuzo Kure***'s (1865–1932) visit to Europe brought back not only the ideas behind Kraepelin's descriptive psychiatry, but also the emerging interest in psychoneuroses and psychotherapy. Also introduced was the term "neurasthenia," in which a wide variety of bodily symptoms were explained as exhaustion of the central nervous system under the influence of physical and social stressors.

Kure's pupil ***Shoma Morita*** (1874–1938), having personal experiences with neurasthenia, developed a psychogenic theory and treatment of neurosis. Subsequently labeled "Morita therapy," his approach deserves a closer description as an example of how Western and Eastern thinking met. (See Goddard, 1991, for a full discussion.) Morita translated Binswanger's work *Fundamentals of Treatment for Mental Illness*; Binswanger recommended a strictly regulated 5-week timetable for "life normalization," which included intellectual and manual activities. Morita was also influenced by American neurologist ***Silas Weir Mitchell*** (1829–1914), who invented a regimen of bed rest, isolation, rich diet, massage, and electrostimulation for neurasthenia, and by neuropathologist and psychotherapist ***Paul Charles Dubois*** (1848–1918), who believed that the therapist's task was to convince the patient that his or her neurotic feelings, thoughts, and behaviors were irrational. By integrating these ideas of Western scientists with thoughts from Zen Buddhism, Morita developed Morita therapy, which for a century has been widely used not only in Japan, but also in China and in some Western societies.

Morita's term for neurosis was *shinkeishitsu*. Everyone is born with *sei no yokubo*, the desire to live, but this drive may be hindered by oversensitivity to oneself and one's limitations. Morita did not regard patients as sick persons, but as healthy persons obsessed by their own anxieties and fears. He described three different kinds of *shinkeishitsu*: (1) the ordinary type, which resembles what is now labeled somatization disorder; (2) the obsessive/phobic type or *taijin kyofusho*, which includes symptoms of present-day agoraphobia, specific phobia, social phobia, and obsessive–compulsive disorder; and (3) the paroxymal neurosis type, which includes symptoms of agoraphobia and generalized anxiety disorder. Morita therapy followed a strict time schedule, starting with complete isolation in a private, familial, homelike room to give a feeling of security for a week; this was followed by light activities and the keeping of a diary, which each patient discussed with a therapist. Then followed a stage of work such as gardening, and finally a stage where the patients were turning toward realities with their families and society. The goal for the treatment was not necessarily the disappearance of symptoms, but the ability to function normally and productively despite the symptoms. Morita endorsed the concept of *arugamama*, meaning "things are as they are," which was the mental attitude patients were encouraged to show toward their symptoms. The emphases on body–mind–nature monoism, affirming and accepting worldly passion and desires, and the practice of daily life were clearly borrowed from Japanese Shintoism, Zen Buddhism, and Asian psychology, but they also owed much to Japanese cultural patterns (e.g., the meaning of and devotion to work, the acceptance of reality, persistence, and dependency).

Throughout the 19th century, German psychiatrists abandoned what they considered to be the value-laden theories of the French and English alienists of the time and toward what they judged to be empirical or observational research. Among this group was ***J. A. Koch*** (1841–1908), who

proposed that the label "moral insanity" be replaced by the term "psychopathic inferiority," which included "all mental irregularities whether congenital or acquired which influence a man in his personal life and cause him, even in the most favorable cases, to seem not fully in possession of normal mental capacity" (1891, p. 67). Koch used the word "psychopathic"—a generic label employed to characterize all personality diagnoses until recent decades—to signify his belief that a physical basis existed for these character impairments. Thus he stated: "They always remain psychopathic in that they are caused by organic states and changes which are beyond the limits of physiological normality. They stem from a congenital or acquired inferiority of brain constitution" (1891, p. 54).

Descriptive Psychopathology in the 20th Century

Kraepelin's comprehensive textbooks at the turn of the 20th century served as one of psychiatry's two major sources of inspiration; the other consisted of Freud's innovative psychoanalytic contributions. As the preeminent German systematist, ***Emil Kraepelin*** (1856–1926; see Figure 1.4) bridged the diverse views and observations of Greisinger and Kahlbaum in his outstanding texts, revised from a small compendium in 1883 to an imposing four-volume eighth edition in 1913. Kraepelin constructed a system that integrated Kahlbaum's descriptive and longitudinal approach with Greisinger's somatic disease view. By sifting and sorting prodigious numbers of well-documented hospital records, and directly observing the varied characteristics of patients, he sought to bring order to symptom pictures and, most importantly, to patterns of onset, course, and outcome. Kraepelin felt that syndromes based on these sequences would be best in leading to accurate identification and distinction among the different conditions that differentiated and caused these disorders. Psychiatric historian Ray Porter (2002) has summarized this contribution of Kraepelin as follows:

> He approached his patients as symptom-carriers, and his case histories concentrated on the core signs of each disorder. The course of psychiatric illness, he insisted, offered the best clue to its nature. . . . Kraepelin's commitment to the natural history of mental disorders led him to track the entire life histories of his patients in a longitudinal perspective which privileged prognosis (likely outcome) as definitive of the disorder. (pp. 184–185)

Kraepelin was born in Germany in the same year as Sigmund Freud. A serious and diligent student, Kraepelin was exposed in medical school to several professors who were instrumental in shaping his style of thinking and research for the rest of his career. Most notable among these was Wilhelm Wundt, the founder of experimental psychology. Wundt himself had been trained by Hermann von Helmholtz, the great physiological theorist. Owing to visual difficulties that deterred him from research with microscopes, Kraepelin began to pursue psychological research, becoming one of Wundt's most distinguished students. Nevertheless, Wundt advised him to pursue medicine rather than psychology, which was then a fledgling science with limited career opportunities. In 1882, Kraepelin began the initial drafts of his first textbook, which later became the standard for educating psychiatrists.

His first text, a 300-page volume titled *Compendium of Psychiatry*, was so successful that it led to several subsequent editions published under the general title *Short Text-*

FIGURE 1.4. Emil Kraepelin.

book of Psychiatry. By the sixth edition of what he subsequently called his *Lehrbuch* or *Textbook of Psychiatry* at the turn of the century, Kraepelin was known throughout the Continent and the English-speaking world. In 1904, he became chairman of the Psychiatric Clinic and Laboratory at the University of Munich—a distinguished department where he was able to bring along with him from Heidelberg such promising young researchers as Alois Alzheimer and Franz Nissl, both already known for their excellent neurohistological studies. At the time of his death in 1926 at age 70, Kraepelin was actively working on a ninth edition of his textbook, which had expanded to four volumes and more than 3,000 pages.

Kraepelin did not set out initially to create the nosology for which he became so famous. Although he proposed a series of revolutionary ideas concerning the nature of clinical syndromes, the astuteness of his observations and the clarity of his writing were what proved to be central to the success of his work. Kraepelin wrote very little about how classification should be organized; that is, he utilized no formal set of principles to rationalize how a nosology should be structured. It was the implicit structure of his books (i.e., their basic table of contents) that served as his classification system. Not to be dismissed was the logic that he presented for organizing syndromes on the basis of clinical symptomatology, course, and outcome. Perhaps it was the input of his mentor Wundt's keen observation and analysis of the behavior of his subjects in his research studies that taught him to provide such richly descriptive characterizations of his patients. Moreover, Kraepelin focused on the overt psychological manifestations of mental disorders, in contrast to his more organically and physiologically oriented contemporaries. The following paragraphs touch on only a few of his conceptions regarding the major forms of psychoses and the syndromes now termed "personality disorders."

Kraepelin constantly revised his diagnostic system, elaborating it at times, simplifying it at others. In the sixth edition of 1899, he established the definitive pattern of two modern major disorders: "manic–depressive psychosis" (now known as bipolar disorder) and "dementia praecox" (now known as schizophrenic disorders). These were clinically vivid syntheses of previously independent concepts that Morel and Kahlbaum had formulated. Within the manic–depressive group, he brought together the excited conditions of mania and the hopeless melancholia of depression, indicating the periodic course through which these moods alternated in the same patient. To be consistent with his disease orientation, he proposed that this disorder was caused by an irregular metabolic function transmitted by heredity.

As recorded previously, many of Kraepelin's predecessors viewed mania and melancholia as a single disease that manifested itself in different forms and combinations over time. Kraepelin borrowed heavily from these formulations, but separated the "personality" and "temperament" variants of the disorder from the clinical state of the disease. Nevertheless, in the fifth edition of his text, he proposed the name "maniacal–depressive insanity" for "the whole domain of periodic and circular insanity"; it included such diverse disturbances as "the morbid states termed melancholia and certain slight colorings of mood, some of them periodic, some of them continuously morbid" (1896, p. 161). Like Kahlbaum, Kraepelin viewed "circular insanity" as a unitary illness. Moreover, he believed that every disorder that featured mood disturbances—however regular or irregular and whatever the predominant affect, be it irritability, depression, or mania—was a variant or "rudiment" of the same basic impairment. To Kraepelin, the common denominator for these disturbances was an endogenous metabolic dysfunction that was "to an astonishing degree independent of external influences" (1896, p. 173).

Four varieties of the cyclothymic disposition identified by Kraepelin were termed "hypomanic," "depressive," "irascible," and "emotionally unstable." He described the hypomanic type as follows:

> They acquire, as a rule, but scant education, with gaps and unevenness, as they show no perseverance in their studies, are disinclined to make an effort, and seek all sorts of ways to escape from the constraints of a systematic mental culture. The emotional tone of these patients is persistently elated, carefree, self-confident. Toward others they are overbearing, arbitrary, impatient, insolent, defiant. They mix into everything, overstep their prerogatives, make unauthorized arrangements,

> as they prove themselves everywhere useless. (1913, p. 221)

In describing the depressive personality type, Kraepelin (1921) wrote:

> There are certain temperaments which may be regarded as rudiments of *manic–depressive* insanity. They may throughout the whole of life exist as peculiar forms of psychic personality, without further development; but they may also become the point of departure for a morbid process which develops under peculiar conditions and runs its course in isolated attacks. Not at all infrequently, moreover, the permanent divergencies are already in themselves so considerable that they also extend into the domain of the morbid without the appearance of more severe, delimited attacks. (p. 118)

Typically, Kraepelin considered this type to be characterized by an inborn temperamental predisposition to "a permanent gloomy emotional stress in all experiences in life" (p. 118). According to him, "the morbid picture is usually perceptible already in youth, and may persist without essential change throughout life" (p. 123).

The irascible type was ostensibly endowed simultaneously with both hypomanic and depressive inclinations. According to Kraepelin, "They are easily offended, hot-headed, and on trivial occasions become enraged and give way to boundless outbursts of energy. Ordinarily the patients are, perhaps, serene, self-assertive, ill-controlled; periods, however, intervene in which they are cross and sullen" (1921, p. 222). The emotionally unstable variant presumably also possessed both hypomanic and depressive dispositions, but manifested them in an alternating (or, as Kraepelin viewed it, true cyclothymic) pattern. He described these patients as follows:

> It is seen in those persons who constantly swing back and forth between the two opposite poles of emotion, now shouting with joy to heaven, now grieved to death. Today lively, sparkling, radiant, full of the joy of life, enterprise, they meet us after a while depressed, listless, dejected, only to show again several months later the former liveliness and elasticity. (1921, p. 222)

Kraepelin had considered hebephrenia, the diagnosis of adolescent psychosis, and dementia praecox to be synonymous prior to the sixth edition of his psychiatric text. In his original treatise, he concluded that the diverse symptom complexes of catatonia and hebephrenia, as well as certain paranoid disturbances, displayed a common theme of early deterioration and ultimate incurability. As he conceived them, each of these illnesses was a variation on Morel's concept of dementia praecox. By subsuming the disparate symptoms of these formerly separate syndromes under the common theme of their ostensible early and inexorable mental decline, Kraepelin brought a measure of order and simplicity to what had previously been diagnostic confusion. In line with the traditions of German psychiatry, Kraepelin assumed that a biophysical defect lay at the heart of this new coordinated syndrome. In contrast to his forebears, however, he speculated that sexual and metabolic dysfunctions were the probable causal agents, rather than the usual hypothesis of an anatomical lesion. Among the major signs that Kraepelin considered central to these illnesses, in addition to the progressive and inevitable decline, were discrepancies between thought and emotion; negativism and stereotyped behaviors; wandering or unconnected ideas; hallucinations and delusions; and a general mental deterioration.

Kraepelin believed that the "autistic" temperament served as the constitutional soil for the development of dementia praecox. Of particular note was Kraepelin's observation that children of this temperament frequently "exhibited a quiet, shy, retiring disposition, made no friendships, and lived only for themselves" (1921, p. 109). They were disinclined to be open and become involved with others, were seclusive, and had difficulty adapting to new situations. They showed little interest in what went on about them, often refrained from participating in games and other pleasures, seemed resistant to influence (but in a passive rather than active way), and were inclined to withdraw increasingly into a world of their own fantasies.

Among the "morbid" personalities, Kraepelin included a wide range of types disposed to criminal activities. As early as 1905, he identified four kinds of persons with features akin to what we speak of today as Cluster B personality disorders. First were the "morbid liars and swindlers," who were glib and

charming but lacked an inner morality and sense of responsibility to others. They made frequent use of aliases, were inclined to be fraudulent con artists, and often accumulated heavy debts that were invariably unpaid; this type proves to be descriptively similar to those we might classify today as having narcissistic personality disorder. The second group included "criminals by impulse"—individuals who engaged in crimes such as arson, rape, and kleptomania, and were driven by an inability to control their urges; they rarely sought material gains for their criminal actions. The third type, referred to as "professional criminals," was neither impulsive nor undisciplined; in fact, such persons often appeared well mannered and socially appropriate, but were inwardly calculating, manipulative, and self-serving. The fourth type consisted of the "morbid vagabonds," who were strongly disposed to wander through life, never taking firm root, lacking both self-confidence and the ability to undertake adult responsibilities.

Although less successful in influencing nosological thinking in the latter half of the 19th and early 20th centuries than Kraepelin, several other distinguished thinkers deserve recognition. ***Philippe Chaslin*** (1857–1923) was a great French theorist whose life's work overlapped with Bleuler's in Switzerland, Kraepelin's in Germany, and Freud's in Austria. A philosopher and linguist at heart, he spent the majority of his professional career at the Salpêtrière Hospital in Paris, where he wrote on a wide range of topics (including history, linguistics, and mathematics, as well as psychiatry). Among his central formulations was the concept of "discordance," a notion he used to describe and explain dementia praecox; Bleuler, who originated the term "schizophrenia" in his 1911 treatise on the subject, stated later that he might have preferred "discordant insanity" as an alternative label had he known of it earlier.

In his major work, *Elements de Seminologie et de Clinique Mentale*, written in 1912, Chaslin conveyed a series of ideas similar to those formulated concurrently by Freud, but with special reference to psychotic delusions. For example, he wrote:

> Delusional ideas seem to have their source in the emotions of the patient of which they are symbolic representations. . . . One could illustrate the origins of delusions by recollecting the mechanisms of dreaming. Propensities, desires, and feelings from the waking state reappear in dreams in symbolic scenes. (1912, p. 178)

Chaslin devoted much of his theoretical writing to articulating different variants of delusions and states of confusion. He spoke of the several ways in which delusions presented themselves—sometimes in isolation, sometimes combined with hallucinations; occasionally incoherent, but also at times systematic and logical, as in paranoid conditions. Regarding confusional states, Chaslin asserted that these temporary periods signified a loosening of intellectual, affective, and motivational functions; he concluded, for example, that the distinctions between confusion and dementia were modest and reflected an assumption that dementia possessed a chronic and deteriorating course.

Chaslin was also concerned, as were many philosophers of the day, with the failure of psychiatric language to adequately represent the nature of the disorders they diagnosed and treated. In describing the difficulties of psychopathological terminology, Chaslin exclaimed:

> I believe that the imprecision of terms is due to the imprecision of our ideas, but I also think that the inexactitude of a language may cause further inexactitude in our ideas. . . . If [the terminology] only helped to combat factual imprecisions, but the opposite is the case; it is often imagined that progress has been made simply because fancy names have been given to old things. (1912, p. 18)

Eugen Bleuler (1857–1939) is universally recognized for his description of what is presently known as "schizophrenia," the term he coined to replace the historic diagnostic label "dementia praecox." The label "schizophrenia" is now judged by many to be unfortunate, suggesting a splitting between segments of the mind—a concept then prevalent in French circles, and a notion Janet had proposed as an alternative to Freud's conception of three levels of consciousness. As evidence now indicates, patients diagnosed with schizophrenia do not suffer any form of splitting, but rather are characterized by disordered thinking leading to delusions and hallucinations.

In 1898 Bleuler took over the headship of the Burgholzli Mental Hospital, an already distinguished center for the clinical study of mental illness. Bleuler daily spent hours talking with his patients, often in their own unusual dialects, searching to gain an understanding of the psychological meaning of their seemingly senseless verbalizations and delusions. Most importantly, he urged his students and residents to be open-minded and to establish an emotional rapport with their patients; he believed that doing so would enable them to track the meaning of the words their patients used, as well as the word associations that might give meaning to their utterings. It was in this regard that he saw the utility of Freud's new free-association methods, and it was on these grounds also that he instilled an interest in his young associate, Carl G. Jung, in Freud's early psychoanalytic concepts.

Bleuler's studies of word associations led to his theory of schizophrenia. That is, the "loosening" or disintegration in patients' capacity to associate ideas and emotions reflected their ostensible inability to connect their thoughts with their feelings, and hence the presumed "split" between these two core psychic processes. Following upon ideas that were then emerging in the writings of both Freud and Janet, Bleuler asserted that his patients would display secondary symptoms that derived from the primary or fundamental thought–feeling disconnection—symptoms that evidenced themselves in an autistic separation from reality, in repetitive psychic ambivalences, and in verbal behaviors akin to dreaming. Although committed to Kraepelin's view that dementia praecox was primarily an organic disease, Bleuler emphasized the presence of psychological ambivalence and disharmony in this impairment, to signify the intellectual–emotional split he believed he observed in these patients.

Bleuler's conception of schizophrenia also encompassed a wider range of syndromes than Kraepelin's notion of dementia praecox. He included several acute disturbances that Kraepelin previously judged to be independent disease entities. Moreover, Bleuler believed that those displaying acute schizophrenic symptoms could recover readily with proper intensive care before their condition devolved into a more chronic state. Observing hundreds of patients diagnosed with dementia praecox in the early 1900s led Bleuler to conclude that it was misleading to compare the type of deterioration they evidenced with that found among patients suffering from metabolic deficiencies or brain degeneration. Moreover, he judged his patients' reactions and thoughts to be qualitatively complex and often highly creative, contrasting markedly with the simple or meandering thinking that Kraepelin observed. Furthermore, not only did many of his patients display their illness for the first time in adulthood rather than in adolescence, but a significant proportion evidenced *no* progressive deterioration, which Kraepelin considered the sine qua non of the syndrome. Thus Bleuler viewed the label "dementia praecox" as misleading, in that it characterized an age of onset and a course of development not supported by the evidence.

As noted, schizophrenia's primary symptoms, in Bleuler's view, were disturbances in the associative link among thoughts, a breach between affect and intellect, ambivalence toward the same objects, and an autistic detachment from reality. The several varieties of patients that displayed these fragmented thoughts, feelings, and actions led Bleuler to term their disorders "the group of schizophrenias." Nevertheless, he retained the Kraepelinian view that the basic impairment in these diverse disorders stemmed from a unitary disease process that was attributable to a basic physiological pathology. As he saw it, this shared neurological ailment produced their common primary symptoms. Bleuler ascribed the content of secondary symptoms to the patients' distinctive life experiences and to their efforts to adapt to their basic disease. Psychogenic factors shaped the unique character of each patient's impairment, but Bleuler was convinced that experience did not itself cause the ailment.

Bleuler recognized that some dispositions left untreated might ultimately evolve into a clinical schizophrenic state, which he termed *schizoidie*. In his initial formulation of the schizophrenia concept in 1911, he also provided one of the first portrayals that approximates what we now call avoidant personality disorder. Discussing several of the contrasting routes that often led to the psychotic syndrome, Bleuler recorded the early phase of certain patients as follows:

> There are also cases where the shutting off from the outside world is caused by contrary reasons. Particularly in the beginning of their illness, these patients quite consciously shun any contact with reality because their affects are so powerful that they must avoid everything which might arouse their emotions. The apathy toward the outer world is then a secondary one springing from a hypertrophied sensitivity. (1911/1950, p. 65)

Bleuler spoke of other personalities as being "irritable of mood"(*reizbare Verstimmung*), as Aschaffenburg (1922) did later in describing them as "dissatisfied personalities" who went through life as if they were perpetually wounded. Applying the label "amphithymia," Hellpach (1920) also depicted a similar pattern of "fussy people" who tended to be of a sour disposition, constantly fretted over whatever they did, and made invidious and painful comparisons between themselves and those of a more cheerful inclination (whose simpler and brighter outlook was both envied and decried).

Adolf Meyer (1866–1950), like Bleuler, was born in Switzerland. He completed his medical training in 1892 at the age of 26, following several predoctoral years in France, England, and Germany. A student of Forel at the University of Zurich, he decided to emigrate to the United States shortly after receiving his medical degree, having heard that Chicago was a city with numerous opportunities for young physicians. Meyer eventually served as a staff pathologist at the Illinois Eastern Hospital for the Insane, remaining there from 1893 to 1895. For the next 7 years, he was director of clinical research laboratories at the Worcester Insane Hospital and was associated with Clark University, both in Massachusetts. Increasingly recognized as a major contributor to neuropathology, as well as a lecturer known for his detailed history taking, interviews, and note taking, Meyer was appointed director of the New York Pathological Institute in 1902, as well as professor of psychiatry at Cornell University Medical School, where he continued autopsied brain research, teaching, and administrative activities until 1910. Along with Freud and Jung, he was awarded an honorary doctoral degree at Clark University in 1909. Owing to his distinguished achievements, Meyer later became chair of a new Department of Psychiatry and director of the Henry Phipps Psychiatric Clinic at Johns Hopkins University in Baltimore, where the aim was to blend scientific research and clinical practice. He remained at Johns Hopkins for over 30 years, building a German-style psychiatric clinic akin to Kraepelin's in Munich; in the process, he became the most influential psychiatrist in the United States and a mentor to an entire generation of both academic and clinical psychiatrists.

Meyer introduced the concept of a "constitutionally inferior" type into American literature at the turn of the century, shortly after his arrival from Germany. Although following Koch's ideas in the main, Meyer sought to separate psychopathic from psychoneurotic disorders, both of which were grouped together in Koch's "psychopathic inferiorities" classification. Meyer was convinced that the etiology of the neuroses was primarily psychogenic—that is, colored less by inherent physical defects or by constitutional inferiorities.

Meyer later became disillusioned with both Kraepelin's and Koch's approaches, particularly their fatalistic views of illness and their strictly deterministic prognosis and outcome for those of a problematic temperament. Meyer turned to a view increasingly shared by psychoanalysts—that is, discarding the disease model and viewing psychiatric disorders not as fundamentally organic conditions, but rather as consequences of environmental factors and life events. Although initially sympathetic to Freud's theories, Meyer soon became critical of the mystic and esoteric nature of psychoanalysis; despite his break from Freud's metapsychology, however, he shared Freud's view regarding the role of life experiences as central to the emergence of all psychiatric disorders.

As early as 1906, Meyer espoused the view that a true understanding of patients could be derived only by studying the individuals' total reaction to their organic, psychological, and social experiences. Although Meyer was the most prominent psychiatrist to introduce the Kraepelinian system in this country, he believed that these disorders were not disease entities, but "psychobiological reactions" to environmental stress. Through his work, Meyer bridged the physiological

orientation of the late 19th century and the psychodynamic orientation of the 20th.

For example, in 1912 Meyer asserted that dementia praecox was not an organic disease but a maladaptive way of reacting to stress, fully understandable in terms of a patient's constitutional potentials and life experiences. To him, these maladaptive reactions led to what he called "progressive habit deteriorations," which reflected "inefficient and faulty attempts to avoid difficulties" (1912, p. 98). He regarded symptoms of mental illness as the end products of abortive and self-defeating efforts to establish psychic equilibrium. His well-reasoned "psychobiological" approach to schizophrenia, which he called "parergasia" to signify its distorted or twisted character, was the most systematic recognition of his interactive and progressive view of the nature of pathogenesis. Of special note also was Meyer's view that parergasia could be present in dilute and nonpsychotic form—that is, without delusions, hallucinations, or deterioration. He considered the classic psychotic symptoms to be advanced signs of a potentially, but not inevitably, evolving habit system that might stabilize at a prepsychotic level. In its nonclinical state, parergasia could be detected from a variety of attenuated "soft signs" that merely suggested the manifest psychotic disorder. Meyer's proposal of a self-defeating and maladaptive reaction system (personality) that paralleled schizophrenia in inchoate form was a highly innovative, but unheeded, notion.

Karl Jaspers (1883–1969) was undoubtedly an influential pioneer of phenomenological and existential psychiatry, though, oddly enough, he did not consider himself a phenomenologist. His system of mental illness approached classification in a unique way; that is, it sought to describe each patient's true subjective experience and how he or she faced mental illness, rather than simply describing overt psychological syndromes as observed by the therapist. To this end, Jaspers made distinctions such as that between "feelings" and "sensations"; he described the former as emotional states of the individual, and the latter as part of the individual's reactions to and perceptions of the environment. The ultimate goal of this system was to enable the therapist to be as sensitive and empathic as possible with the patient. It was Jaspers's contention that the inexhaustibly infinite depth and uniqueness of any single individual—whether mentally ill or healthfully functioning—could not be completely understood and objectified, but that the medical/psychological practitioner must strive for as close an understanding as possible. This existential view of humankind was what set this system apart from the traditional means of diagnosis and treatment. In contrast with the psychoanalysts, who attempted to probe beneath the surface of patients' verbal reports to uncover their unconscious roots, Jaspers focused on patients' conscious self-description of feelings and experiences, believing that their phenomenological reports were the best routes to achieving a true understanding of their world.

Together, Meyer's notion of reaction types, Jaspers's existential phenomenology, and Bleuler's focus on cognitive and emotional experience reshaped Kraepelin's original system into a more contemporary psychiatric nosology. In their classifications, Kraepelin's clinical categories were retained as the basic framework, and Meyer's, Jaspers' and Bleuler's psychological notions provided guides to patients' inner processes and social reactions.

The Rise and Fall of 20th-Century Psychoanalytic Psychopathology

Many consider ***Jean-Martin Charcot*** (1825–1893; see Figure 1.5) the father of clinical neurology. Open-minded, deeply curious, and capable of observing subtle clinical details of his patients' behaviors, Charcot was an extraordinarily astute observer of physical defects and dysfunctions. Charcot was a senior physician at the Bicêtre, and later at the deteriorated Salpêtrière women's hospital, where Pinel had carried out his humane activities earlier in the century. In 1862, along with another young physician of exceptional ability, Edme F. A. Vulpain, Charcot studied the chronically ill women housed in its decaying wards. These two highly motivated and skilled physicians quickly recognized that more than half of those for whom they were responsible had been incorrectly diagnosed, most having been lumped indiscriminately into one or two categories.

Charcot's first discoveries were related to multiple sclerosis (MS), a significant neuro-

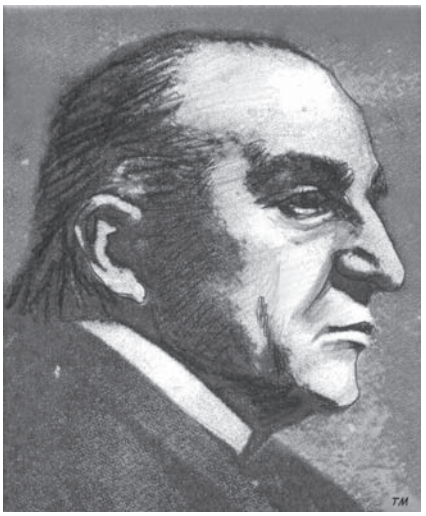


FIGURE 1.5. Jean-Martin Charcot.

logical disorder that was unrecognized as a distinct disease in the 1860s. Collaborating with Vulpain, he demonstrated the classic disintegration of the myelin sheath—the basic anatomical feature of the disorder. Also important was Charcot's recognition of the visual problems typical of those with MS, as well as his patients' tendency to exhibit extreme fluctuations in symptomological intensity over time. Another important contribution was his distinction between MS and the "shaking palsy," or what came to be called Parkinson's disease. Charcot identified features of the latter that Parkinson overlooked, such as patients' blank stares, motionless and stolid expressions, and periodic and involuntary oscillation of hand movements.

Owing to Charcot's distinguished work, the Salpêtrière was granted substantial funds to develop laboratory facilities for clinical research and for weekly lectures by "the master." These lectures were prepared in great detail and with careful thought, although their public presentation appeared to be spontaneous. Charcot had already achieved considerable recognition in France; his work was now quickly recognized throughout the Continent, attracting disciples and students from far and wide. Of special note in his later years was an interest in "hysteria," a label used in his day for patients with clinical signs of pathology that could not be correlated with underlying anatomical or neurological diseases. Because this category was generally considered a catch-all—a place to assign those who could not be properly diagnosed in one or another class of standard disorders—Charcot made a valiant effort to subdivide the variants of those so categorized. He differentiated subgroups still in use, such as those with defective memories, peculiar or inexplicable losses of sensitivity, apparently (false) motoric seizures that simulated epilepsy, and so on.

It was Charcot's contention that all patients with hysteria suffered from a "weak" constitution; that is, they possessed neurological vulnerabilities that made them highly susceptible to ordinary life conditions, such as work-related stresses. Among Charcot's assertions were that these constitutionally weak patients could be readily hypnotized. In fact, Charcot believed that *only* patients with hysteria could be hypnotized, as they were impressionable individuals whose neurologically weak minds could be readily swayed by the suggestions of others. Worthy of note, however, was Charcot's recognition that hysteria could be found in men as well as women, although he asserted that secondary psychological features typically differentiated the genders.

Charcot's stature and ideas concerning hysteria attracted the young Sigmund Freud, a neurologist in training from Vienna, who came to study with him during the winter of 1885. So impressed was Freud with Charcot's lectures that he set out to translate the professor's writings for German-reading neurologists. After this, Freud progressed in his own innovative direction, disagreeing fundamentally with Charcot's neurological assertions regarding hysteria.

Three classes of experience were stressed by Freud and his psychoanalytic colleagues as conducive to psychopathology: (1) the extent to which the earliest and most basic needs of a young child's nurturance and protection are frustrated; (2) the conflicts with which children must deal as they develop; and (3) the general parental attitudes and familial settings in which children's experiences occur and are learned.

The emphasis the psychoanalytic theorists placed on early childhood experience represented their view of disorders in adulthood as direct products of the continued

and insidious operation of past events. For them, knowledge of the past should provide information indispensable to understanding adult difficulties. To the question "What is the basis of adult disorders?", they would answer: "The anxieties of childhood and the progressive sequence of defensive maneuvers that were devised to protect against a recurrence of these feelings."

According to psychoanalysis, therefore, adult patterns of behavior are not the results of random influences, but arise from clear-cut antecedent causes. For the most part, these causes persist out of awareness; that is, they are kept unconscious because of their troublesome character—notably the stressful memories and emotions they contain, and the primitive nature of the child's youthful defenses. Central also to the analytic viewpoint is the concept of psychic conflict. In this notion, behavior is considered to result from competing desires and their prohibitions, which are expressed overtly only through compromise and defensive maneuver, and often in disguised form. Furthermore, all forms of behavior, emotion, or cognition are likely to serve multiple needs and goals; that is, they are "overdetermined." Behavioral expressions and conscious cognitions emerge as surface manifestations of several hidden forces that reside in the unconscious.

The concept of the unconscious—inner thoughts and feelings beyond immediate awareness—was brought to the fore through the dramatic methods of an Austrian physician, ***Franz Anton Mesmer*** (1734–1815). Borrowing Paracelsus's notion of a physically based planetary magnetism, Mesmer believed that many forms of illness resulted from imbalances of universal magnetic fluids. These imbalances, he concluded, could be restored either by manipulating magnetic devices or by drawing upon invisible magnetic forces that emanated from one person to another.

By the late 19th century, both magnetism and hypnotism, a method developed by ***James Braid*** (1795–1860), had begun to fall into disrepute as therapeutic procedures. A modest physician working in a rural region near Nancy in France had heard of James Braid's work at a lecture and decided to explore its possibilities in his limited practice. Well regarded in his local community, ***Ambroise-Auguste Liébault*** (1823–1904) utilized a simple method of inducing sleep by suggesting to patients that they look into his eyes while he spoke to them in quiet tones. In 1866, Liébault published a small book titled *Du Sommeil et des États Analogues* (*Sleep and Analogous States*), in which he stressed that the power of suggestion not only was central to successful hypnotism, but was the primary vehicle of therapeutic efficacy.

Liébault was generally considered a simpleton, if not a quack, by his colleagues. Nevertheless, rumors of his therapeutic successes came to the attention of a well-regarded professor of medicine at the Nancy School of Medicine, ***Hippolyte-Marie Bernheim*** (1840–1919), a young Jewish physician who had recently been appointed to this new medical institution. Bernheim had been treating a patient with sciatica for 6 years with minimal success. He referred this patient to Liébault, who utilized his methods of suggestive sleep and succeeded within 6 months in fully relieving the patient of the disorder. As a result, Bernheim decided to experiment with Liébault's radical hypnotic methods in his own clinic.

We have just discussed Charcot's signal importance in developing methods of clinical neurology. By contrast, his role in fostering a psychoanalytically oriented psychiatry stems less from the intent or the originality of his work than in the incidental part he played in stimulating the ideas of others, notably Freud and Janet. As noted earlier, Charcot studied the diverse and confusing symptoms of hysteria at the Salpêtrière. Because of his neurological orientation, he viewed trances, memory losses, and bodily anesthesia as diagnostically difficult cases of an underlying nervous system disease. It was not until his associates demonstrated that the symptoms of hysteria could be induced by hypnotic procedures that Charcot reconsidered his views of this puzzling ailment. His inability to differentiate between hypnotized and naturally produced paralyses, as well as the frequently noted migration or disappearance of symptoms and the anatomically impossible location of many of the paralyses he saw, convinced him that hysteria could not be a product of a simple injury or local disease of the nervous system. Despite suggestive evidence to the contrary, Charcot could

not abandon his biological perspective. To accommodate his observations, he proposed that hysteria resulted from a wide-ranging and congenital neurological deficiency, and that hypnosis merely served as a precipitant of the inborn defect.

Charcot presented his neurological thesis regarding hypnotism at the French Academy of Sciences in the early 1880s. Shortly thereafter, Bernheim brought to the world's attention Liébault's alternative interpretation concerning the role of suggestion in the hypnotic technique. First, Bernheim wrote, hypnosis could be employed with a variety of ailments; second, its effects stemmed from the power of suggestion; and third, all humans were susceptible to suggestion in varying degrees.

Although Bernheim was an internist and not a neurologist or psychiatrist, he vigorously disagreed with Charcot—maintaining that hysteria was primarily a state of heightened self-suggestion, and that hypnosis was an equivalent state induced by others. Moreover, Bernheim advanced the view that hysteria was essentially a psychogenic disorder, and applied the term "psychoneurosis" to this and similar puzzling symptom syndromes. His belief that unconscious self-suggestion might underlie the symptoms of many mental disorders played a significant role in influencing Freud's thinking. In developing the concept of psychoneurosis, Bernheim sought to parallel the medical tradition of seeking underlying biological causes for the disorder with a comparable notion of underlying psychological causes.

Josef Breuer (1842–1925) was born in Vienna, where his father was a well-known teacher and author of Jewish thought. He helped Freud financially in his early years. Even more importantly, he whetted Freud's curiosity about both hysteria and hypnosis in discussing a young patient of his, later to become famous under the pseudonym of Anna O. The case of Anna O. was described to Freud in 1880; it involved a classical example of hysteria, which followed a period when the young woman had nursed her father through a major illness. Breuer employed a hypnotic technique to encourage his patient to voice her experiences and thoughts at the time her symptoms had emerged. The memories that Anna O. recalled under hypnosis were accompanied by intense outbursts of emotion that she had been unable to vent at the time of her symptoms. Moreover, she became intensely attached to Breuer; uncomfortable with her affectionate feelings toward him, Breuer withdrew from the case.

Some years thereafter, Freud traveled to Paris and later to Nancy, where he observed the methods that Breuer had utilized—both those of Charcot and, later, those of Bernheim. Upon his return from these travels in the late 1880s, Breuer and Freud continued their discussions with a series of new cases employing the methods of hypnosis and the stirrings of emotional catharses. This work ultimately led to a series of papers and the publication of a major book, entitled *Studies on Hysteria*, in 1895. Freud and Breuer formulated their idea in this text that patients with hysteria suffered from repressed memories of emotionally traumatic events—events so distressing that the emotions they aroused could not be faced consciously at the time they occurred. It was Freud and Breuer's contention that the technique for curing hysteria was to unblock the repressed and pent-up emotions that were "kept secret" in the unconscious.

Pierre Janet's (1859–1947) career was an unusual one for a psychiatrist. Janet first taught philosophy at a small college, the Lyceum in Chateau Roux, in the rural province of Berry, and later at the Lyceum in Le Havre, where he remained for over 6 years. He began his early clinical work at the Le Havre mental hospital, where he was assigned the task of examining all incoming women who were deemed to have hysteria. Most of Janet's patients at Le Havre were young, fresh, and unsophisticated, unlike the usual inmates at the major institutions of France, such as the Salpêtrière, who had typically been examined numerous times by scores of physicians and students. By the mid-1880s, Janet had turned to the highly esteemed studies of Jean Charcot, as well as those of other scholars engaging in what was known as "psychical" research.

Janet might have been considered the most original thinker about psychoanalytic processes, had he not been overshadowed by the unusually courageous and innovative Freud. Janet evolved a theory in which neuroses resulted from an inability to integrate co-occurring psychic processes; this thesis foreshadowed, and may have led Bleuler to, the

concept of dementia praecox (schizophrenia) as a split between thought and emotion. As did Freud, Janet observed that painful experiences and undesirable impulses could not be tolerated by his patients. In developing his concept of "dissociation," Janet speculated that intolerable thoughts and feelings might take on an independent existence within a person and manifest themselves in amnesia, multiple personality, hysterical fits, and/or conversion paralyses. In this formulation, Janet recognized that different systems of thought could become pathologically separated, with one or another part lost to consciousness. This strengthened the idea that unconscious processes might persist unmodified within the person.

Despite his capacity to describe his patients and their frequent exotic behaviors and complaints, Janet did not display Charcot's and Freud's relentless curiosity, or their courage in exploring the outer reaches and deeper roots of their patients' psyches. He seemed overly cautious and circumspect, unable to plumb the depths of psychic conflict and sexual pathology. As some have characterized him, he was a "neat and well-stocked pantry, with everything in its proper place."

Sigmund Freud (1856–1939; see Figure 1.6) was arguably the most influential psychologist and physician of the 20th century. His reinterpretation of the observations first made by Charcot and Bernheim initiated an intellectual and cultural revolution of worldwide proportions. His theories have been both extravagantly praised and intensely castigated. Venerated by some and condemned by others, Freud has been spoken of at times as one of history's greatest scientists, and at others as a fraudulent cult leader. Numerous historians refer to him as the greatest psychologist of all time, the profoundest of all human scientists. Others are convinced that the unconscious never existed except in Freud's mind, and that his theories were baseless and aberrational. Some speak of him as a false prophet; others depict him as a courageous fighter for the truth. His most condemning detractors describe him as a neurotic egotist who propounded irrational and fantastic theories. More balanced historians aver that Freud's discoveries merely crystallized previously diffuse ideas of his many predecessors, such as those described in previous pages.

FIGURE 1.6. Sigmund Freud.

Personally and professionally, Freud was a man of divergent dispositions. A militant atheist and radical theorist, he espoused liberated attitudes toward sexuality; at the same time, he was politically conservative, usually somber and unsmiling, impeccably dressed, invariably anxious about finances, clearly suffering in his middle years from assorted psychosomatic symptoms, and fearfully hesitant about modern contrivances. He always felt that he was an outsider. "A godless Jew" and free thinker, yet conservative in personal behavior, prissy, and formalistic, he did not leave his home city until forced to do so following the Nazi takeover of Austria.

Freud devoted his long and fruitful life to the development and elaboration of his theories and techniques. Unlike his great German contemporary Kraepelin, who sought to classify broad groups of disorders with a common course and symptoms, Freud stressed the brightly etched inner memories, the feverish imaginations, and the unique attributes of each patient. And unlike Janet, his French contemporary, who viewed neuroses as the results of an underlying constitutional deficiency, Freud set out to trace the perplexing ambiguities, the afflicted emotional palette, the convoluted psychogenic origins, and the primitive passions that he perceived and explored as the unconscious source and undergirding force of each manifest disor-

der. It was not only the dense interplay of refracted realities in his findings that proved so epochal; the ever-dividing and sprawling new lines of his individualistic philosophy and his orientation toward the implausible and desultory character of life's realities, as well as the odds and ends of its rarefied energies, all served as a foundation for the 20th-century understanding of humankind's complicated and intriguing nature.

According to Freud, each stage of psychosexual development would produce a distinctive set of anxieties and defenses resulting from instinct frustration and conflict. Symptoms and character traits would arise from the persistence into adulthood of childhood anxieties and defenses. Freud's early disciples, notably ***Karl Abraham*** (1877–1925) and ***Wilhelm Reich*** (1897–1957), differentiated the oral psychosexual period into two phases: the "oral-sucking" phase, in which food was accepted indiscriminately, followed by the "oral-biting" period, in which food was accepted selectively, occasionally rejected, and aggressively chewed. In their view, excessive gratifications, conflicts, or frustrations associated with each of these phases could establish different patterns of adult personality. For example, an overly indulgent sucking stage might lead to imperturbable optimism and naive self-assurance. An ungratified sucking period might lead to excessive dependency and gullibility; for example, deprived children might learn to accept anything in order to ensure that they will get something. Frustration experienced at the biting stage might lead to the development of aggressive oral tendencies such as sarcasm and verbal hostility in adulthood.

At a later period in his exploration of the disorders of personality, Freud speculated that character classification could be based on his threefold structural distinction of "id," "ego," and "superego." Thus, in 1931, he sought to devise character types in accord with which psychoanalytic structure was dominant. First, he proposed an "erotic" type—persons whose lives were governed by the instinctual demands of the id. "Narcissistic" individuals were so dominated by the ego that neither other persons nor the demands of id or superego can affect them. "Compulsive" persons were so tightly regulated by the strictness of the superego that all other functions were dominated. Lastly, Freud identified a series of "mixed" types in which combinations of two of the three characterological structures outweighed the third. Freud's compulsive character type has been well represented in the literature, but only in the past 30 years have his proposals for a narcissistic personality disorder gained attention (Millon, 1981, 1996).

Alfred Adler (1870–1937), founder of the school of individual psychology, became an outspoken critic of Freud's views on infantile sexuality shortly before Jung did in 1911. On the basis of his own clinical observations, Adler concluded that superiority and power strivings were more fundamental to pathology than sexuality was. Although many of his patients were not overtly assertive, he observed that their disorder enabled them to dominate others in devious and subtle ways. Phobias and hypochondriasis, for example, not only excused patients from disagreeable tasks, but allowed them to control and manipulate others. Adler hypothesized that these strivings for superiority were consequences of the inevitable and universally experienced weakness and inferiority in early childhood. In this conception, Adler attempted to formulate a universal drive that would serve as an alternative to Freud's universal sexual strivings.

According to Adler, basic feelings of inferiority led to persistent and unconscious compensatory efforts. These were manifested in pathological struggles for power and triumph if individuals experienced unusual deficiencies or weaknesses in childhood. Among healthier personalities, compensation accounted for strivings at self-improvement and interests in social change and welfare. These compensatory struggles or strivings, acquired by all individuals as a reaction to the restrictions imposed by their more powerful parents, led to general patterns of behavior that Adler called "styles of life."

Although chosen by Freud as his heir apparent, ***Carl Gustav Jung*** (1875–1961) did not agree with Freud's emphasis on the sexual nature of development and motivation, and established his own system of analytic psychology in 1913. Jung expanded the notion of "libido," Freud's concept for the basic sexual energies, to include all life-propelling forces. The concept of "racial memories," later termed the "collective unconscious," was proposed to suggest that instinctual

forces were more than seething animalistic impulses; according to Jung, these forces contained social dispositions as well. These primitive dispositions were often expressed in folklore and mystical beliefs. When no acceptable outlet could be found for them in societal life, they took the form of symptoms such as phobias, delusions, and compulsions. Jung's belief in unconscious social dispositions led also to his formulation of two basic personality types, the "extrovert" and the "introvert." Despite these and other original contributions, Jung's views had a minimal impact upon the mainstream of psychodynamic theory and practice.

Karen Horney (1885–1952) contended that neurotic disorders reflected cultural trends learned within the family; she minimized biological determinants and stressed interpersonal relationships. She believed that anxiety and repressed anger were generated in rejected children and led to feelings of helplessness, hostility, and isolation. As these children matured, they developed an intricate defensive pattern of either withdrawal, acquiescence, or aggression as a means of handling their basic anxiety. Although Horney felt that adult patterns resulted largely from early experience, she argued, in contrast with Freud, that therapy should focus on its adult form of expression. First, she averred that the intervening years between childhood and adulthood caused important changes in adaptive behavior. And, second, present-day realities had to be accepted, and the goals of therapy had to take them into account.

Horney's descriptive eloquence was without peer; nevertheless, difficulties arose when she summarized what she referred to as the major "solutions" to life's basic conflicts. Although her primary publications were written over a short period, she utilized different terms to represent similar concepts (Horney, 1937, 1939, 1942, 1945, 1950). Faced with the insecurities and inevitable frustrations of life, Horney identified three emergent modes of relating: "moving toward" people, "moving against" people, or "moving away" from them. In her 1945 book, Horney formulated three character types to reflect each of these three solutions: Moving toward was found in a "compliant" type; moving against, in an "aggressive" type, and moving away, in a "detached" type. In 1950, Horney reconceptualized her typology in line with the manner in which individuals solve intrapsychic conflicts; she termed these solutions "self-effacement," "expansiveness," and "neurotic resignation." Although these sets of three do not match perfectly, they do correspond to the essential themes of Horney's characterology.

Several major thinkers from Great Britain began to formulate what is referred to as the "object relations" approach to psychoanalytic theory in the 1940s and 1950s. Most inventive of these was ***Melanie Klein*** (1882–1960), one of the originators of child psychoanalysis (along with Anna Freud, with whom she vigorously differed and contended for leadership in the British analytic community). Klein's views met with intense opposition in the wider psychoanalytic world, and fierce battles raged within British analytic circles over her inventive concepts. Although she was a vigorous critic of more orthodox psychoanalytic thought, she believed that emphasizing the very earliest and most primitive stages of development was a natural extension of Freud's original formulations. In the United States since the mid-1960s, ***Otto Kernberg*** (1928–) has sought to develop a synthesis of drive reduction and object relations frameworks—an approach that has brought considerable attention to modern analytic thought, as well as generating considerable controversy.

Owing to numerous pragmatic considerations at the time—not the least of which were the advent of effective psychopharmacological medications and the emergence of the sophisticated community mental health movement—the balance of power within American psychiatry shifted slowly but surely away from psychoanalysis in the 1970s. The wider culture had also reconsidered the high repute in which it had formerly held for psychoanalysts: They were no longer seen as wise, generous, and kindly, but were depicted increasingly as irrelevant stumblebums. In a review of what was wrong with psychoanalysis, Richard Weber (1995) stated that concepts such as infantile sexuality were more than objects of disbelief, not so much disproven as incapable of disproof; in his view, they should be relegated to the same scientific status as astrology. And eminent English psychologist Hans Eysenck (1985) asserted that just as chemistry had had to

unshackle itself from the fetters of alchemy, and the brain sciences had had to disengage themselves from phrenology, so too must psychology and psychiatry abandon the pseudoscience of psychoanalysis.

Questions have been raised as to whether or not scientific concepts can be founded on unconscious data. Psychoanalytic theories have been criticized as unscientific mixtures of metaphorical analogies, speculative notions, and hypothetical constructs because their data are anchored so tenuously to the observable world. Added to this rather harsh judgment is the equally critical view that the methods of collecting unconscious data are both unreliable and imprecise. How can concepts of the unobservable unconscious be empirically anchored? Can one accept what a patient says without having it corroborated by external evidence? Is the patient an unbiased judge, or is he or she motivated to agree with the all-knowing therapist?

These and many other questions have been raised about the subjective and methodologically uncontrolled procedures used for the development of psychoanalytic theories. To critics, the ingenious speculations of psychoanalytic theorists are at best a starting point—a preliminary set of propositions requiring reformulation as clearly specified hypotheses that can be confirmed or disproved. Despite these criticisms, psychoanalytic processes may be a necessary part of the study of humankind's pathological functioning. These processes may be difficult to formulate according to the tenets of scientific objectivity, but their existence cannot be denied or overlooked. Efforts to unravel them may fall prey to theoretical obscurity and methodological difficulties, yet the search should be mandatory. To the deeply inquiring and instinctively insightful thinker, the intricate themes of mental life articulated by analysts have a richness and unquestionable accuracy about them.

Current Trends

In the latter decades of the 20th century, several major theorists appear to have developed a strong foundation of ideas that may influence the future course of psychopathology's history. We describe some of them briefly here.

Aaron Timothy Beck (1921–) has been a prominent and insightful contributor to cognitive therapy, especially as applied to a wide range of the Axis I clinical syndromes. More recently, he and his associates have addressed the subject of personality, articulating "cognitive schemas" that shape the experiences and behaviors of numerous personality disorders. Beck focused his early research efforts largely on testing psychoanalytic theories of depression, but when his studies failed to support his hypotheses, he explored a more cognitive explanation of the disorder. He found that most depressed patients had broad negative views of themselves, of the world at large, and of their own future. Beck reasoned that these negative "cognitive distortions," as he termed them, could be reoriented to accord with reality through the application of logic and the rules of evidence. He eventually applied these cognitive investigations to a broad range of disturbances, from anxiety to substance use to personality disorders. Cognitive approaches to the treatment of mental disorders have become more than merely the mainstream of "talking therapies" today. More than one-third of all therapists speak of themselves as cognitive in orientation; the others employ cognitive techniques periodically.

C. Robert Cloninger (1945–) has formulated a recent model of personality dispositions, drawing upon genetic and neurobiological substrates. Cloninger's complex theory is based on the interrelationship of several heritable characteristics or functional dispositions, notably "novelty seeking," "harm avoidance," and "reward dependence." Each of these is associated with different neurobiological systems (dopamaninergic, serotonergic, and noradrenergic, respectively).

More specifically, *novelty seeking* is hypothesized to dispose individuals toward exhilaration or excitement in response to novel stimuli; it leads to the pursuit of potential rewards, as well as an active avoidance of both monotony and punishment. Harm avoidance reflects a disposition to respond strongly to aversive stimuli, leading individuals to inhibit behaviors to avoid punishment, novelty, and frustrations. Reward dependence is hypothesized as a tendency to respond to signals of reward (e.g., verbal signals of social approval), and to resist extinction of behaviors previously associated with re-

wards or relief from punishment. To extend the theme of novelty seeking, for example, individuals high on this dimension but average on the other two dimensions would be characterized as impulsive, exploratory, excitable, quick-tempered, and extravagant—likely to seek out new interests, but inclined to neglect details and to become quickly distracted or bored.

Larry Siever's (1950–) theoretical model has also attempted to link neurotransmitters' properties to the various personality disorders. Siever has developed a dimensional model that has major clinical syndromes at one extreme and the milder personality disorders at the other end. He proposes four major dimensions: "cognitive/perceptual organization," "impulsivity/aggression," "affective instability," and "anxiety/inhibition." For example, schizophrenic disorders are viewed as disturbances of a cognitive/perceptual nature, exhibiting themselves in thought disorders, psychotic symptoms, and social isolation; schizotypal personality disorder would serve as the prototype among the Axis II disorders. Disorders of impulsivity/aggression are hypothesized as resulting in poor impulse control, particularly as evident in aggressive actions. In the more distinct clinical syndromes, Siever suggests the presence of impulsivity/aggression in explosive disorders, pathological gambling, or kleptomania. When this dimension is more pervasive and chronic, it may be seen in persistent self-destructive behaviors, such as those characteristic of borderline and antisocial personality disorders. Problems of affective instability are most clearly observed in the intensity and dysregulation of mood disorders. When this inclination is more sustained over time, it may interfere with the development of stable relationships and self-image, as may be manifested in borderline and histrionic personality disorders. Lastly, the anxiety/inhibition dimension appears to be related to the Axis I anxiety syndromes (e.g., social phobia, compulsive rituals); when it is present at a low threshold over extended periods of development, avoidant, compulsive, or dependent personality disorder may result.

Eric Kandel (1930–) left Austria for the United States with his family in 1939. He attended Harvard University to study the humanities. Intrigued by his reading of Freudian literature, Kandel went to medical school to pursue a career as a psychoanalyst. Early in his training, Kandel undertook work in neurophysiology, in the hope of gaining a clearer understanding of how memory and emotions are biologically generated and intertwined. His early research led him to explore the hippocampus as the primary source of memory formation, but he soon turned to a simpler neurosystem for his intensive analysis. Utilizing the *Aplysia* sea slug, a creature with only 20,000 nerve cells, as an experimental animal, Kandel identified a number of the biochemical changes that accompany memory formation—explicating how short-term memory involves just a minor modulation of the synapses, whereas long-term memory requires new synaptic linkages. More specifically, he showed that a protein termed CREB helps the nervous system retain a memory or a learned skill for a long period of time rather than just briefly.

This work earned Kandel, together with Paul Greengard and Arvid Carlsson, the Nobel Prize in Physiology or Medicine in 2000. Together, they began to decode how the vast numbers of synaptic connections in the brain (at least 100 trillion) are able to communicate continually, as well as to alter their strength, flexibility, and function.

Although ***Paul Meehl***'s (1920–2003) biologically oriented social learning model is limited to schizophrenia, it is notable for both its elegance and specificity. He hypothesized that only a certain class of people, those with a particular genetic constitution, have any liability to schizophrenia. Meehl suggested that the varied emotional and perceptual–cognitive dysfunctions people with schizophrenia display are difficult to explain in terms of single-region disorders. The widespread nature of these dysfunctions suggested to Meehl the operation of a more diffuse integrative neural defect. Although a combination of different neurological disturbances can account for this defect, he opted for an explanation in terms of deficits in synaptic control. More specifically, he believed that the major problem in schizophrenia lies in a malfunctioning of the two-way mutual control system between perceptual–cognitive regions and the limbic motivation center. Meehl proposed that integrative neu-

ral defects are the only direct phenotypic consequences produced by the genetic disorders; these consequences, given the label "schizotaxia," are all that can properly be spoken of as inherited. The imposition of certain social learning histories on schizotaxic individuals results in a personality organization that Meehl called the "schizotype." Four core behavior traits—namely, anhedonia, cognitive slippage, interpersonal aversiveness, and ambivalence—are not innate. However, Meehl postulated that schizotaxic individuals universally learn them, given any existing social learning regimen (from the best to the worst). If the social environment is favorable and a schizotaxic person has the good fortune of inheriting a low anxiety readiness, physical vigor, and a general resistance to stress, the personality organization will remain a well-compensated schizotype, and the individual may never manifest symptoms of clinical schizophrenia.

In England, two child-oriented analysts, ***Michael Balint*** (1896–1970), and ***John Bowlby*** (1907–1990), contributed to an understanding of developmental vicissitudes. Balint's concept of the "basic fault" was derived from studies of patients whose borderline characteristics appeared to be consequences of having missed something during the first year or two of life. In Balint's view, such a fault can lead to one of two extreme reactions. In the so-called "ocnophile" adaptation, infants deal with the experience by clinging excessively to others; in the "philobat" adaptation, children learn to distance themselves from others and rely entirely on themselves. Bowlby stressed "attachment learning," especially that resulting from the loss of a significant early relationship. He spoke of children suffering maternal loss as passing through three phases: protest, despair, and detachment. In the first stage, children evidence anger at their loss; in the second, children begin to lose hope that the mother will ever return; finally, despair turns to detachment (i.e., the children become depressed and unresponsive). Sharing Melanie Klein's object relations model, Bowlby asserted that the manner in which children deal with affectional deprivation will determine how they will react in later life to problematic relationships with loved ones.

In the 1980s, sharp criticisms were raised against the dominance of the DSM and ICD systems, both based on a Kraepelinian vision (Klerman, 1986). Some eminent psychiatrists have asserted that, except for organic disorders, a classificatory diagnosis is less important than a psychodynamic study of personality; that is, rather than fitting a patient's symptoms into a fixed classificatory scheme, a clinician should seek to understand the person in terms of his or her distinctive life experiences (American Psychoanalytic Association, 2006). Others have noted that too much research time is wasted and too many errors are perpetuated because investigators cling to an outdated classification. Proponents, on the other hand, have explained the viability of a Kraepelinian schema by the "fact" that there is a considerable amount of truth contained in the system and the practical implications associated with its labels; that is, they are more than merely sufficient when compared with the power of competing concepts. One of the major problems facing a field as inchoate and amorphous as mental health is its susceptibility to subjective values, cultural biases, and chance events. Were the field a "hard" science, anchored solidly in readily verified empirical fact, progress would presumably derive from advances of a tangible and objective nature. Unfortunately, that is not the case. Nevertheless, the field has endeavored to standardize, as much as possible, the language conventions and classification rules for diagnosing mental disease categories. To say the least, effective communication among clinical centers was seriously compromised, as were useful records for epidemiological statistics and research.

Theodore Millon (1928–) has come to believe that the widespread desire among theorists to unify science should not be limited to explicating physics; that is, it should be possible in all fields of nature that have been subdivided by habit, tradition, or pragmatics (e.g., economics, sociology, geology). He believes unification to be a worthy goal even within the newer sciences, such as personology. Efforts to coordinate the separate realms that constitute the field of the mind and, more specifically, the field of mental disorders should be particularly useful. Rather than independently developing autonomous

and largely unconnected professional activities and goals, a truly mature mental science should embody, and create a synergistic bond among, five explicit elements:

1. *Universal scientific principles* that are grounded in the ubiquitous laws of nature; despite their varied forms of expression, these principles may provide an undergirding framework for constructing narrow-based subject-oriented theories.
2. *Subject-oriented theories*, or explanatory and heuristic conceptual schemas of the mind and mental illness. These theories should be consistent with established knowledge in both their own and related sciences, and should enable reasonably accurate propositions concerning all clinical conditions to be both deduced and understood, enabling thereby the development of a formal classification system.
3. *Classification of personality styles and pathological syndromes*, or a taxonomic nosology that has been derived logically from the theories. The taxonomy should provide a cohesive organization within which its major categories can readily be grouped and differentiated, permitting thereby the development of coordinated assessment instruments.
4. *Personality and clinical assessment instruments*, or tools that are empirically grounded and sufficiently sensitive quantitatively to enable the theories' propositions and hypotheses to be adequately investigated and evaluated. Hence the clinical categories constituting the nosology should be able to be readily identified (diagnosed) and measured (dimensionalized), thus specifying target areas for interventions.
5. *Integrated therapeutic interventions*, or planful strategies and modalities of treatment. These interventions should accord with the theories and be oriented to modify problematic clinical characteristics, consonant with professional standards and social responsibilities.

Perhaps the only realistic and significant question to be posed in appraising a new taxonomy or nomenclature is not whether it mirrors the state of the science perfectly, or whether it provides answers to all possible questions professionals within the discipline may ask, but whether it represents an advance over preceding nosological systems and whether it will be employed with greater clinical accuracy and facility by future practitioners and researchers.

Having participated over two intense 5-year periods as a member of the DSM-III and DSM-IV committees, Millon is considerably more charitable than he once was about the purposes and success with which these task forces met their responsibilities. He has no illusion, however, that the task was completed. As he wrote nearly two decades ago,

> Classifying mental illness must be an outgrowth of both psychology and medicine. As such, efforts to construct a taxonomy must contend with the goals, concepts, and complications inherent in both disciplines (e.g., context moderators, definitional ambiguities, overlapping symptomatologies, criterion unreliabilities, multidimensional attributes, population heterogeneities, instrument deficits, and ethical constraints). (Millon, 1991, p. 245)

Thus the profession remains unsure today whether to conceive depression as a taxon (category) or an attribute (symptom); whether to view it as a dimension (with quantitative degrees of severity) or as a set of discrete types; or whether to conceive it as a neuroendocrinological disease or as an existential problem of life. Although debates on these issues often degenerate into semantic arguments and theoretic hairsplitting, it is naive to assume that metaphysical verbiage and philosophical word quibbling are all that are involved. Nevertheless, the language we use, and the assumptions such language reflects, are very much a part of our scientific disagreements. This volume addresses these substantive and philosophical issues as they may apply to DSM-V and ICD-11. In addition to reviewing the history of psychopathology, this chapter has sought to illustrate that philosophical issues and scientific modes of analysis must be considered in directing the future of mental illness classification. The many recommendations made in this book will not in themselves achieve clear resolutions to all nosological quandaries. It is more likely that their role will be to unsettle prevailing habits and thereby force progress, if only by challenging cherished beliefs and assumptions.

Acknowledgment

Portions of this chapter have been adapted and updated from Millon (2004). Copyright 2004 by John Wiley & Sons. Adapted by permission.

References

American Psychoanalytic Association. (2006). *Psychodiagnostic manual*. Silver Spring, MD: Author.

Aschaffenburg, G. (1922). Constitutional psychopathies. In G. Aschaffenburg, *Handbook of medical practice* (Vol. 4). Leipzig: Barth.

Baillarger, J. (1853). De la melancholie avec stupeur. *Annales Medico-Psychologiquies*, *5*, 251.

Bhugra, D. (1992). Psychiatry in ancient Indian texts: A review. *History of Psychiatry*, *3*, 167–186.

Bleuler, E. (1950). *Dementia praecox oder gruppe der schizophrenien*. Leipzig: Deuticke. (Original work published 1911)

Briquet, P. (1859). *Traite clinique et therapeutique a l'hystérie*. Paris: Baillière.

Burton, R. (1621). *The anatomy of melancholy: What it is, with all the kinds, causes, symptoms, prognostickes, and severall cures of it*. Oxford, UK: Lichfield for Cripps.

Chaslin, P. (1912). *Elements de seminologie et de clinique mentale*. Paris: Asselin et Houzeau.

Comrie, J. D. (1922). *Selected works of Thomas Sydenham, M.D*. New York: William Wood Co.

Cullen, W. (1777). *First lines of the practice of physick for the use of students in the University of Edinburgh* (4 vols.). Edinburgh, UK: Creech.

de la Chambre, M. C. (1640). *Les charactères des passions* (Vol. 1). Paris.

de Sauvages, F. B. (1731). *Treatise de nouvelles classes de maladies*. Paris: Avignon.

de Sauvages, F. B. (1771). *Nosologie methodique dans la quelle les maladies sont rangées par classes*. Paris: Herrisant.

Esquirol, J. (1838). *Des maladies mentales* (2 vols.). Paris: Baillière.

Eysenck, H. J. (1985). *Decline and fall of the Freudian empire*. London: Penguin.

Falret, J. P. (1854). De la folie circulaire. *Bulletin de l'Academie Medicale*, *19*, 382–394.

Falret, J. P. (1862). *Traite pratique des maladies mentales*. Paris.

Feuchtersleben, E. (1847). *The principles of medical psychology: Being the outlines of a course of lectures*. London: Sydenham Society.

Freud, S. (1950). Libidinal types. In *Collected papers* (Vol. 5). London: Hogarth Press. (Original work published 1931)

Freud, S., & Breuer, J. (1895). *Studien über hysterie*. Vienna.

Goddard, K. (1991). Morita therapy: A literature review. *Transcultural Psychiatry*, *28*, 93–115.

Griesinger, W. (1845). *Mental pathology and therapeutics*. London: New Sydenham Society.

Griesinger, W. (1868). Introductory comments. *Archives for Psychiatry and Nervous Diseases*, *1*, 12.

Haslam, J. (1809). *Observations on madness and melancholy*. London: Callow.

Haslam, J. (1810). *Illustrations of madness: Exhibiting a singular case of insanity*. London: G. Hayden.

Heinroth, J. C. (1818). *Lehrbuch der Störungen des Seelenlebens*. Leipzig: Thieme.

Hellpach, W. (1920). Amphithymia. *Zeitschrift für die Gesamte. Neurologie und Psychiatrie*, *19*, 136–152.

Hertt, W. S. (Ed. & Trans.). (1936). Physiognomics. In *Aristotle: Minor works*. Cambridge, MA: Harvard University Press.

Horney, K. (1937). *The neurotic personality of our time*. New York: Norton.

Horney, K. (1939). *New ways in psychoanalysis*. New York: Norton.

Horney, K. (1942). *Self-analysis*. New York: Norton.

Horney, K. (1945). *Our inner conflicts*. New York: Norton.

Horney, K. (1950). *Neurosis and human growth*. New York: Norton.

Kahlbaum, K. L. (1874). *Die Katatonie, oder das Spannungsirresien*. Berlin: Kirschwald.

Koch, J. A. (1891). *Die psychopathischen Minderwertgkeiten*. Ravensburg: Maier.

Klerman, G. L. (1986). Historical perspectives on contemporary schools of psychopathology. In T. Millon & G. L. Klerman (Eds.), *Contemporary directions in psychopathology: Toward the DSM-IV*. New York: Guilford Press.

Kraepelin, E. (1896). *Psychiatrie: Ein Lehrbuch für Studirende und Aertze* (5th ed.). Leipzig: Barth.

Kraepelin, E. (1899). *Psychiatrie: Ein Lehrbuch* (6th ed.). Leipzig: Barth.

Kraepelin, E. (1913). *Psychiatrie: Ein Lehrbuch für Studirende und Aerzte* (8th ed., 4 vols.). Leipzig: Abel.

Kraepelin, E. (1920). *Hundert jahre psychiatrie*. Berlin: Springer-Verlag.

Kraepelin, E. (1921). *Manic–depressive insanity and paranoia*. Edinburgh, UK: Livingstone.

Krafft-Ebing, R. von. (1879). *Lehrbuch der Psychiatrie auf klinischer Girundlage*. Stuttgart: Erike.

Krafft-Ebing, R. von. (1937). *Psychopathia sexualis*. New York: Physicians & Surgeons Books. (Original work published 1882)

Kramer, H., & Sprenger, J. (1928). *Malleus malefacarum* [The witches' hammer] (M. Summers, Trans.). London: Rodker. (Original work published 1487–1489)

Lavater, J. C. (1789). *Essays of physiognomy: for the promotion of knowledge and the love of mankind* (T. Holcroft, Trans.). London: Robinson, Paternoster-Row.

Liébault, A. A. (1866). *Du sommeil et des états analogues consideres surtout au point de vue de l'action du moral sur le physique* [Sleep and analogous states]. Paris: Masson.

Liu, X. (1981). Psychiatry in traditional Chinese medicine. *British Journal o9f Psychiatry*, *138*, 429–433.

Maudsley, H. (1860). *Annual board report at Cheadle Royal Hospital*. Unpublished manuscript.

Maudsley, H. (1876). *Physiology and pathology of mind*. New York: Appleton.

Meyer, A. (1906). Fundamental conceptions of dementia praecox. *British Medical Journal*, *2*, 757–760.

Meyer, A. (1912). Remarks on habit disorganizations in the essential deteriorations. *Nervous and Mental Disease Monographs*, *9*, 95–109.

Meyer, A. (1912). Remarks on habit disorganizations in the essential deteriorations. *Nervous and Mental Disease Monographs*, *9*, 95–109.

Millon, T. (1981). *Disorders of personality: DSM-III, Axis II*. New York: Wiley.

Millon, T. (1991). Classification in psychopathology: Rationale, alternatives, and standards. *Journal of Abnormal Psychology*, *100*, 245–261.

Millon, T. (2004). *Masters of the mind*. Hoboken, NJ: Wiley.

Millon, T. (with Davis, R. D.). (1996). *Disorders of personality: DSM-IV and beyond* (2nd ed.). New York: Wiley.

Morel, B. A. (1857). *Traité de dégénérescences physiques, intellectuelles et morales de l'espèce humaine*. Paris: Baillière.

Paracelsus. (1941). *Von den Krankheiten, so die Vernufft berauben* (G. Zilboorg, Trans., & H. Sigerist, Ed.). Baltimore: Johns Hopkins University Press. (Original work published 1567)

Peck, A. L. (Ed. & Trans.). (1965). *Aristotle: Historia animalium*. Cambridge, MA: Harvard University Press.

Porta, G. B. della. (1957). *Natural magic* (D. J. Price, Ed.). New York: Basic Books. (Original work published 1558)

Porta, G. B. della. (1586). *De humana physiognomia*. Apud J. Cacchium: Vici Aequensis.

Porta, G. B. della. (1588). *Phytognomonica*. Naples: Saluianum.

Porter, R. (2002). *Madness*. New York: Oxford University Press.

Robinson, D. R. (1976). *An intellectual history of psychology*. New York: Macmillan.

Roccatagliata, G. (1973). *Storia de la psichiatria antica*. Milan: Ulrico Hoepli.

Sacher-Masoch, L. (1870). *Venus in furs*. Dresden: Dohrn.

Thomasius, C. (1692). *Recent proposals for a new science for obtaining a knowledge of other men's minds*. Halle, Germany: Christopher Salfeld.

Tredennick, H. (Ed. & Trans.). (1967). *Aristotle's prior analytics*. Cambridge, MA: Harvard University Press.

Voisin, F. (1851). *The analysis of human understanding*.

Weber, R. (1995). *Why Freud was wrong: Sin, science and psychoanalysis*. New York: Basic Books.

Willis, T. (1971). *De anima brutorum* [Two discourses concerning the soul of brutes]. Gainesville, FL: Scholar's Facsimiles & Reprints. (Original work published 1672)

Willis, T. (1978). *Cerebri anatome* [The anatomy of the brain and nerves]. Birmingham, AL: Classics of Medicine Library. (Original work published 1664)

Zilboorg, G., & Henry, G. W. (1941). *A history of medical psychology*. New York: Norton.

CHAPTER 2

Themes in the Evolution of the 20th-Century DSMs

ROGER K. BLASHFIELD
ELIZABETH FLANAGAN
KRISTIN RALEY

A recent book by Wedding, Boyd, and Niemiec (2005) is titled *Movies and Mental Illness: Using Films to Understand Psychopathology*. Wedding and colleagues start their book with a quotation from a screenwriter for *A Beautiful Mind*, in which the writer makes an analogy between watching movies about psychopathology and going to a zoo. In both instances, the viewer is presented with instances of wondrous diversity. Yet an organized zoo, in many ways structured around the principles of biological classification, can illustrate how this diversity can both be explained and demonstrate regularities that a person might miss when first looking at the wild, scary, and beautiful creatures of the zoo.

Consistent with the quotation that they use to start their book, Wedding and colleagues (2005) base each chapter in their book on a family of mental disorders as represented in the current, official edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM). Discussions of the movies are placed in the book according to the diagnostic concept that each movie illustrates, at least in the authors' opinion. By using DSM in this way, Wedding et al. are drawing on one of the basic purposes of a classification: It is a systematic organization of concepts describing the diversity of mental disorders. According to Blashfield and Draguns (1976), the five purposes of a classification of psychopathology are as follows: to serve (1) as a *nomenclature*, or a list of accepted nouns that mental health professionals use to talk about the patients that they see; (2) as a basis for *information retrieval*, so that novices can search for existing knowledge about mental disorder categories; (3) as a *descriptive system*, in which the name of each mental disorder summarizes the behaviors, thoughts, and emotions of individuals with that disorder; (4) as a *predictive system*, in which classificatory concepts allow both professionals and nonprofessionals to know which treatments are likely to lead to the best outcomes and to know what is most likely to happen in the course of each disorder if it is untreated; and finally (5) as a basis for a *theory (or theories) of psychopathology*, which will allow scientists, professionals, and laypeople to understand these disorders.

Sadler (2005) has performed a detailed philosophical analysis of the current classifications of psychopathology as they are

embodied in the most recent editions of DSM (DSM-IV-TR; American Psychiatric Association [APA], 2000a) and of the *International Classification of Diseases* (ICD-10; World Health Organization [WHO], 1993). Sadler focuses on the implicit values that have shaped these classifications. In his analysis, he comments repeatedly on an inherent tension within modern psychiatric classifications between what he labels "pragmatic" values (i.e., values associated with clinical practice) and "scientific" values (i.e., values associated with research). Pragmatic values include such things as the utility of classificatory concepts both for making treatment decisions and for communicating to patients and the public; the acceptance of these concepts across languages and cultures; and the ease with which they fit the way in which humans view psychopathology. Scientific values, in contrast, are more epistemic. Included within scientific values are such issues as diagnostic reliability; the level of empirical support for classificatory concepts (validity); and the relationship between classificatory concepts and the development of scientific theories about the phenomena being classified.

This chapter is a short overview of the history of changes in classification systems during the middle and late 20th century. As we present this overview, the tension between clinical/pragmatic values and scientific values is a repeated theme. However, the reader will also note, as Sadler (2005) does, that other value systems (e.g., economic and political values) have also had an impact on these changes in classification.

A Brief History of the ICDs

In the medical field, the official classification of medical disorders is currently known as the *International Statistical Classification of Diseases and Related Health Problems*—or, more briefly, as the *International Classification of Diseases* or ICD (WHO, 1992b). Although we devote most of this chapter to discussing the evolution of the DSMs, we begin with a very brief account of how the ICDs have been developed. This classification system was first adopted internationally in 1900 under the name *The Bertillon Classification of Causes of Death*, in honor of Jacques Bertillon, head of the International Statistical Institution at the time of its inception. Between 1909 and 1938, this document went through four additional iterations. After World War II, the WHO met once again to revise the system for the sixth time. It was decided at this time to expand the classification to all diseases, thereby including causes of morbidity as well as mortality. The revised classification became the *International Classification of Diseases, Injuries, and Causes of Death* (ICD-6; WHO, 1948). With the inclusion of all other diseases came a section entirely devoted to mental disorders. The ICD-6 section on mental disorders was not widely accepted, however; instead, most countries, including the United States, had their own unique classification systems (Stengel, 1959).

The WHO periodically and systematically attempted to update its classification of all medical diseases. Initially, the goal was to make these revisions every 10 years. However, since medical knowledge has been growing exponentially, this task has overwhelmed the WHO, which faces a number of other and more immediate tasks. Thus the last major revision to the ICDs occurred in the early 1990s and was published as ICD-10. The section of ICD-10 devoted to mental disorders was also published separately, in two forms: a green version for use by general clinicians (WHO, 1992a), and a blue version with diagnostic criteria that could be used in research on psychopathology (WHO, 1993). These two different versions reflect the WHO's attempt to resolve the competing tensions between pragmatic/clinical values and scientific/research values.

DSM-I

By the late 1940s, American psychiatry was undergoing change. Before World War II, the American Psychiatric Association (APA), which was the outgrowth of an earlier organization titled the Superintendents of Insane Asylums, had been dominated by psychiatrists who focused on severe mental illnesses and who believed that most mental disorders were biological diseases (Shorter, 1997). During World War II, an innovative, psychoanalytically oriented physician named William Menninger became the head

of military psychiatry. Through Menninger, the Army developed a new classification system called Medical 203 (Houts, 2000).

Given the concerns that developed from frustration with the American Psychiatric Association's earlier classification of mental disorders, they formed a Committee on Nomenclature and Statistics. This committee was charged with the task of integrating and revising the existing systems; the goal was to develop a classification system that would encompass all areas of psychological disturbance and would address problems of the emerging outpatient population. The resulting document, DSM-I, was published by the American Psychiatric Association in 1952. According to Houts (2000), DSM-I was quite similar structurally to the Medical 203 system created by Menninger and his colleagues. DSM-I contained prose descriptions of mental disorders, and the disorders therein were organized into two major categories: disorders with organic causes and disorders of psychogenic origin. The latter disorders were labeled "reactions," reflecting the contemporary thought in psychiatry that mental disorders were the results of interactions among biological, psychological, and environmental forces.

Three features of this earliest DSM are worth noting. The first is that the major goal of DSM-I was to create a classification system that all American psychiatrists could accept and use. Thus this system was not intended to be a revolutionary document that would alter thinking about mental disorders; instead, the values governing its development were pragmatic. The classification was written for psychiatrists and was intended to be used only by psychiatrists. Second, DSM-I essentially organized mental disorders into three broad groups: the psychoses (many of which were then placed into categories based on etiology); the neuroses (i.e., disorders that could be treated by psychoanalysts); and the character disorders (i.e., psychological disorders that were largely untreatable and often involved forensic decisions). Third, the DSM-I prose definitions of categories were intentionally vague. These definitions were added to increase the clinical utility of this classification, but they were intended for use as diagnostic guidelines, rather than as strict rules that could be used to differentiate between separate disorders.

From a political perspective, the development of DSM-I was stimulated by a group of psychiatrists who were distressed with the status quo in American psychiatry after World War II. At that time, psychiatry was becoming increasingly psychoanalytic in its focus (Grob, 1991; Shorter, 1997). However, the American Psychiatric Association still tended to be dominated by old-line psychiatrists working in state hospitals, who focused on the psychoses and tried to find biologically based treatments for their patients. William Menninger, who had helped develop and write Medical 203, became politically active within psychiatry and gained recognition in a collection of "young Turks" named the Group for the Advancement of Psychiatry (GAP) (Grob, 1991). The GAP pushed for the creation of DSM-I and influenced its clinical/pragmatic approach to classification, which was consistent with the social-psychiatric focus of its psychoanalytic members.

DSM-II

Although DSM-I gave American psychiatry a common nomenclature, international communication about mental disorders was still a veritable Tower of Babel. To better define the problem at hand, the WHO recruited a British psychiatrist named Erwin Stengel to conduct an overview of the different classification systems in use throughout the world. Stengel's (1959) review was a comprehensive assessment of the variations in the terminology used in each system. His conclusions were that many diagnostic concepts contained their own etiological implications, and that the terms used represented different theories regarding psychopathology (e.g., psychoanalytic, biological, etc.), thus drawing lines among theoretical camps. Stengel's observations led to an eighth revision of the mental disorders section of ICD (WHO, 1967); however, his suggestion of operational definitions was apparently ignored. The American Psychiatric Association was involved in this revision and subsequently published its own version of ICD-8 known as DSM-II (APA, 1968).

DSM-II did not differ greatly from DSM-I. The most significant difference was the removal of the term "reaction" from most of the diagnoses. This change could be viewed

as an attempt to define mental disorders as actual entities—in other words, a return to a more biological theoretical stance. However, those involved in the revision process denied that this was the case, offering the explanation that the removal of the term was an effort to rid the classification of theoretical biases toward etiological explanations for disorders that did not have known causes (Spitzer & Wilson, 1968). Although the authors of DSM-II asserted that the revised terms were theory-neutral, the use of names like "schizophrenia" rather than "schizophrenic reaction" did seem to reify the disorders in accordance with a disease model (Millon, 1986).

The main goals of DSM-II were primarily sociopolitical in nature. DSM-II was an attempt by American psychiatry to unite with psychiatrists around the world in agreeing upon a nomenclature that all psychiatrists would use. Thus DSM-II and the mental disorders section of ICD-8 were virtually the same. However, differences did exist. ICD-8 was strictly a nomenclature (i.e., simply a list of names of acceptable diagnoses, with no definitions for those names). Physicians using ICD-8 were expected to know the meanings of diagnoses within their specialty as a result of their medical training. The authors of DSM-II, in order to make this system more clinically useful, did offer short, broadly worded definitions of its categories.

Although the goal of DSM-II's developers was to create a document that would be accepted by consensus, both in the United States and around the world, its publication was not without controversy. For instance, William Menninger's brother, Karl Menninger, thought that the deletion of the term "reaction" was a major mistake, and he objected loudly by labeling DSM-II "sheer verbal Mickey Mouse" (Menninger, 1969). More importantly, the gay and lesbian communities united around their dissatisfaction with the inclusion of homosexuality as a mental disorder in the classification. In 1971, gay activists demanding a change created major disruptions at the annual convention of the American Psychiatric Association (Kutchins & Kirk, 1997). A young associate member of the DSM-II committee named Robert Spitzer was instrumental in suggesting a change that could be acceptable to both organized psychiatry and to the gay and lesbian communities. Bayer (1987) provides a detailed historical analysis of the controversies within the American Psychiatric Association about the status of homosexuality as a diagnosis.

Another political development stimulated by the publication of DSM-II was concern about the vagueness of the definitions of diagnostic categories within this system. A few years earlier, a philosopher named Carl Hempel (1965) had presented an influential paper at a meeting of research-oriented psychiatrists, in which he argued that the science of the field could not improve until psychiatry had better and more specific definitions of its central categories (Kendell, 1975; Sadler, 2005). Hempel's criticism fit with a series of research studies published in the 1950s and 1960s expressing concern with the poor reliability of psychiatrists and psychologists when making diagnoses (for overviews of this literature, see Blashfield, 1984; Kreitman, 1961; Matarazzo, 1978).

Two additional publications served as disturbing demonstrations of this concern. One was a controversial study by David Rosenhan (1973) published in the prestigious journal *Science*. Rosenhan and his colleagues went to inpatient facilities requesting admission; all reported a vague symptom suggesting a possible auditory hallucination. Otherwise, all personal information they reported was accurate. All were admitted, and all but one were diagnosed as having schizophrenia. This study led to a major outcry in both the professional and lay public literatures about the validity of psychiatric diagnoses as represented in DSM-II (Farber, 1975; Rosenhan, 1975; Spitzer, 1975). Interestingly, the controversy centering around this paper has continued into the 21st century (Slater, 2004). The other important study was a collaborative paper in which British and American psychiatrists diagnosed patients based on videotaped interviews (Kendell et al., 1971). British psychiatrists used a range of diagnoses for these patients. American psychiatrists, however, persistently diagnosed all of the patients as having schizophrenia. American psychiatrists were embarrassed by this demonstration of their diagnostic sloppiness.

The development of the first two editions of DSM provided American psychiatry with the common nomenclature that it so desperately needed; however, in many ways,

DSM-II offered little more than names. The classification's value in terms of description was questionable; the concepts were poorly defined and often overlapped. Moreover, the descriptive prose used to define these concepts contained subjective phrases that necessarily led to disagreements among psychiatrists as to the meanings of the concepts, and therefore disagreement as to the appropriate diagnosis for a given patient (Ward, Beck, Mendelson, Mock, & Erbaugh, 1962). Diagnostic unreliability became a prominent criticism of psychiatric diagnosis (Wilson, 1993; Zubin, 1967). However, some authors have argued that the focus on reliability as a necessary area of scientific improvement in DSM-II was actually a smokescreen for political change by biologically oriented psychiatrists attempting to gain control of the classification system (Kirk & Kutchins, 1992).

Although the first two DSMs met, to some degree, the sociopolitical need for a unified classification system that would be applicable in outpatient clinical settings, critics of these systems expressed concerns about the social impact of the diagnostic labels provided therein. These critics tended to view mental illness and other forms of deviant behavior as largely politically defined and reinforced by social factors and agencies (Foucault, 1965; Schacht, 1985). Psychiatric diagnoses were considered to be self-fulfilling prophecies, in which patients adopted the behaviors implied by the labels. Another social concern, the negative stigma associated with many psychiatric diagnoses, was also used as a criticism of official diagnostic labels (Matza, 1968). In the Rosenhan (1973) study mentioned above, the individuals who faked the auditory hallucinations and were admitted to psychiatric hospitals reported that their normal behavior was often misconstrued as pathological by the psychiatric staff.

DSM-III

In the 1970s, concern over the diagnostic unreliability and conceptual ambiguity of DSM-II led to an increasing interest in developing more reliable diagnostic categories for the purpose of research in the mental health field. At Washington University in St. Louis, a group of psychiatrists took on the task of outlining what they considered to be the necessary steps to creating such a category: (1) description of the symptoms, (2) laboratory studies of the disorder, (3) distinguishing the category from other disorders, (4) follow-up studies of the disorder, and (5) family studies (Robins & Guze, 1970). These researchers emphasized that psychiatry is a branch of medicine, and that mental illnesses are discrete disease entities, the etiologies of which can be discovered through rigorous scientific study. This approach to psychopathology was viewed as being similar to Emil Kraepelin's late-19th-century conceptualization of mental disorders as biological diseases whose underlying essences could be understood through strict observations of the symptoms and course of each disorder. Because of this similarity, the Washington University group and those who ascribed to their ideas became known as the "neo-Kraepelinians" (Blashfield, 1984; Klerman, 1978); however, others have rejected this title (see Andreasen, 2007).

Adhering to Robins and Guze's (1970) proposed paradigm for developing diagnostic categories, the group at Washington University produced a paper in which they outlined diagnostic criteria for 14 mental disorders that they believed had sufficient empirical evidence supporting their scientific validity (Feighner et al., 1972). The Feighner criteria, so named for the first author of this collaborative work, did not set forth diagnostic descriptions in the prose form characteristic of the first two DSMs. Instead, the criteria for a syndrome were listed in terms of specific signs and symptoms. Each of the disorders proposed by Feighner and colleagues (1972) was considered to be a discrete group with set boundaries (i.e., a patient either met criteria for the diagnosis or did not). Feighner et al. recognized that not all symptoms of a disorder will be present in any one individual; therefore, their criteria stipulated a requisite number (e.g., five of nine) as necessary for a given diagnosis. Feighner and colleagues stated that a symptom could be considered present if a patient (1) sought the help of a physician for the symptom, (2) was sufficiently disabled by the symptom that normal functioning was disrupted, (3) took medication for the symptom, or (4) exhibited the symptom to such a degree that the examiner believed it to be clinically sig-

nificant. Three years after the publication of the Feighner criteria, the list of 14 disorders was expanded to 25 in an effort termed the Research Diagnostic Criteria (RDC; Spitzer, Endicott, & Robins, 1975).

The Feighner criteria and the RDC served as responses to criticisms of psychiatric diagnoses as unreliable and perhaps even invalid. Robert Spitzer, lead author of the RDC, provided evidence across multiple studies suggesting that for most diagnostic categories the RDC offered better reliability than DSM-II did (Meier, 1979). This movement represented a paradigmatic shift from an emphasis on psychoanalytic conceptualization, which had dominated North American psychiatry for the better part of a century, to a medical model of mental illness. Although the practice of psychiatric diagnosis had been the topic of much debate, the neo-Kraepelinians emphasized the importance of a universally accepted classification system as a means for conducting research and gaining knowledge in the area of psychopathology (Klerman, 1978). Having made a name for himself in the politics of the American Psychiatric Association through his work on DSM-II and his contributions to the resolution of the debate over the inclusion of homosexuality as a mental disorder, Spitzer was appointed to the position of chair of the Task Force on Nomenclature and Statistics, and was given the onerous task of organizing the third edition of DSM.

By taking the lead in the development of DSM-III (APA, 1980), Spitzer was in charge of choosing the members of the task force. Just as a U.S. president chooses a cabinet of like-minded individuals who will support and enact his decisions, the task force that created DSM-III consisted of individuals who adhered to Spitzer's ideology. Many of these individuals came from the Washington University group or had been trained at Washington University. Thus the task force could be understood as an "invisible college"—a group of individuals at diverse locations who were associated professionally and ideologically (Blashfield, 1984; Price, 1963). This is important to note because the final product, DSM-III, was intended for use by all mental health professionals; yet it was formulated around the views held by a particular group of individuals. One of Spitzer's goals for DSM-III was that it would be devoid of the psychoanalytic underpinnings present in the first two editions of the manual (Bayer & Spitzer, 1985). However, by adhering to the neo-Kraepelinian ideals of psychiatry as a branch of medicine, the DSM-III task force was using an implicit theoretical blueprint to construct its classification.

DSM-III (APA, 1980) differed from its predecessors on a number of levels. First, unlike DSM-I and DSM-II, DSM-III was an attempt at a strictly scientific classification system based on observable evidence rather than on a consensus of clinicians (Kendler, 1990). In particular, there was little attempt to merge the creation of DSM-III with changes in the mental disorders section of ICD-9. As a result, DSM-III was viewed by some in the international community as a typically American creation, in which U.S. psychiatrists assumed that they knew best and the rest of the world would follow after them (Stone, 1997).

Because DSM-III did not focus on consensus, the very process of writing the manual changed dramatically. In creating DSM-I and DSM-II, committees of experts had taken a rational approach to defining categories by basing their decisions on experience, precedent, and the utility of the final product. At that time, the scientific literature on mental disorders was scarce and had little impact on the DSMs. In contrast, the authors of DSM-III, in their effort to create an empirically based classification, attempted to organize the system around the existing literature. However, many of the debates that arose during this endeavor could not be solved by referring to the literature or scientific observation; thus some decisions were necessarily political in nature, reflecting the fact that science is not immune to political processes (Schacht, 1985).

The second major departure from DSM-I/DSM-II was in the actual presentation of the disorders. Instead of prose descriptions of syndromes that made implicit reference to the underlying "essence" of a disorder, DSM-III described disorders in terms of specific diagnostic criteria, following the model created by Feighner and colleagues (1972). This shift was an effort to improve diagnostic reliability, which had been less than optimal in the first two editions of the manual. Although the diagnostic criteria used in DSM-III were more specific than the vague-

ly worded prose definitions in DSM-I and DSM-II, these new definitions did not quite provide clear, operational specifications of when a symptom was or was not present. The actual operationalization of DSM-III diagnoses occurred through the development of a cottage industry that appeared in the 1980s and 1990s—the creation and publication of semistructured diagnostic interviews to assess patients (see APA, 2000b). These semistructured interviews were focused primarily on patients' verbal reports about their symptoms and were based to some extent on observations of patients' behaviors, eventually becoming the "gold standards" by which research diagnoses were made (see, e.g., Sohler & Bromet, 2003). The interclinician reliability of diagnoses made with these interviews was a vast improvement over diagnoses made using the earlier DSMs. However, semistructured interviews, particularly those focused on individual mental disorders, require special training and are time-consuming to administer. Therefore, they are rarely used in standard clinical practice.

DSM-III's third distinction was that it was a multiaxial classification (Williams, 1985). The categories in DSM-I and DSM-II were not based on a single organizing principle; instead, the organizing principles varied within parts of these classifications. Thus the organization of organic brain syndromes in the DSM-I was structured according to etiology. In contrast, the psychotic disorders were largely separated by views of the course of those disorders, and the DSM-I organization of mental retardation was a simple nominal scaling of the single dimension of intellectual functioning. The confusion inherent in a system without a consistent organizing principle led the authors of DSM-III to develop a diagnostic system in which patients would be diagnosed on five axes. This allowed clinicians to describe the patient's presentation in terms of psychopathological symptoms (Axis I), personality style and/or mental retardation (Axis II), relevant medical disorders (Axis III), environmental factors (Axis IV), and overall role impairment (Axis V). Interestingly, although this five-axis structure has been maintained across the subsequent editions of DSM, neither clinicians nor researchers seem particularly enamored of it (Fabrega, Ahn, & Mezzich, 1991).

Such drastic changes to the classification of psychopathology did not occur without criticisms. During the period of more than 5 years during which DSM-III was conceived, controversy surfaced over the exclusion of the term "neurosis" in the diagnostic categories. Spitzer and the neo-Kraepelinians recognized the term as stemming from psychoanalytic theory and therefore avoided it. The psychoanalysts lost the battle, and their influence in psychiatry since then has continued to diminish. In an attempt to parry criticisms by psychoanalysts that DSM-III was being created by a group of anti-psychoanalytic, pro-biological-model psychiatrists (which it was), Spitzer strenuously argued that DSM-III was intended to be theory-neutral and should only contain diagnostic terms that could be accepted by a broad spectrum of psychiatrists, regardless of their theoretical orientations (Bayer & Spitzer, 1985).

DSM-III-R

DSM-III was truly a revolutionary classification in both its scope and its impact (Stone, 1997). The changes made in DSM-III relative to its predecessors were large. DSM-I, for instance, was created by a committee of 7 people and was 130 pages long (including appendices). DSM-II had 10 members on its committee and was a total of 134 pages long. The DSM-III had a central task force of 19 members, with 13 auxiliary committees ranging in size from 4 to 18 members, and the entire manual was an astonishing 494 pages in length. The number of categories in the three editions increased from 106 in DSM-I to 182 in DSM-II to 265 in DSM-III.

The impact of DSM-III was also revolutionary. Within 5 years, it had been published in 16 other languages. Mezzich, Fabrega, Mezzich, and Coffman (1985) showed that DSM-III was used as frequently as, if not more frequently than, ICD-9 in a number of countries around the globe. Also, since the diagnostic criteria for many of the DSM-III categories had been created without any research to test them, a large number of studies appeared in the early 1980s that analyzed the DSM-III diagnostic criteria and made cogent comments on problems

with particular criteria (Skodol & Spitzer, 1987).

Only 7 years after the publication of DSM-III, the American Psychiatric Association published a revised version of the manual, known as DSM-III-R (APA, 1987). The original goal of this revision was to update the diagnostic criteria by integrating recent research findings, and the changes were intended to be minor. However, the modifications to the manual extended beyond criteria updates. Names of disorders were changed (e.g., paranoid disorder became delusional disorder); specific criteria were altered (e.g., the criteria for histrionic personality disorder); and some categories were reorganized (e.g., panic disorder was explicitly linked to agoraphobia). Six diagnostic categories, including ego-dystonic homosexuality, were dropped from the classification entirely, while approximately 33 new categories were added (e.g., body dysmorphic disorder, trichotillomania, and a whole new set of sleep disorders).

Despite the limited goals of DSM-III-R, major controversies developed as this system was being written. This time, the antagonists within the controversy were not psychoanalysts, but feminists, who attacked DSM-III-R as a male-generated classification that could have a negative impact on women. The focus of their concerns were three disorders: premenstrual syndrome (PMS), masochistic personality disorder, and paraphilic rapism (Blashfield, 1998). The arguments in this controversy were largely based on politics rather than science. The argument against PMS as a diagnostic category was that it blamed female anatomy for emotional states, and that if it were in fact a disorder, it would be more appropriately categorized as a gynecological syndrome than as a psychiatric one (Gallant & Hamilton, 1988; Spitzer, Severino, Williams, & Parry, 1989). Masochistic personality disorder was attacked as a way for psychiatry to blame victims of abuse for being abused (Caplan, 1985). Paraphilic rapism was thought to provide a psychiatric excuse for the behavior of serial rapists. Spitzer and his colleagues attempted to change the names of PMS and masochistic personality disorder to periluteal phase dysphoric disorder and self-defeating personality disorder, respectively, in order to deflect these attacks. They also proposed the addition of sadistic personality disorder, to counter the argument that self-defeating personality disorder would be used to blame the victim. Paraphilic rapism was deleted altogether. The feminists were ingenious in their political attacks. Rather than attempt to persuade particular subcommittees in the DSM-III-R process, the feminists waited until DSM-III-R was being considered by the American Psychiatric Association's Board of Directors. They had gained allies in this setting, particularly among some individuals who had become alienated by the entire DSM-III-R process. In the end, the American Psychiatric Association's Board of Trustees came up with a compromise that angered both sides: The three controversial disorders—sadistic personality disorder, self-defeating personality disorder, and late luteal phase dysphoric disorder—were all moved to a new appendix in DSM-III-R for "disorders needing further study."

DSM-IV

While ICD-10 was still in progress, the American Psychiatric Association decided to revise DSM-III once more, with the intention of coordinating this revision with ICD-10. The result, DSM-IV (APA, 1994), contained 354 categories (as compared to the 297 in DSM-III-R) and was 886 pages long. Not only did the classification provide diagnostic criteria, but supplementary material on the different disorders was also included. For example, in addition to the new criteria for histrionic personality disorder, DSM-IV contained three pages of ancillary information—including diagnostic features; associated features and disorders; specific culture, age, and gender features; prevalence; and differential diagnosis. The organization of the document was the same as that of DSM-III. Some diagnostic criteria were changed or expanded upon, but this iteration was explicitly designed to address the criticism that although the authors of DSM-III had sought to make the classification a purely scientific endeavor, in the end decisions still depended heavily on expert consensus. The revision process was structured in such a way as to limit the use of expert consensus and to maximize the impact of scientific inquiry. However, it was recognized that values are necessarily involved in diagnosis, and the authors of DSM-IV did

not overlook the important role of the sociopolitical function of classification. Also of concern was the clinical utility of the classification (Frances, Widiger, & Pincus, 1989). DSM-III was designed to provide a basis for more reliable research, but the DSMs are used in clinical practice and must be serviceable in that sense.

The steering committee, comprising 27 members (4 of whom were psychologists), oversaw the progress of 13 work groups, each with 5–16 members and usually at least 20 advisors. The guidelines for how the work groups were to go about their tasks were strictly laid out, in an effort to limit personal biases as much as possible (Blashfield, 1998). Interested parties may also look to the five-volume companion set of sourcebooks (which contain edited papers by members of the work groups) for a scholarly foundation for comprehending the decisions that were made. The work groups approached their tasks in three major steps. First, each group conducted extensive literature reviews of the disorders in their domain. Then the work groups requested access to descriptive data from researchers in the field. These data were reanalyzed by each work group, and the results of their analyses led to decisions regarding which criteria were in need of revision. Finally, a series of field trials were conducted for specific topics, the results of which affected decisions to alter diagnostic criteria.

The political process in the development of DSM-IV was quite different from what had occurred with DSM-III and DSM-III-R. In those earlier editions, Spitzer and the neo-Kraepelinians who worked with him had a firm hand in the work of the individual committees. In DSM-IV, a new psychiatrist, Allen Frances, became the chairperson (Frances, Pincus, Widiger, Davis, & First, 1990). Spitzer became an advisor to the DSM-IV process, but he had relatively little impact on its resulting changes. Frances delegated much more responsibility to the individual work groups that oversaw the details of the changes in DSM-IV. From a political standpoint, this division of labor probably contributed to the acceptance of the resulting classifications, but there were also problems with this process. Dividing psychopathology into discrete segments is not reasonable. Despite this, different work groups were reluctant to give up their rights to a particular domain, even when it might be better categorized elsewhere. Thus, for example, thoughtful decisions about whether schizotypal disorder should be viewed as a schizophrenia spectrum disorder or whether it should be classified as a personality disorder were hard to make. In the same way, making a decision about whether posttraumatic stress disorder (PTSD) should be classified as an anxiety disorder or as a form of an adjustment disorder (it is classified as the latter in ICD-10) were harder to make in this work-group-based organization.

Another concern about the DSM-IV work groups surfaced after the manual was published. Paralleling the changes in the DSMs with the creation of DSM-III was a major political shift within psychiatry away from psychoanalysis and toward a biologically based approach to mental health issues. In particular, the standard form of treatment offered by most psychiatrists by the end of the 1990s was the use of medications to relieve targeted symptoms and syndromes. Implicitly, the drug-producing companies had large amounts of money invested in treatments for particular mental disorders. Thus changes in the definitions or status of the diagnoses for which drug companies produced targeted drugs were major issues. In 2006, Cosgrove, Krimsky, Vijayaraghavan, and Schneider documented the extent to which members of the various DSM-IV work groups had received financial support from drug companies. According to their analysis, over half (56%) of these members had at least one financial tie to pharmaceutical companies. In fact, every member of the Mood Disorders Work Group and the Schizophrenia and Other Psychotic Disorders Work Group was associated financially with at least one drug company (Cosgrove et al., 2006).

DSM-IV-TR

Another issue that generated some controversy regarding the creation of the DSMs, especially after the publication of DSM-III, was the rapidity with which new editions were published. For researchers, particularly those relying on semistructured interviews to make diagnoses, these rapid changes presented serious problems (Zimmerman, 1988). With each new edition of DSM, changes in the diagnostic criteria necessitated changes in the semistructured interviews.

Often these changes, even when they seemed small, could have a substantial impact on decisions about who did or did not meet the definitions for the various mental disorders (Blashfield, Blum, & Pfohl, 1992). From DSM-III to DSM-III-R to DSM-IV, psychiatric diagnoses were talked about as being "moving targets" that impeded careful, systematic research on specific disorders.

In response to this criticism, Frances and others involved with DSM-IV promised that the American Psychiatric Association would wait for a longer period of time before attempting to create DSM-V. As this chapter is being written (early 2008), the work groups for DSM-V have been formed and are starting to work. The intent is for DSM-V to appear in May 2012 (APA, 2008). However, like politics, economics play a role in the production of the DSMs. The American Psychiatric Association made substantial amounts of money with the publication of DSM-III and the subsequent editions. All American mental health professionals and mental health settings must have copies of this classification, as must all major college and university libraries. The DSMs have represented a major product line for the American Psychiatric Association that this organization cannot afford to ignore.

DSM-IV-TR was different from the other editions in that it included no major, substantive changes from DSM-IV, in terms of either the categories or their definitions. Because the time between DSM-III-R and DSM-IV was so short, each work group primarily focused on revising diagnostic criteria and dealing with any major controversies of relevance to the diagnoses within its purview (e.g., whether self-defeating personality disorder should be moved from the appendix to the personality disorders section). Work on the supporting textual material describing the various mental disorders was not completed until later. These changes were incorporated into DSM-IV-TR. In many ways, DSM-IV-TR does not deserve its designation as a separate edition; however, that designation did lead to substantial new sales of DSM.

DSM-V and Beyond

The fifth edition of the DSM is currently expected to be published in May 2012, as noted above. One goal for DSM-V is to expand the research base for the diagnoses, and in particular to develop a priori classificatory questions that can be researched, rather than having to answer post hoc classificatory questions with existing data sets. To this end, the American Psychiatric Association published *A Research Agenda for DSM-V* (Kupfer, First, & Regier, 2002), which included a set of white papers outlining research plans for neuroscience; developmental science; the personality and relational disorders; mental disorders and disability; and culture and psychiatric diagnosis. This volume was followed by the more theoretical *Advancing DSM: Dilemmas in Psychiatric Diagnosis* (Phillips, First, & Pincus, 2003), which addressed various issues: disorder versus nondisorder; the DSM diagnostic groupings; laboratory testing and neuroimaging; schizotaxia and schizophrenia; subthreshold mental disorders; multiaxial assessment; personality disorders; and relationship disorders. Even the foci of these chapters highlight the values of DSM-V: neuroscience, personality disorders, and the underlying mechanisms of disorders. From 2004 to 2007, invited conferences were held to discuss the current research for individual groups of disorders (e.g., mood disorders, personality disorders, and anxiety disorders). In April 2006, David Kupfer and Darrel Regier were appointed as Chair and Vice-Chair of the DSM-V task force; in July 2007, DSM-V work group chairs were appointed. Work group members were appointed in April 2008.

In light of the steadily increased focus on the scientific values of classification across the DSMs as documented above, we would expect that scientific issues will remain the foci of the DSM-V process. First and colleagues (2004) noted that a major change in the DSM-V should be an increased emphasis on clinical utility. To promote this more pragmatic focus, we comment on two types of research studies that might be considered in future efforts to improve the clinical utility of the DSMs.

Clinical Utility

Making the *Diagnostic and Statistical Manual of Mental Disorders* useful to clinicians has been designated as an important goal by the American Psychiatric Association. The

introduction to DSM-IV-TR (APA, 2000) indicates, "Our highest priority has been to provide a helpful guide to clinical practice" (p. xxiii). In addition, in one chapter of *A Research Agenda for DSM-V*, the joint American Psychiatric Association–National Institute of Mental Health committee charged with outlining a research agenda for DSM-V suggested that examining clinicians' conceptualizations of DSM-IV disorders is important for creating a reliable and valid DSM (Rounsaville et al., 2002). Furthermore, the American Psychiatric Association's ad hoc committee on psychiatric diagnosis and assessment has called for studies investigating DSM's clinical utility (First et al., 2004).

Defining clinical utility, developing its theoretical basis, and designing a research agenda to investigate it, however, have proven difficult. First and colleagues (2004) have taken the lead in defining clinical utility as "the extent to which the DSM assists clinical decision makers in fulfilling the various clinical functions of a psychiatric classification system" (p. 947), and have defined these functions as including conceptualizing diagnostic entities, communicating clinical information, using diagnostic categories and criteria sets in clinical practice, choosing effective interventions to improve clinical outcomes, and predicting future clinical management needs. First and colleagues have also outlined how changes to clinical utility can be assessed: "Improvements to clinical utility can be measured in terms of 1) their impact on the use of DSM, 2) their enhancement of clinical decision making, and 3) whether they lead to improvement in clinical outcomes" (2004, p. 953).

One Method of Developing Utility

A few researchers have been applying principles from cognitive psychology to understand how clinicians use diagnostic concepts when discussing patients. For instance, we have been involved in a set of studies looking at the discrepancies between the ways in which clinicians think about mental disorders and the actual structure of the DSM (Flanagan & Blashfield, 2006, 2007; Flanagan, Keeley, & Blashfield, 2008).

In this research, 76 psychologists and psychiatrists were asked to sort 67 DSM-IV mental disorders into groups of "similar diagnoses" or diagnoses that had "similar treatments." The exact disorders used in this research are listed in Table 2.1. Then these clinicians were asked to indicate relationships among the groups of diagnoses by either making progressively larger and then smaller groups of disorders (i.e., creating hierarchical "nodes"), or placing the groups of diagnoses next to each other in two-dimensional space. The amount of distance between each pair of diagnoses was then represented by the hierarchical node at which two diagnoses were first put in the same group, or the number of inches between each pair of diagnoses.

These distances were then assembled into a dissimilarity matrix and submitted to two types of hierarchical agglomerative data analysis: single-linkage cluster analysis and Ward's method of cluster analysis. The goal of this research was to compare the similarities between clinicians' sortings of disorders and other published suggested changes to DSM. In the absence of a more rigorous empirical investigation of clinical utility, this research could show the similarities among the proposed suggestions and the way clinicians think about mental disorders. The Ward's method analysis of clinicians' sortings of DSM disorders appears in Table 2.1. In this table, there are three primary groupings: The first group has a strong medical component; the second group includes the more traditional serious mental illnesses; and the third group includes the more commonly diagnosed disorders in psychiatry. What is most interesting about this organization is that these groupings are more similar to the structure of DSM-I (APA, 1952) or DSM-II (APA, 1968) than they are to that of more recent versions of DSM. The older versions of DSM were organized by simulating a clinician's decision-making process. First the organic disorders were divided from the nonorganic disorders; then the nonorganic disorders were divided into the psychotic disorders versus the neuroses; and then the personality disorders were separated from the neuroses. Similarly, when making a diagnostic decision, a clinician would first decide whether a disorder was organically based, then whether it had a psychotic component, and then whether the disorder had a neurotic or a personality basis. Notice that the clinicians' view of mental disorders does not include the Axis I–Axis II distinction or the separation of adult from childhood dis-

TABLE 2.1. Clinicians' Groupings of DSM-IV Disorders According to Ward's Method

I. Organic mental disorders
  A. Disorders often seen in children
    1. Encopresis
    2. Enuresis
    3. Sleepwalking disorder
    4. Sleep terror disorder
    5. Nightmare disorder

  B. Medically related disorders
    1. Substance-induced sleep disorder
    2. Psychotic disorder due to a medical condition
    3. Sleep disorder due to a medical condition
    4. Circadian rhythm sleep disorder
    5. Primary insomnia
    6. Primary hypersomnia

  C. Neurological disorders
    1. Delirium
    2. Dementia

II. Serious mental illnesses
  A. Substance use disorders
    1. Substance dependence
    2. Substance abuse
    3. Substance-induced disorder
    4. Substance-induced psychotic disorder

  B. Schizophrenia-related disorders
    1. Disorders with psychotic features
      a. Paranoid
      b. Schizotypal
      c. Schizoid
    2. Schizophrenic disorders
      a. Delusion disorder
      b. Brief psychotic disorder
      c. Schizophrenia
      d. Schizoaffective disorder
      e. Shared psychotic disorder

  C. Mood disorders
    1. Cyclothymia
    2. Dysthymia
    3. Major depressive disorder
    4. Depression or mania with psychotic features
    5. Bipolar I
    6. Bipolar II

III. Commonly treated psychiatric disorders
  A. Externalizing disorders
    1. Childhood forms
      a. Tourette's disorder or other tic disorders
      b. Attention-deficit/hyperactivity disorder
      c. Oppositional defiant disorder
      d. Conduct disorder
    2. Adult forms
      a. Intermittent explosive disorder
      b. Pathological gambling
      c. Pyromania
      d. Kleptomania
      e. Antisocial personality disorder

(cont.)

TABLE 2.1. *(cont.)*

- B. Internalizing disorders
  - 1. Personality-related disorders
    - a. Altered-mental-state disorders
      - (1) Dissociative amnesia
      - (2) Amnestic disorders
      - (3) Depersonalization disorder
      - (4) Dissociative identity disorder
    - b. Personality disorders
      - (1) Avoidant personality disorder
      - (2) Obsessive–compulsive personality disorder
      - (3) Dependent personality disorder
      - (4) Histrionic personality disorder
      - (5) Narcissistic personality disorder
      - (6) Borderline personality disorder
  - 2. Anxiety-related disorders
    - a. Body-focused disorders
      - (1) Bulimia nervosa
      - (2) Anorexia nervosa
      - (3) Focus on fear of disease (hypochondriasis, body dysmorphic disorder)
      - (4) Focus on physical symptoms (somatization disorder, conversion disorder, pain disorder)
    - b. Anxiety disorders
      - (1) Trichotillomania
      - (2) Obsessive–compulsive disorder
      - (3) Generalized anxiety disorder
      - (4) Phobias
      - (5) Panic disorder
      - (6) Posttraumatic stress disorder
      - (7) Separation anxiety disorder

Note. *Gender identity disorder*, *paraphilias*, *sexual dysfunctions*, and *adjustment disorder* were removed from this cluster analysis, since they were deemed to be outliers by single-linkage cluster analysis. From Flanagan, Keeley, and Blashfield (2008, p. 695). Copyright 2008 by the American Psychological Association. Adapted by permission.

orders, both of which are part of the hierarchical structure of DSM-IV.

Also of note in the clinicians' sortings is that at the lower levels of the hierarchy, the DSM groupings were preserved. For instance, the DSM-IV mood disorders were grouped together (as were the dissociative disorders, the anxiety disorders, and the psychotic disorders). However, there were interesting exceptions to this trend. Antisocial personality disorder was grouped with other acting-out disorders rather than with the Cluster B personality disorders. Separation anxiety disorder was included with the other anxiety disorders rather than with the childhood disorders. The Cluster A personality disorders (i.e., paranoid, schizoid, schizotypal) were grouped with the psychotic disorders rather than with the other personality disorders, and the DSM-IV eating disorders and somatoform disorders were grouped together into a "body-focused disorders" group.

These groupings are at variance with suggestions in the current literature for reorganizing the mental disorders for DSM-V. For instance, based on genetic and structural/comorbidity data, Watson (2005) suggests reorganizing the DSM-IV mood and anxiety disorders into three groups: "bipolar disorders," "distress disorders," and "fear disorders." In that regrouping, major depressive disorder, dysthymia, PTSD, and generalized anxiety disorder are classified as distress disorders. However, clinicians in the study mentioned above kept the DSM-IV mood and anxiety disorders in separate groups. These results suggest that a reorganization such as Watson's would not have high clinical utility.

Another suggestion in the current literature is designating an obsessive–compulsive spectrum of disorders (Castle & Phillips, 2006; Hollander, 2006), which would include neurological disorders (e.g., autism), preoccupations with bodily sensations or ap-

pearance (e.g., body dysmorphic disorder), and impulsive disorders (e.g., trichotillomania) in addition to obsessive–compulsive disorder. In the clinicians' groupings described above, trichotillomania was grouped with obsessive–compulsive disorder, but autism and body dysmorphic disorder were not. These results suggest that an obsessive–compulsive spectrum of disorders would also not have high clinical utility.

A further suggestion in the literature is to return to Achenbach's (1966) original distinction between externalizing and internalizing disorders. Krueger and Markon (2006) used meta-analytic methods to group DSM disorders along this continuum. Clinicians' grouping of the DSM-IV impulse control disorders, childhood acting-out disorders, and antisocial personality disorder into an externalizing spectrum of disorders is similar to current suggestions. However, the clinicians' inclusion of the DSM-IV dissociative, personality, somatoform, and eating disorders on the internalizing spectrum does not match current suggestions. Despite this latter finding, these results suggest that organizing at least some of the DSM disorders along an externalizing–internalizing spectrum has the potential to increasing the utility of the DSMs for clinicians.

A Second Method of Developing Utility

Another innovative and potentially informative approach to a classification of psychopathology is to use patients' descriptions of their subjective experiences of these disorders to refine or perhaps redefine the diagnostic criteria, so that they more accurately reflect the phenomenology of mental disorders. This change could help clinicians to better understand the true experiences of their patients and refine ways to help them. In order to illustrate our argument, we use borderline personality disorder as an example.

There is evidence that the DSM-IV criteria for borderline personality disorder do not match people's subjective experience of the disorder. Miller (1994) investigated the subjective experiences of 10 individuals who met DSM-III-R diagnostic criteria for this disorder. Participants were given a minimally structured interview in which they were asked to tell the researcher about themselves and their everyday lives. Through this methodology, Miller was able to obtain a rich understanding of the ways in which each person's symptoms affected his or her life, and to assess the extent to which these experiences matched the depiction of the disorder in the DSM.

Despite the lack of structure in Miller's interview and the heterogeneity of symptoms of people with borderline personality disorder, this study found remarkable similarities across participant narratives. In DSM-IV-TR, people with borderline personality disorder are described as having an "identity disturbance: markedly and persistently unstable self-image or sense of self" (p. 710). In Miller's (1994) study, however, participants described a cohesive identity and sense of self, although the explicit diagnosis of borderline personality disorder was notably absent from their self-descriptions. Across approximately six 90-minute interviews, participants were able to describe clearly and consistently their preferences and who they were or wanted to be. They made distinctions between those times when they were able to be themselves and those times when they were not, due to trying to please others or to hide their insecurities. They further described themselves as estranged from others and inadequate in relation to social standards. As Miller concluded, "rather than having an impaired sense of self, they seemed to have a sense of themselves as impaired" (p. 1216). In response to these feelings, participants would invoke a variety of strategies for feeling better, which to an observer might look like identity diffusion and impulsivity (e.g., making changes in appearance, adopting different lifestyles). These failed strategies were compounded by a reluctance to reveal themselves, especially to therapists, and involuntarily choosing to be thought of as having a lack of identity rather than to show their flawed identities. Central to their narratives were themes of struggling through life and attempting to assuage feelings of despair. Subsequently, perhaps a more valid diagnostic criterion for borderline personality disorder would be "sense of self as estranged and inadequate" rather than "identity disturbance: markedly and persistently unstable self-image or sense of self." This contrast suggests that there may be important, accuracy-increasing differences

between patients' subjective experiences and the perceptions of these same experiences by others (i.e., clinicians or researchers).

Another of Miller's (1994) findings relates to conceptions of the emotional lives of people with borderline personality disorder. According to DSM-IV-TR, their emotional experience is characterized by either "chronic feelings of emptiness" or "affective instability due to a marked reactivity in mood (e.g., intense episodic dysphoria, irritability, or anxiety usually lasting a few hours and only rarely more than a few days)" (p.710). Rather than reporting feelings of emptiness or marked changes in mood, participants in Miller's study described relentless feelings of despair. Each person described an ever-present wish not to be alive and made at least one reference to spending hours in bed crying. Miller further commented that the pain felt by persons with this disorder was hard to capture in words: "the sense of emotional pain conveyed by these patients was overwhelming . . . much of the impact [of their words] may be lost without hearing the pain in their voices or experiencing the redundancy of such comments in the narratives" (p. 1217). This study suggests that what may appear to an observer as "emptiness" or "affective instability" may be experienced subjectively by a person with the disorder as chronic emotional pain and despair.

These examples contrasting the diagnostic criteria of borderline personality disorder with the subjective experiences of people who have this disorder suggest that in order to validly represent mental disorders, and to help clinicians diagnose and treat people with mental disorders, DSM must accurately reflect the inner experiences of people with mental disorders and offer clinicians a window into these experiences. Paradoxically, then, using patient-subjective based criteria could also address our other primary concern for DSM-V—increasing the clinical utility of the manual. An important, although perhaps unintended, effect of including patient-subjective criteria could be an increase in empathic "bridges" between clinicians and their patients. If clinicians can arrive at a more accurate understanding of patients' own experiences of mental disorders, they may be more likely to achieve the degree of empathic reflection that has been found to be one of the most important factors predicting positive treatment relationships and outcomes (Gehrs & Goering, 1994; Horvath & Symonds, 1991; Neale & Rosenheck, 1995).

Postscript

We have begun this chapter by discussing the early ICDs and DSMs, along with the clinical/pragmatic goals that dominated these early classifications. As we have moved through discussions of more recent editions of DSM, the story has shifted to discussions of empirical research, political struggles, economics, and changes occurring both within and between the mental health professions.

The three of us who are writing this chapter are clinical psychologists. With different levels of involvement, all three of us have spent considerable portions of our time functioning as clinicians—a role in which our primary responsibility is the care of our clients. Although we have all devoted considerable energy to looking at the classificatory issues in psychopathology, none of us as clinicians are happy with the current, official DSM-IV(-TR) system. To us as clinicians, the DSMs seem mostly irrelevant to what we do. In our various clinical roles, we are required to make diagnoses, and we do so. But we rarely find that process informative.

Our dissatisfaction with what the DSMs have evolved to become can be summarized by referring to a famous 1960s movie titled *Who's Afraid of Virginia Woolf?* This movie is about one night in the lives of two couples who meet and interact with each other. The emotional intensity of *Who's Afraid of Virginia Woolf?* is almost unequaled by any other American-made movie. The level of anger, interpersonal destructiveness, and relentless psychological assault is hard to explain. An important psychological puzzle in this movie is how the main characters in the movie can maintain some of level of homeostasis in their dance of anger with each other, so that the interactive pattern continues on and on.

The Wedding and colleagues (2005) book mentioned at the start of this chapter, about the representation of psychopathology in

movies, discusses *Who's Afraid of Virginia Woolf?* as depicting "other psychotic disorders." The reason for this diagnostic assignment is that the main couple in the movie (played by Elizabeth Taylor and Richard Burton who, in their nonacting lives, were married twice to each other and then divorced twice) appear to hold a joint delusional belief in the existence of a son who was never born. Although this diagnosis may be defensible, this diagnosis (or any other DSM label) seems irrelevant to the profound psychological issues represented in the interactive pattern among the four characters in the movie.

This short historical overview of the DSMs has begun with a comment on the goals of psychiatric classification. We have listed five purposes of such classification: (1) nomenclature, (2) information retrieval, (3) description, (4) prediction, and (5) concept formation for a theory (or theories) of psychopathology. Forty years ago, when one of us (Roger K. Blashfield) wrote about these purposes, he viewed them as being hierarchical (i.e., lower-order purposes had to be met in order for the higher-order purposes to be met). His opinion at the time was that DSM-II functioned satisfactorily as a nomenclature and as a basis for information retrieval. The major scientific problem of the time was to create a classification that was descriptively useful.

Now our opinion has changed. The DSMs have failed to meet any of these goals, starting with the most fundamental of being a nomenclature. To understand this claim, think again of *Who's Afraid of Virginia Woolf?* and the multiaxial diagnoses for the central characters in this movie. If you have seen this film, are those what you remember about it—the characters' diagnoses? Probably not. In the same way, what is striking about a movie like *Ordinary People* is that the clinician in the movie spends little or no time trying to come up with a DSM diagnosis. And even if the clinician were to decide on a diagnosis, we don't think that such a diagnosis would have much (if any) influence on the process of treatment depicted in that movie, or on its resolution as shown in the last therapy scene of the movie.

Psychiatric diagnoses have become institutionalized, and they serve as crude, but somewhat useful, leads for scientific literatures. However, diagnostic concepts trivialize the complexity of people, their lives, and what might help them—just as movies like *Who's Afraid of Virginia Woolf?* and *Ordinary People* are trivialized when thought of simply in terms of the diagnostic concepts they potentially represent. Psychopathology and its manifestations in people are not trivial matters. People with psychopathology and the clinicians who treat them deserve better help than the editions of the DSM have offered.

References

Achenbach, T. M. (1966). The classification of children's psychiatric symptoms: A factor-analytic study. *Psychological Monographs*, *80*(7, Whole No. 615).

American Psychiatric Association (APA). (1952). *Diagnostic and statistical manual of mental disorders*. Washington, DC: Author.

American Psychiatric Association (APA). (1968). *Diagnostic and statistical manual of mental disorders* (2nd ed.). Washington, DC: Author.

American Psychiatric Association (APA). (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

American Psychiatric Association (APA). (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., revised). Washington, DC: Author.

American Psychiatric Association (APA). (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

American Psychiatric Association (APA). (2000a). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.

American Psychiatric Association (APA). (2000b). *Handbook of psychiatric measures*. Washington, DC: Author.

American Psychiatric Association (APA). (2008). *DSM-V: The future manual*. Washington, DC: Author. Retrieved from *www.psych.org/MainMenu/Research/DSMIV/DSMV.aspx*.

Andreasen, N. (2007). DSM and the death of phenomenology in America: An example of unintended consequences. *Schizophrenia Bulletin*, *33*, 108–112.

Bayer, R. (1987). *Homosexuality and American psychiatry: The politics of diagnosis*. Princeton, NJ: Princeton University Press.

Bayer, R., & Spitzer, R. L. (1985). Neurosis,

psychodynamics and DSM-III: A history of a controversy. *Archives of General Psychiatry*, *42*, 187–196.

Blashfield, R. K. (1984). *The classification of psychopathology: Neo-Kraepelinian and quantitative approaches*. New York: Plenum Press.

Blashfield, R. K. (1998). Diagnostic models and systems. In A. S. Bellack & M. Hersen (Eds.), *Comprehensive clinical psychology* (pp. 57–80). New York: Pergamon Press.

Blashfield, R. K., Blum, N., & Pfohl, B. (1992). The effects of changing Axis II diagnostic criteria. *Comprehensive Psychiatry*, *33*, 245–252.

Blashfield, R. K., & Draguns, J. G. (1976). Toward a taxonomy of psychopathology: The purpose of psychiatric classification. *British Journal of Psychiatry*, *129*, 574–583.

Caplan, P. J. (1985). *The myth of women's masochism*. New York: Dutton.

Castle, D. J., & Phillips, K. A. (2006). Obsessive–compulsive spectrum of disorders: A defensible construct? *Australian and New Zealand Journal of Psychiatry*, *40*, 114–120.

Cosgrove, L., Krimsky, S., Vijayaraghavan, M., & Schneider, L. (2006). Financial ties between DSM-IV panel members and the pharmaceutical industry. *Psychotherapy and Psychosomatics*, *75*(3), 154–160.

Fabrega, H., Ahn, C., & Mezzich, J. (1991). Can multiaxial diagnosis predict future use of psychiatric hospitalization? *Hospital and Community Psychiatry*, *42*, 920–924.

Farber, I. E. (1975). Sane and insane: Constructions and misconstructions. *Journal of Abnormal Psychology*, *84*(6), 589–620.

Feighner, J. P., Robins, E., Guze, S. B., Woodruff, R. A., Winokur, G., & Munoz, R. (1972). Diagnostic criteria for use in psychiatric research. *Archives of General Psychiatry*, *26*, 57–63.

First, M. B., Pincus, H. A., Levine, J. B., Williams, J. B. W., Ustun, B., & Peele, R. (2004). Clinical utility as a criterion for revising psychiatric diagnoses. *American Journal of Psychiatry*, *161*, 946–954.

Flanagan, E. H., & Blashfield, R. K. (2006). Do clinicians see Axis I and Axis II as different kinds of disorders? *Comprehensive Psychiatry*, *47*, 496–502.

Flanagan, E. H., & Blashfield, R. K. (2007). Clinicians' folk taxonomies of mental disorders. *Journal of Philosophy, Psychiatry, and Psychology*, *14*, 249–270.

Flanagan, E. H., Keeley, J., & Blashfield, R. K. (2008). An alternative hierarchical organization of the mental disorders of DSM-IV. *Journal of Abnormal Psychology*, *117*, 693–698.

Foucault, M. (1965). *Madness and civilization*. New York: Vintage Books.

Frances, A., Pincus, H. A., Widiger, T. A., Davis, W. W., & First, M. B. (1990). DSM-IV: Work in progress. *American Journal of Psychiatry*, *147*, 1439–1448.

Frances, A., Widiger, T. A., & Pincus, H. A. (1989). The development of the DSM-IV. *Archives of General Psychiatry*, *46*, 373–375.

Gallant, S. J., & Hamilton, J. A. (1988). On a premenstrual psychiatric diagnosis: What's in a name? *Professional Psychology*, *19*, 271–278.

Gehrs, M., & Goering, P. (1994). The relationship between the working alliance and rehabilitation outcomes of schizophrenia. *Psychosocial Rehabilitation Journal*, *18*, 43–54.

Grob, G. N. (1991). Origins of DSM-I: A study in appearance and reality. *American Journal of Psychiatry*, *148*, 421–431.

Hempel, C. G. (1965). *Aspects of scientific explanation and other essays in the philosophy of science*. New York: Free Press.

Hollander, E. (2006). Obsessive–compulsive disorder and spectrum across the life span. *International Journal of Psychiatry in Clinical Practice*, *9*, 79–86.

Horvath, A. O., & Symonds, D. (1991). Relation between working alliance and outcome in psychotherapy. *Journal of Counseling Psychology*, *38*, 139–149.

Houts, A. C. (2000). Fifty years of psychiatric nomenclature. *Journal of Clinical Psychology*, *56*, 935–967.

Kendell, R. E. (1975). *The role of diagnosis in psychiatry*. Oxford, UK: Blackwell Scientific.

Kendell, R. E., Cooper, J. E., Gourlay, A. J., Copeland, J. R. M., Sharpe, L., & Gurland, B. J. (1971). Diagnostic criteria of American and British psychiatrists. *Archives of General Psychiatry*, *25*, 121–130.

Kendler, K. S. (1990). Toward a scientific psychiatric nosology: Strengths and limitations. *Archives of General Psychiatry*, *47*, 969–973.

Kirk, S. A., & Kutchins, H. (1992). *The selling of DSM*. New York: Aldine de Gruyter.

Klerman, G. L. (1978). The evolution of a scientific nosology. In J. C. Shershow (Ed.), *Schizophrenia: Science and practice*. Cambridge, MA: Harvard University Press.

Kreitman, N. (1961). The reliability of psychiatric diagnosis. *Journal of Mental Science*, *107*, 878–886.

Krueger, R. F., & Markon, K. E. (2006). Under-

standing psychopathology: Melding behavior genetics, personality, and quantitative psychology to develop an empirically-based model. *Current Directions in Psychological Science*, *15*, 113–117.

Kupfer, D. J., First, M. B., & Regier, D. A. (Eds.). (2002). *A research agenda for DSM-V*. Washington, DC: American Psychiatric Association.

Kutchins, H., & Kirk, S. A. (1997). *Making us crazy*. New York: Free Press.

Matarazzo, J. D. (1978). The interview: Its reliability and validity in psychiatric diagnosis. In G. G. Wolman (Ed.), *Clinical diagnosis of mental disorders*. New York: Plenum Press.

Matza, D. (1968). *On becoming deviant*. Englewood Cliffs, NJ: Prentice-Hall.

Meier, A. (1979). The Research Diagnostic Criteria: Historical background, development, validity, and reliability. *Canadian Journal of Psychiatry*, *24*, 167–178.

Menninger, K. (1969). Sheer verbal Mickey Mouse. *International Journal of Psychiatry*, *7*, 415.

Mezzich, J. E., Fabrega, H., Mezzich, A. C., & Coffman, G. A. (1985). International experience with the DSM-III. *Journal of Nervous and Mental Disease*, *173*, 738–741.

Miller, S. G. (1994). Borderline personality disorder from the patient's perspective. *Hospital and Community Psychiatry*, *45*, 1215–1219.

Millon, T. (1986). On the past and future of the DSM-III: Personal recollections and projections. In T. Millon & G. L. Klerman (Eds.), *Contemporary directions in psychopathology: Toward the DSM-IV* (pp. 29–70). New York: Guilford Press.

Neale, M. S., & Rosenheck, R. A. (1995). Therapeutic alliance and outcome in a VA intensive case management program. *Psychiatric Services*, *46*, 719–721.

Price, D. S. (1963). *Little science, big science*. New York: Cambridge University Press.

Phillips, K. A., First, M. B., & Pincus, H. A. (Eds.). (2003). *Advancing DSM: Dilemmas in psychiatric diagnosis*. Washington, DC: American Psychiatric Association.

Robins, E., & Guze, S. B. (1970). Establishment of diagnostic validity in psychiatric illness: Its application to schizophrenia. *American Journal of Psychiatry*, *126*, 107–111.

Rosenhan, D. L. (1973). On being sane in insane places. *Science*, *179*, 250–258.

Rosenhan, D. L. (1975). The contextual nature of psychiatric diagnosis. *Journal of Abnormal Psychology*, *84*(5), 462–474.

Rounsaville, B. J., Alarcon, R. D., Andrews, G., Jackson, J. S., Kendell, R. E., & Kendler, K. S. (2002). Basic nomenclature issues for DSM-V. In D. J. Kupfer, M. B. First, & D. A. Regier (Eds.), *A research agenda for DSM-V*. Washington, DC: American Psychiatric Association.

Sadler, J. Z. (2005). *Values and psychiatric diagnosis*. New York: Oxford University Press.

Schacht, T. E. (1985). DSM-III and the politics of truth. *American Psychologist*, *40*, 513–521.

Shorter, E. (1997). *A history of psychiatry*. New York: Wiley.

Skodol, A. E., & Spitzer, R. L. (1987). *An annotated bibliography of DSM-III*. Washington, DC: American Psychiatric Press.

Slater, L. (2004). *Opening Skinner's box*. New York: Norton.

Sohler, N. L., & Bromet, E. J. (2003). Does racial bias influence psychiatric diagnoses assigned at first hospitalization? *Social Psychiatry and Psychiatric Epidemiology*, *38*, 463–472.

Spitzer, R. L. (1975). On pseudoscience in science, logic in remission, and psychiatric diagnosis: A critique of Rosenhan's "On being sane in insane places." *Journal of Abnormal Psychology*, *84*(5), 442–452.

Spitzer, R. L., Endicott, J., & Robins, E. (1975). *Research Diagnostic Criteria (RDC) for a selected group of functional disorders*. New York: New York State Psychiatric Institute.

Spitzer, R. L., Severino, S., Williams, J. B. W., & Parry, B. L. (1989). Late luteal phase dysphoric disorder and DSM-III-R. *American Journal of Psychiatry*, *146*, 892–897.

Spitzer, R. L., & Wilson, P. T. (1968). A guide to the new nomenclature. In *Diagnostic and statistical manual of mental disorders* (2nd ed., pp. 120–134). Washington, DC: American Psychiatric Association.

Stengel, E. (1959). Classification of mental disorders. *Bulletin of the World Health Organization*, *21*, 601–663.

Stone, M. H. (1997). *Healing the mind: A history of psychiatry from antiquity to the present*. New York: Norton.

Ward, C. H., Beck, A. T., Mendelson, M., Mock, J. E., & Erbaugh, J. K. (1962). The psychiatric nomenclature: Reasons for diagnostic disagreement. *Archives of General Psychiatry*, *7*, 198–205.

Watson, D. (2005). Rethinking the mood and anxiety disorders: A quantitative hierarchical model for DSM-V. *Journal of Abnormal Psychology*, *114*(4), 522–536.

Wedding, D., Boyd, M. A., & Niemiec, R. (2005).

Movies and mental illness: Using films to understand psychopathology (2nd ed.). Cambridge, MA: Hogrefe & Huber.

Williams, J. B. W. (1985). The multiaxial system of DSM-III: Where did it come from and where should it go? I. Its origins and critiques. *Archives of General Psychiatry*, *42*, 175–180.

Wilson, M. (1993). DSM-III and transformation of American psychiatry: A history. *American Journal of Psychiatry*, *150*, 399–410.

World Health Organization (WHO). (1948). *Manual of international statistical classification of diseases, injuries, and causes of death* (6th rev.). Geneva: Author.

World Health Organization (WHO). (1967). *Manual of the international statistical classification of diseases, injuries, and causes of death* (8th rev.). Geneva: Author.

World Health Organization. (1992a). *The ICD-10 classification of mental and behavioural disorders: Clinical descriptions and diagnostic guidelines*. Geneva: Author.

World Health Organization (WHO). (1992b). *International statistical classification of diseases and related health problems* (10th rev.). Geneva: Author.

World Health Organization (WHO). (1993). *The ICD-10 classification of mental and behavioural disorders: Diagnostic criteria for research*. Geneva: Author.

Zimmerman, M. (1988). Why are we rushing to publish DSM-IV? *Archives of General Psychiatry*, *45*, 1135–1138.

Zubin, J. (1967). Classification of behavior disorders. *Annual Review of Psychology*, *18*, 373–406.

CHAPTER 3

On the Wisdom of Considering Culture and Context in Psychopathology

Joseph P. Gone
Laurence J. Kirmayer

Since the advent of the "new cross-cultural psychiatry" in the late 1970s (Kirmayer, 2006; Kleinman, 1977; Littlewood, 1990), psychiatric researchers have distinguished between the historical enterprise of exporting conventional categories of "mental disorder" throughout the world's diverse cultural communities for the purposes of comparative study, and the more recent commitment to examining the cross-cultural viability and coherence of such categories within culturally local frameworks of distress, illness, and dysfunction (Kirmayer, 2007a). Such reflexive awareness concerning the Western cultural foundations of the categories and constructs used in cross-cultural studies of psychopathology parallels a broader conceptual and methodological revolution in the social sciences (Rabinow & Sullivan, 1987). This reflexive stance has encouraged attention to the ways in which science and technical practices are embedded in local and international systems of power and knowledge, and has urged caution in generalizing or applying dominant approaches to disparate cultures and communities.

And yet, as helping professions rooted in an understanding of the human condition, psychiatry and psychology aim for theories of psychopathology that can be used across social and cultural contexts. An international diagnostic nosology should provide a common language allowing psychiatrists everywhere to exchange knowledge about specific patients, have ready access to current technical approaches, and contribute to the advance of psychiatric science. Unfortunately, this project of a global scientific psychiatry tends to view culture as a distraction from the project of developing a body of universal knowledge. That is, cultural diversity becomes an obstacle to scientific research and delivery of care, or else a matter of trivial differences—of "window dressing" on the essential core of universal human experience that might ground a universal nosology.

This more dismissive view of the relevance of culture for world psychiatry is part of the legacy of European empire, in that it assumes that the pertinent categories, concepts, principles, and practices—constructs that emerged almost exclusively from certain subpopulations or social strata within a handful of European and North American societies—constitute a universal, transcen-

dent, ahistorical, and "culture-free" basis for recognizing "natural kinds" (i.e., the categories that are immediately given to perception or that can be readily discerned by "carving nature at its joints") within the domain of psychopathology. This ethnocentrism is also evident in the way non-Western cultures are frequently construed by Westerners: There is a tendency to dichotomize self and other, to view the world as "us and them." We have *knowledge*, while they have *beliefs*; we see things as they truly are, while they are deluded by their stubborn traditions and superstitions. The imperialist roots of this thinking are evident in the resultant asymmetries in valuing truth claims: European American epistemological practices yield transcendent technical knowledge, while other epistemological traditions yield mere folk knowledge comprising beliefs rather than truths about the world. In actual fact, however, the concepts and categories of contemporary psychiatry are not transcendent, culture-free outcomes of objective observation and scientific research; instead, they carry forward the legacy of their own cultural histories (Gaines, 1992; Mezzich et al., 1999; Mezzich, Kleinman, Fabrega, & Parron, 1996; Young, 1995). To explore this further, we briefly consider the neo-Kraepelinian nosological project before turning to questions about the place of culture within mainstream scientific work on psychopathology.

The Neo-Kraepelinian Vision and the Nature of Contemporary Nosology

The historically contingent nature of the reigning nosology—and thus its salience as a "cultural artifact" (i.e., a creation or product that emerges within a unique time and place)—remains evident despite its increasing circulation and influence around the world. More specifically, modern versions of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM; American Psychiatric Association [APA], 1980, 1987, 1994, 2000) are the products of the neo-Kraepelinian movement that emerged in the 1960s at Washington University in St. Louis and that has dominated psychiatry since the 1970s (Wilson, 1993; Woodruff, Goodwin, & Guze, 1974; Young, 1991).

The Neo-Kraepelinian Vision

Klerman (1978) outlined several commitments of this neo-Kraepelinian movement, including the conviction that the study of psychopathology and its treatments belongs properly within the field of medicine; that mental illnesses/disorders are discrete entities with etiologies that can be discovered principally within the realm of disordered biology (as opposed to the previously dominant mode of explanation, derived from psychoanalysis, that privileged intrapsychic dynamics); that psychiatric research on psychopathology should depend principally on statistical inference in the context of modern scientific methodology; that the progress of science in the context of understanding psychopathology requires extensive concern with the standardization of diagnostic concepts and categories for implementation in research and treatment settings in reliable and valid ways; and that the relationships among and between discrete psychiatric disorders should be represented in scientifically valid classification schemes, with explicit diagnostic criteria for the disorders so classified (see Blashfield, 1984, for discussion and amplification of these commitments). The neo-Kraepelinian concern with standardized categories of disorder (consisting of detailed and relatively explicit criteria with accompanying decision rules for determining category membership) was central to the revolutionary reformulation of psychiatric nosology codified in DSM-III (APA, 1980) and its descendants.

There can be little doubt that cumulative scientific progress in understanding the origins, outcomes, and treatments of psychiatric distress requires some semblance of standardization, in order for independent research findings to coalesce and be built upon in useful ways. In addition, it seems sensible to specify and represent the relationships among and between various kinds of "mental disorders" within some heuristic classification scheme. The challenge is how to select among the almost infinite range of principles that could serve as organizational bases for structuring such a taxonomy (Millon, 1991). Given the fact that psychiatric scientists typically conduct their research within medical contexts, and thus tend to privilege the biological foundations of medi-

cal practice, psychiatric research has emphasized the empirical specification of "underlying pathophysiology" for various conditions and disorders as the sine qua non of the scientific project to advance our understanding of psychopathology. It is important to note, however, that many psychopathologists—especially researchers trained in clinical psychology and the remaining "psy-" disciplines—ardently contest the commitment of the neo-Kraepelinians to biological reductionism, arguing instead that psychopathology may result from emergent psychological or social processes that are not simply reducible to biology (Henningsen & Kirmayer, 2000; Kirmayer & Young, 1999; see also Beutler & Malik, 2002, for recent critiques). Nevertheless, the pursuit of underlying pathophysiology reflects the dominant trend in psychopathology research, as evidenced by the distribution of research funds, journal citations, and professional prestige.

Within the dominant frame of contemporary psychiatry, then, psychopathology is concerned with the fundamental biological processes (i.e., "basic" genetic, anatomical, and physiological processes in complex interactions that are "influenced" by the organism's environment) that go awry in instances of illness or disorder. Such pathophysiology is characterized as "underlying" because the precise means and mechanisms within the brain and body that are presumed to culminate in the reported symptoms or observable signs of psychiatric disorder are elusive and (in almost every instance) unknown. Thus the empirical pursuit of "endophenotypes" and other correlates of underlying pathophysiologies for a wide variety of mental disorders represents the dominant paradigm in the scientific investigation of psychopathology. Finally, within the traditions of scientific medicine, it is presumed that the empirical identification of etiology in the form of distinctive pathophysiology will ultimately *define* the disorders in question—in much the same fashion that modern medicine currently understands Down's syndrome, general paresis, or phenylketonuria in terms of their underlying pathophysiologies.

If the etiological pursuit of underlying pathophysiology characterizes the dominant paradigm in psychopathology research, then the obvious implication for developing a classificatory strategy is to organize the nosology in terms of kinds of pathophysiologies. The dilemma, of course, is that psychopathologists have yet to empirically identify *any* pathognomonic features of a purported mental disorder, much less its definitive pathophysiology. In the interim, the reigning taxonomic strategy depends on grouping taxa by similarities in "phenomenology"[1] (in most instances, based on clusters of symptoms at the syndromal level; see DSM-IV-TR [APA, 2000] for discussion). It is important to recognize that no credible psychopathologist—including the psychiatric scientists who developed the various revisions of DSM—would suggest that the disorders currently classified within DSM are validated constructs that warrant much scientific confidence. Nevertheless, the majority of psychopathologists are confident in the validation strategies described by Robins and Guze (1970) and elaborated by Kendell (1989) for empirically evaluating the merits of purported disorders as viable "hypothetical constructs" (Morey, 1991, drawing upon MacCorquodale & Meehl, 1948). These and other closely related strategies constitute normative science within a neo-Kraepelinian psychiatry.

In sum, although psychopathology researchers acknowledge that the hypothetical constructs contained in even the most recent version of DSM are "splendid fictions" (Millon, 1991, p. 246), most imagine that systematic inquiry within the paradigm just described will one day yield a much less arbitrary nosology that more closely approximates "carving nature at its joints." In response, we simply observe that the promise of the neo-Kraepelinian pursuit of distinctive pathophysiologies for the wide variety of mental disorders remains a matter of *professional faith.* The currently authorized nosological categories, consisting of nearly 300 of Millon's splendid fictions, reflect numerous political, aesthetic, and pragmatic commitments that yield abundant evidence of prescientific or nonscientific arbitrariness—all of which arise as expressions of cultural processes and practices (as indeed does scientific inquiry itself). To illustrate the impact of such processes and practices, we briefly consider the cultural history of one hypothetical construct within the domain of con-

temporary psychopathology—namely, posttraumatic stress disorder (PTSD).

The Cultural Construction of PTSD

The advent of DSM-III saw a movement to attribute a vast array of problems to trauma exposure, gathered together under the umbrella of PTSD. The ensuing years have seen the expansion of this category, which to some extent has absorbed other conditions formerly linked to adverse life events. Conventional histories of this construct suggest that PTSD has afflicted survivors of psychological trauma for millennia (Herman, 1992; Trimble, 1985), and that it has merely awaited discovery in the reports and behaviors of its sufferers by intrepid psychopathologists. In contrast to these seamless accounts, cultural historians have contended that the current conceptualization of PTSD, as both a category of clinical attention and a kind of crippling experience, is rather newly arrived on the historical stage (Hacking, 1996; Lerner, 2003; Leys, 1996, 2000; Young, 1995, 1996a, 1996b). These cultural analysts argue that PTSD, instead of possessing the timeless universality and intrinsic unity assumed in our "received" notions of the disorder, has only recently been "glued together" (Young, 1995, p. 5) from fragmentary shards of theory, politics, and practices spanning more than a century. Analytic attention to cultural processes and practices is therefore relevant not just for charting the varieties of PTSD experience among the world's diverse peoples, but also for grounding our conceptual understanding of the PTSD construct itself as one increasingly prominent category within the early-21st-century classification of psychopathology.

The construct of PTSD singles out the health consequences of the adaptive conditioned fear response to life-threatening danger. Although exposure to the threat of violence, pain, and injury readily gives rise to the specific forms of conditioned emotional response and avoidance learning held to underlie PTSD, this captures only a small part the human response to trauma and virtually never exists in isolation (Kirmayer, Lemelson, & Barad, 2007). The same traumatic events that give rise to PTSD have a wide range of other personal and social effects on biobehavioral systems involving fear, attachment, coherence, hope, identity, and sense of justice (Silove, 1999). The act of singling out a single biobehavioral response as a discrete disorder creates a measure of diagnostic clarity and precision—but, despite a considerable body of research, the extent to which this response should be framed as psychopathology and the degree of correspondence with the actual experience of suffering individuals both remain contested (Konner, 2007).

Even a casual inspection of recent versions of DSM indicates some evolution of psychiatric thought relative to PTSD since its initial incorporation into the official nosology of DSM-III in 1980. For example, the Criterion A definition of the qualifying stressor has changed between versions; the number of symptoms required for clinical inference of the disorder has increased; and the duration of symptoms necessary for diagnosis has ultimately been fixed at 1 month. More illuminating still are the questions and controversies that occupied the PTSD sub-Work Group of the DSM-IV Task Force during preparation of DSM-IV (Davidson et al., 1996). These included how narrowly to define the Criterion A stressor; what duration of symptoms to adopt for distinguishing between normative and pathological reactions to trauma; which subtypes and course specifiers to include; what number of avoidance symptoms to require for a diagnosis; whether to classify PTSD as an anxiety disorder or a dissociative disorder, or within a new class of trauma-related disorders; how to make sense of the high rates of comorbidity between PTSD and other mental disorders; and whether to include a new form of pathological posttraumatic response indicated by "extensive characterological changes" (p. 592) attributed to repeated and prolonged trauma. In some instances, the associated empirical investigation known as the PTSD Field Trial (Kilpatrick et al., 1998) obtained data related to these questions (e.g., neither broad nor narrow definitions of the qualifying stressor appeared to significantly alter sample prevalence of the disorder), but in most instances such data, even when obtained, were insufficient to resolve these questions (e.g., reduction of the required avoidance symptoms increased PTSD prevalence, and yet the overall implications of this

increase remained unclear in the face of delimiting sample characteristics).

For the purposes of our argument, the most important acknowledgment is that data alone will never be adequate to resolve these and many other similar questions and controversies surrounding PTSD (see McNally, 2003, for an elaboration), *as long as* independent and reliable measures of distinctive pathophysiology or specific etiology remain unestablished for the disorder. In fact, as the cultural historians suggest, it seems debatable whether meaningful human responses to "traumatic" experience are even of the "natural kind" variety that might be amenable to scientific demonstrations of distinctive pathophysiology or confirmations of specific etiology. As a result, for a hypothesized syndrome currently without *any* pathognomonic indicators that might unify diverse patient profiles, decisions regarding conceptualization of the disorder at this stage of inquiry are rendered largely by expert consensus (sometimes with recourse to data, but often not). Such consensus is made and unmade in *cultural* terms, through enduring and recognizable "logics" of expertise, argument, inquiry, and influence. Nowhere was this consummation of expert consensus more evident than in the historical events surrounding the initial inclusion of PTSD within DSM-III in 1980.

According to Scott (1990), PTSD was born of an unusual political alliance between psychiatrists such as Robert Lifton and Chaim Shatan and activists affiliated with Vietnam Veterans Against the War (VVAW) beginning in the late 1960s. This alliance's escalating campaign for medical recognition of "post-Vietnam syndrome" found footing in a 1975 meeting with psychiatrist Robert Spitzer, the architect of DSM-III, at the Anaheim convention of the American Psychiatric Association. As Scott recounts, Spitzer there dismissed the alliance's proposal for inclusion of the new syndrome, explaining that psychiatric researchers John Helzer and Lee Robins at Washington University had demonstrated with their data that the problems of returning Vietnam veterans were already subsumed under existing disorders (major depression, substance use disorder, etc.). Spitzer challenged the alliance to provide contradictory evidence. Later that year, following additional lobbying, Spitzer agreed to form a task force on the issue and invited alliance members Lifton, Shatan, and Jack Smith (a VVAW activist) to join himself and two other psychiatrists on the official Advisory Committee on Reactive Disorders. With Spitzer's attention frequently drawn elsewhere, according to Scott, the alliance members reasoned that he could be most effectively persuaded to include the syndrome if fellow committee member Nancy Andreasen, then a specialist in treating patients with burns, was first to be convinced of the merits of their cause. Despite ongoing opposition from the Washington University researchers, Andreasen and Spitzer eventually accepted that combat veterans were probably suffering from a distinct psychiatric illness. Given the limited empirical literature available at the time, this recognition depended principally on a series of compelling case studies, presented by outsiders to the psychiatric establishment, including the only member of any DSM-III advisory committee not to have obtained a graduate degree.

In sum, this alliance of "radical" psychiatrists and retired soldiers obtained official recognition of PTSD "because they were better organized, more politically active, and enjoyed more lucky breaks than their opposition" (Scott, 1990, p. 308). Of course, the implications of this watershed historical moment would be difficult to overemphasize. As Scott has observed, official recognition by the American psychiatric establishment accorded PTSD the status of "objective knowledge," which in turn undergirds what people experience as "objective" reality: "each new clinical diagnosis of PTSD, each new warrantable medical insurance claim, each new narrative about the disorder reaffirms its reality, its objectivity, its 'just thereness' " (p. 308). Such reaffirmations of objectivity are possible only if our perspective is fundamentally ahistorical and deeply inattentive to cultural processes and practices, the result of which is the *reification* of a provisional psychiatric construct for which pathophysiology and etiology remain unknown. And, in an ironic twist, such instances of unwarranted reification actively create culture by virtue of prescribing novel forms of illness experience, even as they disavow the relevance of culture for the nosological project.

Unwarranted reification represents a significant liability for any scientific endeavor, including the empirical opportunities and theoretical possibilities that remain unacknowledged and unexplored, owing to the premature foreclosure of conceptual alternatives. Such conceptual alternatives to contemporary PTSD are again suggested in the work of the cultural historians, who chart the rise of modern notions of "trauma" alongside late-19th-century investigations of hysteria, dissociation, and hypnosis. Ruth Leys (2000) asserts that our conceptualization of trauma and its pathologies continues to vacillate between two historical paradigms: mimetic theory, in which the symptoms of trauma are held to involve a kind of unconscious imitation of the original traumatic event, generally through dissociative mechanisms; and antimimetic theories, in which the posttraumatic symptoms are more or less direct consequences of the violent threat or assault. The dilemma is that mimesis can reflect pathological processes mediated by mechanisms of repression or dissociation in memory, or instead can be created factitiously by similar processes of recollection and recall (Young, 2007). Antimimetic theories circumvent this problem by positing psychophysiological effects of trauma (unmediated by the sufferer's own agency or unconscious dynamics) on subsequent symptoms. The so-called "memory wars" of the 1990s perhaps most clearly illustrated this paradigmatic tension (Crews, 1995). Both theories are represented within DSM-IV by the inclusion of PTSD (with a decided "antimimetic" deemphasis of attendant dissociative phenomena) and dissociative identity disorder (a mimetic pathology with a clear emphasis on purported traumatic etiology [Gleaves, 1996; Hacking, 1995b]); the former diagnosis has garnered current respectability within scientific psychiatry, while the latter has not.

In addition, Allan Young (1996a) observed that since their inception, the posttraumatic pathologies reported by survivors of 19th-century railway accidents were difficult for physicians to differentiate from neurological insult, neurotic disposition (leading to trauma-related "hysteria" and other "functional" disorders), or malingering (in pursuit of monetary damages). Young then raised the uncomfortable question of whether traumatic experience in fact caused the PTSD symptoms experienced by the combat veterans in his study, or whether these veterans only later attributed the cause of their long-standing symptoms to previous trauma in post hoc fashion as a direct response to treatment discourse. These concerns continue to trouble the field, insofar as psychopathologists have come to acknowledge the etiological importance of some preexisting "phenotypic expression of vulnerability" in PTSD (Yehuda & McFarlane, 1995) and to "worry" about recent evidence suggesting that a substantial proportion of Vietnam veterans—perhaps as many as 75%, as reviewed by McNally (2003)—have received disability payments for PTSD or have taken part in research studies as "cases" of PTSD, even though they may never have actually experienced combat (Frueh et al., 2005).

Even such brief attention to the cultural history of trauma and its pathologies serves to remind us that, far from the timeless universality and intrinsic unity frequently ascribed to the diagnostic entity, PTSD is a construct of rather recent invention. Certainly humans throughout history have responded to extremely distressing events with extreme distress. Such distress was undoubtedly evidenced through posttraumatic changes in individual cognition, emotion, and behavior. In our particular historical context, it would appear that Westerners (and increasingly the rest of the world as well, especially individuals making bids for international asylum to escape war, torture, and oppression) experience such distress in the increasingly popular genre of PTSD (Pole, Gone, & Kulkarni, 2008). The notion of PTSD serves these social and political functions, which in turn reinforce its coherence as a discrete entity, but this coherence is purchased at the expense of attention to a wide range of other individual responses to trauma. For every nightmare, flashback, amnesia, and exaggerated startle response currently assessed in traumatized patients, the avolition, weakness, headache, nausea, giddiness, photophobia, palpitations, paraesthesias, paralyses, double vision, altered posture, unsteady gait, feeble pulse, pressured speech, loss of appetite, and shortness of breath that characterized 19th-century pathological responses to psychological trauma (Kinzie & Goetz, 1996; Young, 1996a) have fallen by

the wayside. And yet many of these symptoms may continue to be prominent features of posttraumatic distress in diverse cultural settings (Kirmayer, 1996). In short, PTSD as currently configured is a malady of our time, emergent from and dependent upon the same cultural processes and practices that actively constitute contemporary life.

Implications for Cultural Analysis

This brief foray into the origins of PTSD as a nosological category and pathological construct raises questions about its claim to be a timeless, culturally universal entity. While scientific methods hold the prospect of refining our knowledge of how the world works, at any point in time scientifically derived knowledge remains an approximation that incorporates culturally and historically contingent features reflecting the origins of our constructs and the contexts of their use (Collins & Pinch, 1993). In the case of psychiatric nosology, we might consider that this cultural and historical embedding is not a defect or limitation of current scientific knowledge, but a necessity, since psychiatric distress, like all human experience, takes shape from cultural particulars. Psychiatric disorders reflect the outcome of interactions between biological processes and a social surround mediated by psychological mechanisms over the developmental trajectory of a human lifespan. The notion that a comprehensive or complete nosology can be created without regard to culture and context, therefore, can be sustained only by adopting a reductionist perspective that minimizes or ignores the fact that human beings are fundamentally social and cultural beings. Nevertheless, such reductionism is frequently embraced and promoted in the name of a scientific psychopathology, based on the assumption that modern psychiatry pursues transcendent understanding of disorders that exist in the world as natural kinds. In other words, the contemporary recognition of flashbacks and amnesia as symptoms of PTSD, as opposed to photophobia and double vision, is justified on the basis that recent systematic investigations have yielded historical progress in our approximation of the natural kind known as PTSD. But what are the conceptual grounds for presuming that PTSD or any other DSM disorders are natural kinds as opposed to "human kinds"—that is, intentional categories that emerge from our social institutions, knowledge, and practices?

Natural Kinds and Intentional Categories

In 1980, anthropologist and psychiatrist Arthur Kleinman (1986) conducted a landmark study in Hunan, China, that shed light on the universality of categories of common mental disorders. Kleinman studied a group of patients who had received the diagnosis of neurasthenia (*shenjing shuairuo*)—a syndrome marked by somatic complaints such as headache, fatigue, dizziness, and muscle tension, which was a common form of distress routinely diagnosed by Chinese psychiatrists in clinical settings. Neurasthenia (or "nervous weakness") was originally described by the American neurologist George Beard in the late 1800s and soon became a common diagnosis worldwide (Beard, 1869). After the 1920s, the popularity of neurasthenia waned in the West as it was gradually replaced by other construals of psychopathology, most recently clinical depression. Nevertheless, neurasthenia persisted as a professional diagnostic label and a mode of illness experience throughout China up to the 1990s (Lee, 1998).

Applying the diagnostic criteria from the DSM to 100 Chinese patients diagnosed with neurasthenia, Kleinman (1986) determined that the vast majority of these individuals met criteria for major depressive disorder. When they were treated with tricyclic antidepressants, most patients showed some improvement in their symptoms of depression; however, many continued to see themselves as suffering from neurasthenia, pointing to symptoms of depleted energy and other somatic symptoms or difficulties in their lives, which they attributed to their catastrophic experiences during the Cultural Revolution. Instead of concluding that Chinese neurasthenia was identical to clinical depression, Kleinman argued that neurasthenia and depression were in fact distinct forms of distress that did not always co-occur among Chinese patients. Subsequent work has borne this out (Zheng et al., 1997). Nevertheless, drawing on Eisenberg's (1977) differentia-

tion between subjective illness experience and objective disease process, Kleinman assumed that both neurasthenic and depressive syndromes were superficially divergent expressions of the same underlying disease: a "universal core depressive disorder" (1986, p. 66). In short, for Kleinman, neurasthenia was a somatized form of an "underlying" depressive disease. As a folk and professional category, and as a cultural "idiom of distress," neurasthenia had its own sociomoral uses and implications. Kleinman emphasized this sociomoral dimension of experience (though he diplomatically downplayed the continuing role of political repression), but was largely uninterested in neurasthenia as a psychopathological construct for which one might seek to understand underlying mechanisms.

In a critique of Kleinman's report, psychological anthropologist Richard Shweder (1988) noted an unresolved tension in the study's conclusions between a positivist and a constructivist perspective on the diagnostic problem at hand. According to Shweder, neopositivists remain interested in discovering "natural kinds," those phenomena that "exhibit a causation independent of what they mean to us, independent of our involvement with them, independent of our experience with them or evaluation of them, independent of our aesthetic or emotional response to them" (p. 488). In contrast, he continued, constructivists remain interested in discovering "intentional categories," those phenomena that "exhibit whatever causation they may have by virtue of what they mean to us, by virtue of our conceptions and representations of them and reactions to them" (p. 488). Natural kinds thus include such phenomena as trisomy 21 and dopamine. Intentional categories may include such phenomena as psychopathic deviance or *la belle indifférence*.[2]

While Kleinman explicitly adopted a constructivist perspective for many of his analyses of Chinese neurasthenia, Shweder worried about Kleinman's characterization of this syndrome as somatized depression: Could not depression just as easily be construed as a psychologized form of neurasthenia? Shweder wondered what rationale might be offered in support of Kleinman's clear preference for a depression-centered discourse: "If a disease process is different from an illness experience and if depression is a disease process (as well as an illness experience), then what precisely is that depressive disease process that is other than an illness experience, and how do we know that neurasthenia is a somatized version of it?" (1988, p. 494). Here Shweder laid bare the fundamental problem of cross-cultural analysis in psychopathology research—namely, the challenge of determining how we might reconcile divergent frames of reference, modes of representation, and modalities of experience that give rise to diverse patterns of dysfunctional or disordered experience and expression within and between culturally distinctive communities throughout the world. One implication of the distinction between natural kinds and intentional categories is that while attention to cultural processes and practices throughout the diverse regions of the world may be helpful and illuminating for investigation of disorders of the "natural kinds" variety, such attention is absolutely indispensable for investigation of disorders of the "intentional categories" variety. In other words, if human pathological reactions to traumatic experiences are indeed widely contingent on time, locale, and ethos, then conceptualizing, classifying, investigating, and treating such reactions are heavily dependent on the historically and culturally contingent frameworks of meaning that mediate such pathological experiences. But what are the grounds for imagining that such cultural frameworks of meaning might actually mediate the experience of many forms of psychopathology?

The Depth and Sweep of Culture: The Case of Emotional Experience

A robust cross-cultural psychopathology takes as its point of departure the recognition of the *co-constitution* of mind and culture. More specifically, cultural psychologists and psychiatrists are concerned with the manner in which human beings—and the cultures they dynamically and interactively construct and reproduce—give rise to "culturally constituted persons" who are both producers and products of the intentional worlds they inhabit (Shweder, 1991). For our purposes, "culture" may be understood as the socially patterned and historically reproduced systems of semiotic practices that both facili-

tate and constrain human meaning making (Geertz, 1973; Gone, Miller, & Rappaport, 1999). Culture is *social* (and often public) because such systems must be shared; there is no culture of one. Culture is *patterned* because such systems are organized and utilized systematically in order to be intelligible to others; they are not randomly recreated with each usage. Culture is *historically reproduced*, in that successive generations are socialized into using the intelligible systems of their communities (which is not to argue that culture is simply "transmitted" from one generation to the next, as innovations and modifications are constantly introduced both in the process of socialization and as subsequent generations adapt to novel circumstances). Finally, cultural practices are *symbolic*, in that they allow for the ascription and communication of meaning or "intelligibility" to others.

In other words, culture comprises shared patterns of activity, interaction, and interpretation. Perhaps the most salient example of culture is language, which serves as the primary semiotic system available to human beings for achieving mutual intelligibility, as well as the principal medium of intergenerational cultural reproduction. The study of mind and mentality within enculturated human communities makes it clear that cultural meanings and practices are just as central to realizing personhood as biological mechanisms or processes are. That is, human experience is crafted, constituted, or constructed from the complex and divergent ways in which culture and biology come together to render such experience possible. Thus, obviously, there is no culture without human biology—but, similarly, biology in the absence of culture is neither recognizable nor sustainable as human experience (Kirmayer, 2006; Wexler, 2006). Our point here is simply that, contrary to the evident commitments of the neo-Kraepelinians and the disciplinary traditions of psychiatry and psychology (which routinely refer to culture as rather superficially "shaping" or "influencing" putatively more basic biological processes),[3] there is no compelling reason to routinely privilege biology as more fundamental than culture to many of the constructs of interest within psychopathology.

One example of the reductionist bias toward psychological processes that are in fact co-constituted by both biology and culture occurs routinely in the psychological study of emotion, one of the most basic constituents of psychopathological experience. Most forms of psychopathology are accompanied by troubling emotions, and specific kinds of emotional experience provide the phenomenological basis for two of the most prevalent DSM diagnostic classes (at least as surveyed in the affluent West): the "mood" and "anxiety" disorders. As a result, the psychology of emotion figures prominently in the study of psychopathology.

The Dominant Approach to Emotion Research

Oatley and Jenkins (1992) traced the conceptual paradigms that have guided emotion research in the discipline back to Darwin (1872/1965) and James (1890). Whereas Darwin emphasized the biological and evolutionary significance of emotional processes and James emphasized the phenomenology of emotional experience, both writers conceptualized emotions as *primarily* intrinsic biological or physiological properties of the organism. The Darwinian tradition in particular inspired research by Ekman (1984) into the cross-cultural prevalence of emotion. Drawing on the presumed evolutionary significance of facial expression in the communication of internal emotional states to other members of one's species, Ekman discovered that respondents from many of the world's cultures expressed consistent associations of certain facial expressions with comparable emotion terminology, suggesting the universality of at least six basic or core emotions. For Ekman, the cultural and linguistic diversity encountered in these investigations was less interesting than the search for affective universals.

Cognitive investigations of emotional experience by psychologists have also tended to assume a universal biological core to emotion (Oatley & Jenkins, 1992). Building on the early idea of James that an emotion was the "feeling of the reaction to an event" (Oatley & Jenkins, 1992, p. 58), the neo-Jamesian tradition declared that "emotion was perception of a generalized arousal plus an attributional label" (p. 58). Although this idea represents a step beyond the view of emotion as fundamentally a biological pro-

cess, it suggests that a cognitive attributional label has been overlaid on the physiological core of emotion. Focusing on the cognitive mechanisms involved in emotional experience, contemporary psychology tends to emphasize the specificity and function of emotions, including their effects on attention and memory as well as their communicative roles in social interactions. These investigations have acknowledged that "the conditions that elicit an emotion distinguish it from other emotions" (Oatley & Jenkins, 1992, p. 60), and a growing body of work has examined the social determinants and consequences of emotion. Although these relatively recent developments in psychology seem to be conceptual moves in the right direction, there remains a conceptual bias toward viewing emotions as a set of biophysical and intrapsychic states. In this view, emotions are natural kinds, and culture is relegated to the role of configuring the situations that elicit emotions and shaping their outward expression.

But is culture really so peripheral to the psychology of emotional experience? In the past two decades, philosophers, cultural historians, cross-cultural psychologists, and anthropologists have trained critical attention on the dominant conceptualization of emotional experience and expression. Grounded in constructivist approaches to the study of self, personhood, and social relations, these scholars have proposed a reconceptualization of affect that transcends the Western notion of emotions as primary physiological processes with secondary cognitive, social, or cultural overlays. The result has been a new paradigm for emotion research that acknowledges biology, but that also gives serious attention to the cultural construction of experience (Abu-Lughod & Lutz, 1990; Griffiths, 1997; Gross, 2006; Harré, 1986; Kitayama & Markus, 1994; Leavitt, 1996; Lutz & White, 1986; Reddy, 2001; Rosaldo, 1984; Shweder, 1993; White, 1993).

The Constructivist Alternative for Emotion Research

The primary challenge facing the new paradigm for emotion research is overcoming the Cartesian dualism evident in most Western academic traditions (Leavitt, 1996) that gives rise to a familiar series of conceptual dichotomies (e.g., natural vs. cultural) that shape Western discourse. Several such dichotomies are evident in both scientific and Western folk discourse about emotions: mind versus body, cognition versus affect, thinking versus feeling, rational versus emotional, conscious versus unconscious, intentional versus unintentional, controlled versus uncontrolled, and so forth (Kirmayer, 1988; White, 1993). These conceptual oppositions are deeply ingrained in Western thinking and have resulted in "two-layer" theories (Lutz & White, 1986) or "dual-process" models (White, 1993) of emotion that conceptualize affect as "psychobiological processes that respond to cross-cultural environmental differences but retain a robust essence untouched by the social or cultural" (Abu-Lughod & Lutz, 1990, p. 2). Thus, with regard to the study of emotions, "any phenomenon acknowledged to be culturally variable (e.g., the language available for talking about emotion) is treated as epiphenomenal to the essence of emotion" (Lutz & White, 1986, p. 408).

Instead of replicating such dualisms, cross-cultural researchers with serious commitments to examining the individual as an embodied agent in a sociocultural context must transcend such thinking. Leavitt (1996) described an appropriate outcome with regard to the study of affect:

> We would have to see emotions as primarily neither [cultural] meanings nor [psychobiological] feelings, but as experiences learned and expressed in the body in social interactions though the mediation of systems of signs, verbal and nonverbal. We would have to see them as fundamentally social rather than simply as individual in nature; as generally expressed, rather than as generally ineffable; and as both cultural and situational. But we would equally recognize in theory what we all assume in our everyday lives: that emotions are *felt* in bodily experience, not just known or thought or appraised. (p. 526)

Although Leavitt was perhaps a bit too dismissive of the private, inchoate, and sometimes inexpressible qualities of emotions, his larger point is clear: The dominant characterization of emotions as fundamentally individual, interior, biological events must be counterbalanced with attention to their cultural, social, and expressive dimensions.

What concretely, then, does all of this imply for the study of emotional experience—and, by extension, to the study of psychopathology as well? First, as a research construct, emotions must be understood to include biological, psychological, linguistic, social, and cultural processes that are unified in the embodied person engaged in situated and meaningful action. Second, claims regarding the uniformity of emotional experience across cultures (at least in any nuanced sense) seem implausible. An affective experience that is substantively constituted by its semiotic context cannot possibly be universal (i.e., mean the same thing) across all cultural communities of the world (see Wierzbicka, 1999, for numerous examples). Finally, the meanings of emotional experience, as facilitated and constrained by linguistic practices in particular, are situated within wider conceptual webs of cultural meaning regarding personhood, social relations, spirituality, the moral order, and so on (Harré, 1986; Lutz & White, 1986; Shweder, 1993; White, 1993). Of particular interest here is the manner in which such local webs of meaning inform and construct emotional experience for the person. Thus a systematic exploration of local ethnopsychology (i.e., theories of mind, self, and personhood) must be central to studies of emotional experience and psychopathology across cultures.

An illustration of these issues is found in the work of anthropologist Theresa O'Nell (1996) on depression among the Salish Indians of the Flathead reservation in northwestern Montana. Similar to Kleinman (1986) in his investigations of Chinese neurasthenia, O'Nell discovered that depression on the Flathead Indian reservation was explicitly associated with community experiences of colonial conquest and historical oppression, as exacerbated by ongoing contention with European American racism. Most importantly, O'Nell determined that depressive-like experiences among the Salish were explicitly cast in relational terms (e.g., these were characterized by feelings of interpersonal loneliness rather than intrapsychic sadness). The relational orientation of this sociocentric society thus gave rise to three persistent states of being that shared symptoms of DSM major depression: feeling bereaved, feeling aggrieved, and feeling worthless. Of these, only the third condition was at all likely to lead to suicide, while the first was in fact esteemed as a mark of maturity among elderly Salish tribal members, who were seen to grieve appropriately for the many losses experienced by members of Flathead society over the previous century and more. The lesson here is that forms of psychopathology that are characterized by distressing or disordered emotional experience may be configured quite differently for individuals from societies that construe the person in more egocentric or individualistic terms and from those that are more sociocentric (Kirmayer, 2007b).

Implications for Cross-Cultural Psychopathology

Cross-cultural work on emotions has shown that most complex feelings are tied to specific developmental experiences and social scenarios, which depend in turn on social structure and cultural knowledge and practice. If culture thus has the depth and sweep to actively co-constitute the varieties of emotional experience around the world, then human emotions are best understood not as "natural kinds" but instead as "human kinds," born of an interaction between biological processes and cognitive and social construals (Griffiths, 2004; Hacking, 1995a, 1999; Hinton, 1999). This interactional, biosocial view points to a way to integrate our understanding of the embodied substrate of emotion with the complex social and cultural practices that give meaning and import to emotional experience as they unfold through development. Neo-Kraepelinian psychiatry—with its commitment to biological reductionism and the accompanying presumption that "real" psychiatric disorders are natural kinds—cannot do justice to this complex interaction.

DSM and the Problem of Cultural Imperialism

As we have already observed, the publication of DSM-III (APA, 1980) was a landmark historical, scientific, and political achievement, signaling the advent of neo-Kraepelinian psychiatry in the United States. Owing to standardized criterion sets with explicit application algorithms, modern versions of

DSM afford reliable psychiatric diagnosis, and thereby permit a cumulative science of psychopathology. Of course, construct validity for the hundreds of postulated disorders within DSM remains elusive; instead, psychopathologists employ DSM under the optimistic assumption that over time, accumulating evidence from research studies using standardized diagnostic criteria will enable them to "bootstrap" their way to diagnostic validity.

Nevertheless, DSM has come to dominate the ways in which mental health professionals in the United States and in many other countries classify and diagnose psychiatric illness—and, as a consequence, to suffuse the ways in which patients (and the broader public) make sense of their distress and dysfunction. That is, in everyday clinical practice, the *hypothetical* constructs classified within DSM take on a privileged ontological status in the lives of patients, professionals, and institutions through routine processes of reification. Indeed, it is through clinical praxis that the scientific and clinical conjectures codified in DSM become accepted as authorized knowledge and authoritative discourse. In actuality, then, DSM simultaneously serves two different purposes that are potentially at odds with one another: namely, as a provisional *scientific taxonomy* for facilitating empirical research on the one hand, and as an institutionalized *professional manual* for guiding clinical practice on the other (Gone, 2003b). The tensions between these functions (and epistemic stances) are greatly exacerbated in cross-cultural applications of DSM, especially those in which enduring asymmetries in cultural capital and political power lend themselves to the unwarranted hegemony of Western psychiatric discourse.

Psychiatric Services and Western Cultural Proselytization

Contemporary views of culture recognize that most individuals have access to multiple cultural systems, and that the "culture" of specific communities is actually made up of many competing and contesting streams or positions. Acknowledging the importance of cultural difference is not simply a matter of taking account of variations in developmental experiences, social contexts, and commitments. Cultures are unequally accorded or invested with power and authority. The power attached to specific cultural systems and communities arises from a specific history of domination and control that may continue to exert effects on ways of thinking long after the machinery of domination has been challenged or dismantled.

In psychiatric research concerned with the mental health status of historically oppressed ethnic/racial minority communities in the United States, for example, psychopathologists must recognize that the "culture" of the clinic is not the "culture" of the community. More specifically, the assumptions, assertions, aspirations, and attributions that mental health professionals routinely rely on are grounded in the categories and conventions of Western therapeutic discourse, including those contained within DSM. Such discourse has emerged historically from northern European and European American sensibilities regarding normative and disordered psychological, emotional, and behavioral functioning (Gaines, 1992). As a result, the therapeutic discourse that anchors mainstream clinical activity undertaken in many non-Western cultural contexts may diverge in substantial ways from local assumptions and expectations of wellness, health, and "the good life" (as we have already seen in the context of emotional experience and expression). Moreover, for much of the history of psychiatry, the profession has worked in cooperation or collusion with the powers of colonial domination (Bhugra & Littlewood, 2001; Jackson, 2005; Keller, 2007; McCulloch, 1995; Sadowsky, 1999). More specifically, the privileging of Western theories of psychopathology and therapeutic discourse has been associated with longstanding efforts by European Americans to express or achieve cultural dominance over other peoples through processes of colonization, and to maintain dominance through racialized hierarchies of power and authority.

This historical bid for European American cultural dominance frequently involved the explicit devaluation, disruption, and displacement of these alternate frames of reference, modes of representation, and modalities of experience. A small group of psychiatric thinkers and practitioners has challenged this collusion, rejecting the rac-

ist ideologies that rationalized colonial violence, and supporting the political struggles that have sought to transform or overthrow colonial regimes (Fanon, 1982). The result is an important literature that has examined the impact of colonial systems of racism and oppression on the identities, personalities, and psychological well-being of colonized subjects, as well as the possibilities for liberatory psychiatric practice (Vergès, 1996). This literature would benefit from contemporary reconsideration in light of the changing forms of structural violence (Gilroy, 2004). Nevertheless, despite these occasional (and politically marginalized) efforts, both cultural divergences and asymmetries in power render the provision of conventional psychiatric services to historically oppressed communities a politically suspect activity that may advance Western cultural proselytization in the guise of therapeutic knowledge and activity.[4]

A Postcolonial Discourse of Distress

We can illustrate the kinds of ideological dangers we have in mind with reference to two of the most prevalent forms of DSM psychopathology: alcohol dependence and major depression. In an ethnographic investigation on a northern Plains Indian reservation, Gone (2007, 2008c) identified a prototypical "discourse of distress" concerning problematic drinking and depression in contemporary Native American tribal life. According to one especially instructive respondent (pseudonymously named "Traveling Thunder"), these problems could be traced to disrupted ceremonial tradition in the context of historical dominance by European Americans. More specifically, Traveling Thunder identified four historical epochs in his characterization of the causes of pathological depression and drinking on the reservation. The first epoch was the era of "Paradise," a precolonial existence in which such pathologies were largely unknown, owing to the perfect harmony and balance wrought by community adherence to the strict observation of social custom and sacred ritual. The second was the era of "Conquest," or the colonial encounter in which the genocidal and assimilative activities of European Americans led to the annihilation of custom and ritual. The third epoch was the era of "Loss," in which the postcolonial effects of the annihilation of custom and ritual led to anomie, and in turn to substance abuse, depression, and sometimes suicide. Finally, the current epoch is the era of "Revitalization," in which the Creator has "pitied" the people enough to facilitate a communal reclamation of indigenous custom and ritual.

In one particularly illuminating moment during the interview, Gone (2007) asked Traveling Thunder to reflect on the conditions under which he would refer a distressed loved one to the mental health professionals at the local reservation clinic. His reply lacked any trace of ambivalence:

> That's kind of like taboo. You know, we don't do that. We never did do that. . . . If you look at the big picture, you look at your past, your history, where you come from . . . and you look at your future where the Whiteman's leading you, I guess you could make a choice. Where do I want to end up? And I guess a lot of people . . . want to end up looking good to the Whiteman. . . . Then it'd be a good thing to do: go [to the] white psychiatrists . . . in the [reservation clinic] and say, . . . "Go ahead and rid me of my history, my past, and brainwash me forever so I can be like a Whiteman." (p. 294)

Thus, for Traveling Thunder, the activity of "white psychiatrists" on the reservation was explicitly marked as an extension of the colonizing project, in which indigenous selfhood remains a site of neocolonial engagement and resistance. As an alternative, Traveling Thunder proposed the reclamation of indigenous selfhood through the reestablishment of ritual practice. Such practice serves to link the human self to other-than-human Persons[5] in the respectful offering of gifts and prayers in exchange for the compassionate outpouring of prosperity and blessings. In the process, alienation and anomie are simultaneously (but secondarily) resolved through the establishment of a robust cultural identity (Gone, 2006a, 2008b, in press-a).

A central feature of Traveling Thunder's discourse of distress was its reliance on observations, inferences, and insights drawn from the sociohistorical and spiritual levels of experience and analysis. From this perspective, mental health problems—including the anomie, demoralization, depression, substance abuse, and suicide found on the reservation—were understood as direct

consequences of the European American colonial encounter that disrupted ritual relationships and community responsibilities to powerful other-than-human Persons. It follows that the most effective remedy for pathological drinking and depression within the community would be a restoration and return to individual and collective ceremonial practice (Gone, 2007). In sum, this contemporary ethnopsychological discourse configures wellness (i.e., life lived "in a good way") quite differently from the "mental health" of psychiatry and the associated professions, and posits quite different etiologies for serious distress (Gone, in press-c). For Traveling Thunder, pathological drinking and depression were functions of culture, history, and identity, contrasting sharply with the reigning psychiatric emphasis on genetic predispositions, chemical imbalances in the brain, and other biologically reductionist explanations as fundamental to these disorders.

As we have already noted, the concepts, categories, principles and practices of neo-Kraepelinian psychiatry—including the codifications of DSM—remain cultural artifacts, the meanings and mechanisms of which emerge from and depend on their cultural intelligibility within a shared discursive frame. As a result, casually embracing DSM in one's cross-cultural professional activity risks irrelevance at best, or an often subtle (but sometimes overt) Western cultural proselytization in the guise of therapeutic progress at worst. Certainly the ideological hazards of this nearly invisible "West is best" cultural imperialism in postcolonial societies and contexts such as Traveling Thunder's reservation homeland remain worrisome and require serious consideration and redress.

Decolonizing Psychiatry

"Postcolonial" is a term that has been used to characterize the struggles for liberation undertaken by formerly colonized peoples as they assert their social, political, and cultural autonomy. Such struggles, however, have not eliminated structures of domination established during colonial eras or prevented the emergence of new strategies of exploitation rooted in national or ethnic interests. These structures and strategies have the effect of maintaining inequalities, with profound consequences for the quality of life of formerly colonized peoples in postcolonial societies. In addition, recent processes of globalization have facilitated shifts in strategies of domination toward systems of power structured by consumer capitalism and the interests of multinational corporations and their associated economic institutions. For this reason, the prefix "post-" in "postcolonial" probably warrants scare quotes to denote the fact that many oppressive features of colonization have not ended, but instead have mutated or gone underground, only to reemerge in powerful new forms. Indeed, the increasingly global influence of Western psychiatry—accompanied by its material and discursive power to undermine or displace local notions of self, personhood, identity, emotion, social relations, spirituality, distress, wellness, and healing around the world—would seem to require a great deal more ethical attention to the role of psychiatric services as vehicles to export specific cultural values, particularly those of secularism and especially individualism.

By virtue of their creation, utilization, and dissemination by psychiatrists, the psychopathological constructs classified within DSM are generally cast in terms that locate the "disorder" within an individual. This reflects a "causal attributional bias" that may result in blaming the person for his or her affliction. In a now-classic article, Caplan and Nelson (1973) criticized "the tendency to hold individuals responsible for their own problems" (p. 199)—first, by focusing on "person-centered" characteristics while downplaying or ignoring situationally relevant factors; and, second, by attributing causal significance to any person-centered variables found to be statistically associated with the social problem in question. Caplan and Nelson reviewed a sample of published articles indexed in *Psychological Abstracts* to demonstrate that in research with African Americans, psychologists invested "disproportionate amounts of time, funds, and energy in studies that lend themselves, directly or by implication, to interpreting the difficulties of black Americans in terms of personal shortcomings" (p. 204), rather than in terms of situational factors or systemic inequalities. They identified several social and political functions served by such construals of social problems, and concluded that "person-blame interpretations are in everyone's

interests except those subjected to analysis" (p. 210).

In light of these observations, let us return to the alternative presented by Traveling Thunder, who observed that the epidemic of distress in his reservation community appeared to have emerged hand in hand with the ravages of colonization. Traveling Thunder's account emphasized situational factors and systemic inequalities rather than "person-centered" biogenetic or intrapsychic factors. Accordingly, Traveling Thunder asserted that the community rather than the individual ought to be the focus of therapeutic attention and intervention, and that the problems faced by individuals and the community might best be characterized as an existential and spiritual crisis. Like the expressions of depression among the Salish recounted by O'Nell, and like the current appropriations of the term "historical trauma" among many other indigenous peoples and communities (Brave Heart & DeBruyn, 1998; Gone, 2008b, in press-a), Traveling Thunder's discourse embeds psychopathological experience in the larger meanings of *collective* experiences of longstanding European American subjugation. This focus on social and historical context as a way of characterizing individual suffering is in marked contrast to the dominant ideological commitments of neo-Kraepelinian psychiatry, in which mental disorders are presumed to be natural kinds that afflict individuals through presently unknown pathophysiological processes. To the extent that they employ this decontextualized view of psychiatric disorders, mental health services in the reservation context cannot help engaging in the sort of "person blaming" decried by Caplan and Nelson.

The basic remedy for this unfortunate state of affairs is to resituate individual and social suffering in its cultural and historical contexts. This has a political dimension, insisting on the importance of the interpretive frames and perspectives of the culturally diverse subjects of psychiatry theory. But the development of situated theory in psychopathology is not simply a matter of "political correctness." It requires a vibrant program of cross-cultural research on various forms of psychopathology—a program that seriously engages "emic" (local or emergent) frames of reference, in addition to the "etic" (external and imposed) models of psychology and psychiatry. Such research would not ignore conventional approaches to the investigation of psychopathology, but would recognize that the relationship between local and external models and frames of reference requires systematic study through open-ended empirical work that does not assume that either framework will provide all the answers. In some instances, the compelling validity of local understandings may directly challenge the constructs of DSM, demonstrating their inapplicability or irrelevance to local forms of suffering (i.e., Kleinman's [1988] "category fallacy"). In other cases, emic constructs may lead to models of wider applicability and so themselves become etic constructs. This systematic empirical project is based on the conviction that many forms of psychopathology (including some of the most popular and prevalent diagnoses) are "human kinds" best approached through careful investigation of the local, lived meanings of experience, rather than "natural kinds" that can be adequately characterized in terms of universal biological mechanisms and corresponding categories of experience.

Culture, Context, and Experience in Psychiatric Science and Clinical Practice

In line with the reflexive stance central to contemporary social studies of science, we have so far approached the importance of culture for psychiatric nosology through its impact on the nosological enterprise itself. This framework targets not the ethnocultural characteristics of patients per se, but instead addresses the cultural embedding of diagnostic theory and practice, especially as it pertains to everyday clinical concerns. The model discussed here is quite general and, in the context of preceding observations and insights, argues for professional recognition of the pervasive effects of culture and context on every aspect of the psychiatric enterprise.

The Uses of Psychiatric Nosology and the Impact of Diagnosis

Psychiatry covers a broad domain of human problems. Mental illness is not one thing,

but a congeries of heterogeneous problems—including forms of brain dysfunction, psychopathological processes that result from various forms of learning, problems that reside in interpersonal interaction, and problems that consist of incoordination or contradiction among these different levels of organization (Kirmayer & Young, 1999). These problems are related to one another by family resemblances, so that there is no common essence or single characteristic shared by every psychiatric disorder, except at a very high level of abstraction. Although, as we have argued, biological, psychological, and social factors contribute to all of these problems, the relative importance of causal and aggravating factors varies for each type of problem as well as for each individual, episode, and situation. As a result, no one solution to the structure and function of psychiatric nosology will work. In particular, neither genetics research nor neuroimaging will tell us what to include in a nosology unless we decide to redefine the domain of psychiatry narrowly in terms of these technologies (Robert, 2007; Robert & Plantikow, 2005).

The construction of a nosology and related diagnostic instruments and techniques reflects specific goals or purposes. Earlier, we have discussed the tensions between the use of psychiatric nosology as a provisional scientific classification of psychopathology and as a manual for professional practice. Diagnostic systems have additional uses in other domains, including the determination of health care policy and the regulation of other social institutions. The scope and content of a diagnostic system may have profound effects on the design and function of health care systems, including resource allocation and access to care. In the wider social context, diagnoses serve to position individuals by assigning them the sick role, and thus identifying the persons as legitimately distressed or disabled and deserving of help, compensation, or support. Diagnosis also has implicit functions. For the clinician, assigning a diagnostic label serves to name and contain the confusion and threat presented by the suffering patient (Kirmayer, 1994). For patients, a diagnostic label and its connotations are used to draw out the implications of an illness and, when the condition is chronic, to (re)construct aspects of personal identity. These implicit meanings of diagnosis may also have powerful social implications, conferring stigma or prompting other practices of exclusion.

A psychiatric nosology, then, is not simply a systematic ordering of categories found in nature, but constitutes a map and charter of a social world. Nosology provides a map, in that it marks off specific domains and establishes borders and boundaries whose crossing makes a difference to individuals' social status. Nosology also functions as a social charter because this act of mapping creates an "official" reality and authorizes the architects and users of the diagnostic system to exercise specific forms of social power. In the context of the clinic, diagnosis is part of constructing a problem list, identifying the issues that require some form of help or clinical attention. Clinical problem lists commonly go well beyond the specific entities of diseases or disorders to include social problems, interpersonal conflicts, and existential dilemmas—all of which figure in patients' suffering, and which may influence the appropriate intervention for specific disorders or may be primary foci of concern in their own right.

Clinical Epistemology and the Place of Culture in Psychopathology

Scientific research and clinical assessment involve different epistemological assumptions. Clinical knowledge is constrained by the temporal frame of the clinical encounter and its specific goals for problem identification and solution. The focus is on signs and symptoms, and on what can be identified through history taking, systematic interviewing and observation, physical examination, and laboratory tests. The aim is to use this information to infer the underlying disorder that accounts for a patient's distress and that can then be targeted for intervention. The implicit theory of medical semiotics views symptoms simply as more or less veridical reports of bodily events or physiological perturbations (Kirmayer, 1994). Although the mapping from pathophysiology to symptom may be many-to-one (nonspecific symptoms may result from many different forms of pathology) or one-to-many (a single pathology may have variable clinical manifestations), in practice medical semiotics commonly as-

sumes a one-to-one mapping or isomorphism from physiological disturbances to bodily experience and from bodily experience to symptom report. Hence symptoms are taken as indicators of underlying pathophysiological processes. Given the lack of independent biomarkers for psychiatric disorders, an assumption is also made that clusters of symptoms (syndromes) are sufficient to identify distinct forms of pathology. Furthermore, it is tacitly assumed that the diagnostic nosology identifies all the clinically significant forms of pathology that can occur. The accuracy and completeness of the nosological map are therefore matters of great importance for science, clinical care, and policy. Problems that fall outside the nosology are not accorded the same level of interest, status, or priority by researchers, clinicians, and policymakers. This makes the nosology an important regulator of psychiatric science and practice.

This is an especially important issue in cross-cultural work because of the epistemological problem identified by Kleinman (1988) as the "category fallacy." Efforts to apply a set of diagnostic categories developed in one cultural context in a different setting may obscure important cultural differences. Although it may be possible to identify people who fit the diagnostic criteria, this does not ensure the local validity of the category; nor does it rule out the possibility that individuals with related forms of suffering are not captured by the diagnostic criteria. Local categories of illness may yield better indicators of distress, and better predictors of prognosis and treatment outcome. Testing this possibility requires specific research methods (Canino, Lewis-Fernandez, & Bravo, 1997).

An additional epistemological problem arises from what Hacking has called "the looping effect of human kinds" (Brinkmann, 2005; Hacking, 1995a, 1999)—an elaboration on our prior consideration of "natural kinds" and "intentional categories." Hacking recognized intentional categories (or human kinds) as those that depend on specific ways of construing experience. In such instances, the very act of diagnosing a given pathology in an individual harbors the potential to alter that individual's experience of the pathology, as well as the subsequent scientific and professional construals of that individual's behavior. These ways of construing behavior circulate in the larger society, becoming social and cultural norms, models, and practices that alter other individuals' interpretations of their own experiences. Hence changes in cultural assumptions or cognitive models will lead to new conceptual categories that are reified and stabilized by recursive social processes of dissemination and enactment. The changing forms of "trauma" reviewed earlier, as well as the evolution of "hysteria," provide clear examples of this phenomenon (Hacking, 1995b, 1998).

Although we have previously employed Shweder's (1988) distinction between natural kinds and intentional categories (or human kinds), many psychiatric categories are best thought of as what Hacking terms "interactive kinds," in which there is a transaction between natural distinctions and culturally constructed concepts. Cognitive theory would suggest that panic disorder and major depression are two examples of a specific version of interaction that Hacking calls "biolooping," in which (culturally mediated) modes of construing experiences of the body and the self lead to physiological disturbances (Hinton & Hinton, 2002; Hinton, Hinton, Pham, Chau, & Tran, 2003). To the extent that these disturbances follow a biologically dictated final common pathway, the disorders may be viewed as ultimately independent of our construals, and hence as natural kinds (Cooper, 2004). For example, if there were a core syndrome of neurasthenia or depression involving a state of physiological depletion, we might fix on this aspect to define a category of pathology independently of how an individual arrives at that state (Kirmayer & Jarvis, 2005). However, there may be forms of psychopathology in which the culturally and cognitively mediated modes of construal are essential to defining the problem. In the case of "intentional" behavior, which is distinguished by the fact that the person can give reasons for their action (and that the reasons are causally implicated in the action), there is a loop that depends on the distinctively human capacity for self-awareness. For this type of problem, there is no way to define the pathology without characterizing the nature of the disturbances in self-awareness, self-representation, and self-control, which in turn depend on particular cultural con-

cepts of self and personhood, and on larger systems of values, social institutions, and discursive practices (Kirmayer, 2006).

None of these distinctions means that we must dispense with constructing categories, but we must recognize that the larger social contexts of psychiatry—including cultural notions of personhood and affliction—loop back at multiple levels into our nosology; into the process of clinical assessment and diagnosis; and into the vicious circles of attention, attribution, and behavior that constitute many forms of psychopathology (Kirmayer & Sartorius, 2007). Psychiatric nosologies, therefore, do not simply describe problems out there in the world, but actively contribute to the ways in which people construe and experience their distress. That is, as we have seen, psychiatric nosologies actively create culture even as they reflect cultural processes and practices.

Implications for Research

To the extent that social processes of meaning construction and positioning are central to the cause, course, and outcome of various forms of psychopathology, research must include systematic attention to the range of variables reflecting cultural variations in human experience. Given the marked heterogeneity within ethnocultural groups, this must go beyond mere comparisons of individuals on the basis of their ethnic identity, to examine the impact of specific knowledge, behaviors, or practices that can be linked to putative psychopathological processes. This type of research would decompose "culture" and "ethnicity" into explicit components or dimensions (e.g., specific practices associated with the body, concepts of personhood, explanations of affliction, techniques of healing) that can be studied in interaction with other biological, psychological, and social processes. However, a basic insight of anthropology is that the components of culture are not arbitrarily arranged, but constitute coherent systems (even if they contain tensions and contradictions), so that the interactions between different components must be studied to understand the tradeoffs that may occur for individuals following one or another illness trajectory.

Beyond this incorporation of culture as sets of interrelated components, "factors," or parameters that configure human biology, psychology, and the social contexts that govern behavior (whether pathological or adaptive), we have argued that the refinement of theory in psychopathology requires systematic attention to the social, cultural, and historical dimensions of human suffering and of the conceptual systems we devise to categorize, explain, and intervene. These deserve critical analysis not only for political reasons, since they have served as instruments of oppression or exclusion, but also because, in the nature of human experience, our conceptual categories shape our lives in ways that can give rise to new types of problems and solutions. Study of these "intentional kinds" and social looping effects requires different methods from those that currently dominate psychopathology research, including the critical and interpretive strategies of the social sciences, but also empirical studies of the social, economic, and political shaping of psychiatric knowledge and practice (Healy, 2004; Horwitz & Wakefield, 2007). This social analysis is not only a corrective to the tendency to promote specific models that serve special interests; it also opens a space for fresh thinking about the nature of psychopathology and well-being.

Implications for Clinical Practice

Although psychiatric diagnoses serve as a form of explanation, they differ in important ways from the biographical accounts common in personal narratives (McHugh & Slavney, 1988). Psychiatric nosologies contain generic information on postulated diseases and disorders. The act of diagnosis maps a patient's idiosyncratic story and clinical presentation onto a general set of categories. It does this by abstracting the essential characteristics of the patient's history and illness experience, paring away the irrelevant details, and seeing through the obscuring masks of style of narration and illness behavior to uncover the essence of a prototypical disorder. This, at least, is how disease categories and nosologies are constructed as systems of ideal types. Some concession to individual variability in illness manifestations occurs in the construction of polythetic categories, in which a case needs only a certain number of symptoms from a list to meet diagnostic criteria.

In contrast to this abstracting, decontextualizing, and essentializing process in the construction of disease categories, clinical explanation moves in the opposite direction. To convey a meaningful diagnosis to a patient and plan an appropriate clinical response, the clinician must particularize, qualify, and contextualize illness explanations. Often, however, clinicians simply present a generic story to patients modeled on the disease prototype. Unfamiliar symptoms or problems are reinterpreted to fit a specific prototype, or discounted and ignored as minor and irrelevant. Insofar as a patient's experience does not fit the template, the discrepancies are viewed as irrelevant or the patient is viewed as a poor historian, oblivious to or misinterpreting the true nature of his or her condition (Kirmayer, 1988, 1994). This stripping down of illness experience to fit the diagnostic paradigm is justified on the basis of the notion that diagnostic entities have essential biological characteristics, and that what is crucial about the patient's condition can be typified by these core features. However, this does not address the basic mandate of medicine. People bring symptoms and predicaments to their doctors, not just diseases or disorders. These predicaments may contribute to the cause, course, and outcome of specific disorders. Because these predicaments are socially constituted, they will differ across social and cultural contexts, giving rise to potentially important differences in the nature of psychopathology.

Strategies for including social and cultural context in clinical assessment of psychopathology include the cultural formulation presented in an appendix to DSM-IV-TR (APA, 2000). This was introduced by a working group of psychiatrists, psychologists, and anthropologists, to provide a minimal list of the sorts of contextual factors to be considered in assessing psychopathology (Mezzich et al., 1999). The list includes identity, illness explanations, functioning, family or social supports, and the relationship with the clinician. Since its introduction, many case studies using the cultural formulation have been published, but there has been no systematic assessment of its utility. Given all of the arguments we have adduced above, it would seem that something akin to the cultural formulation is crucial to provide balance to the decontextualized view of problems inherent in DSM. Clinical experiences with cultural consultation clearly demonstrate the potential of the cultural formulation to identify errors in diagnosis and produce more comprehensive and culturally appropriate assessment and treatment plans (Kirmayer, Groleau, Guzder, Blake, & Jarvis, 2003). Much further work is needed to elaborate the cultural formulation, evaluate its utility, and give it more prominence as a way to foster clinical thinking that moves from abstract category to lived reality.

Conclusion

In this chapter, we have considered what is at stake in the assimilation of local discourses of distress into the increasingly global discourse of neo-Kraepelinian psychiatry—with its overt construal of various forms of psychopathology as "natural kinds" arising from distinctive, underlying pathophysiologies. We have outlined an alternative perspective that gives due weight to culture in the study of psychopathological experience. This view centers on the importance of meaningful human predicaments as a way to understand the interaction of biological, psychological, and social processes in the emergence of distinct (though not discrete) forms of psychopathology—forms that depend to varying degrees on social context for their shape, content, and "natural history." In this view, the diagnostic entities found in psychiatric nosologies may not reflect natural kinds (occurring in nature independently of our cognitive and cultural construals), but are the outcomes of social-interactional and historical processes that include our culturally mediated ways of understanding and interpreting human suffering.

For the most part, culture functions as a taken-for-granted background that sustains our common sense and tacit knowledge of the social world, as well as our clinical models, institutions, and practices. We recognize culture only at the margins, in the encounter with those we view as different or "other." Confronting our own cultural assumptions through encounters with others has been commonplace throughout human history, but most often cultural diversity has been subordinated to a single set of categories, concepts, and values imposed by dominant

groups, which work to devalue and disqualify alternative frameworks for experience.

Psychiatry itself has practiced this form of conceptual imperialism, and challenges to this hegemonic view are few and far between. In recent years, however, migration and telecommunications have brought new levels of cultural diversity into clinical settings in many parts of the world. Culture then presents itself as a daily problem of recognizing and addressing diversity in the clinical application of psychiatric nosology (Kirmayer & Minas, 2000). This diversity cannot be addressed with theories of psychopathology and a psychiatric nosology based on research conducted in only one or a few cultural contexts (Alarcón et al., 2002). Enlarging the study of psychopathology by emphasizing the contextual shaping of psychiatric problems holds the prospect of generating a more accurate view of the sources of suffering and the mechanisms of psychopathology. Attention to culture, then, is not only a matter of serious ethical, political, and pragmatic issues in the delivery of mental health care, but a basic requirement for a science of psychopathology that seeks to understand our nature as cultural beings. Human biology is *cultural* biology. The wisdom—we should say the necessity—of attending to culture in the development of psychopathological theory and in the preparation of future nosologies therefore emerges on the grounds of both scientific and political aspirations.

Acknowledgment

Portions of this chapter are from Kirmayer (2005). Copyright 2005 by S. Karger AG, Basel. Adapted by permission.

Notes

1. We use scare quotes here to acknowledge the fact that the "phenomenology" of DSM-III (APA, 1980) and DSM-IV (APA, 1994) gives scant recognition to the realm of inner experience explored by several generations of phenomenologically oriented philosophers and psychologists working within a Continental tradition. Instead, "phenomenology" in the DSM system means discrete symptoms, signs, and behaviors that can be reliably measured by an external observer.
2. In fact, the distinction between natural and intentional kinds (like that between positivist and constructivist epistemologies) is overstated, and the examples themselves point to the difficulty of making a sharp contrast. Ian Hacking (1999) has described the wide range of uses of the notion of social construction, and has also provided some compelling examples of the social construction of psychiatric disorders (Hacking, 1995b, 1998). However, most examples of intentional categories in the area of psychiatry are what Hacking has called "interactive kinds," built out of an interaction between more or less obdurate features of the natural world (including our own physiology and psychology) and socially mediated responses.
3. Historically, in the study of culture and psychopathology, this has been framed as a contrast between "pathogenesis," usually assumed to involve biological processes or physical interactions with the environment, and "pathoplasticity," the cultural shaping of the expressions of more basic pathogenic processes.
4. One of us has argued this claim in more detail in a series of papers (see Gone, 2003a, 2004a, 2004b, 2006b, 2008a, 2009, in press-b; Gone & Alcántara, 2007).
5. In the traditions of many indigenous peoples—particularly those who were hunters—animals and other "natural" beings were seen to possess some of the same qualities of human personhood (e.g., autonomy, intentionality, and so forth) and hence are best termed "other-than-human Persons." The capital P serves to convey respect for their often sacred status.

References

Abu-Lughod, L., & Lutz, C. A. (1990). Introduction: Emotion, discourse, and the politics of everyday life. In C. A. Lutz & L. Abu-Lughod (Eds.), *Language and the politics of emotion* (pp. 1–23). Cambridge, UK: Cambridge University Press.

Alarcón, R. D., Bell, C. C., Kirmayer, L. J., Lin, K.-H., Ustun, T. B., & Wisner, K. L. (2002). Beyond the funhouse mirrors: Research agenda on culture and psychiatric diagnosis. In D. J. Kupfer, M. B. First, & D. A. Regier (Eds.), *A research agenda for DSM-V* (pp. 219–289).

Washington, DC: American Psychiatric Association.

American Psychiatric Association (APA). (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

American Psychiatric Association (APA). (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., rev.). Washington, DC: Author.

American Psychiatric Association (APA). (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

American Psychiatric Association (APA). (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.

Beard, G. (1869). Neurasthenia, or nervous exhaustion. *Boston Medical and Surgical Journal*, *3*(13), 217–221.

Beutler, L. E., & Malik, M. L. (2002). *Rethinking the DSM: A psychological perspective*. Washington, DC: American Psychological Association.

Bhugra, D., & Littlewood, R. (Eds.). (2001). *Colonialism and psychiatry*. New Delhi: Oxford University Press.

Blashfield, R. K. (1984). *The classification of psychopathology: Neo-Kraepelinian and quantitative approaches*. New York: Plenum Press.

Brave Heart, M. Y. H., & DeBruyn, L. M. (1998). The American Indian Holocaust: Healing historical unresolved grief. *Journal of the National Center*, *8*(2), 60–82.

Brinkmann, S. (2005). Human kinds and looping effects in psychology. *Theory and Psychology*, *15*(6), 769–791.

Canino, G., Lewis-Fernandez, R., & Bravo, M. (1997). Methodological challenges in cross-cultural mental health research. *Transcultural Psychiatry*, *34*(2), 163–184.

Caplan, N., & Nelson, S. D. (1973). On being useful: The nature and consequences of psychological research on social problems. *American Psychologist*, *28*(3), 199–211.

Collins, H., & Pinch, T. (1993). *The Golem: What everyone should know about science*. Cambridge, UK: Cambridge University Press.

Cooper, R. (2004). Why Hacking is wrong about human kinds. *British Journal for the Philosophy of Science*, *55*, 73–85.

Crews, F. (1995). *The memory wars: Freud's legacy in dispute*. New York: New York Review of Books.

Darwin, C. (1965). *The expression of the emotions in man and the animals*. Chicago: University of Chicago Press. (Original work published 1872)

Davidson, J., Foa, E. B., Blank, A. S., Brett, E. A., Fairbank, J., Green, B. L., et al. (1996). Posttraumatic stress disorder. In T. A. Widiger, A. J. Frances, H. A. Pincus, R. Ross, M. B. First, & W. W. Davis (Eds.), *DSM-IV sourcebook* (Vol. 2, pp. 577–605). Washington, DC: American Psychiatric Association.

Eisenberg, L. (1977). Disease and illness. *Culture, Medicine, and Psychiatry*, *1*, 9–23.

Ekman, P. (1984). Expression and the nature of emotion. In K. Scherer & P. Ekman (Eds.), *Approaches to emotion* (pp. 319–343). Hillsdale, NJ: Erlbaum.

Fanon, F. (1982). *Black skin, white masks* (1st Evergreen ed.). New York: Grove Press.

Frueh, B. C., Elhai, J. D., Grubaugh, A. L., Monnier, J., Kashdan, T. B., Sauvageot, J. A., et al. (2005). Documented combat exposure of US veterans seeking treatment for combat-related post-traumatic stress disorder. *British Journal of Psychiatry*, *186*, 467–472.

Gaines, A. D. (1992). From DSM-I to III-R: Voices of self, mastery and the other: A cultural constructivist reading of U.S. psychiatric classification. *Social Science and Medicine*, *35*(1), 3–24.

Geertz, C. (1973). *The interpretation of cultures*. New York: Basic Books.

Gilroy, P. (2004). *Postcolonial melancholia*. New York: Columbia University Press.

Gleaves, D. H. (1996). The sociocognitive model of dissociative identity disorder: A reexamination of the evidence. *Psychological Bulletin*, *120*, 42–59.

Gone, J. P. (2003a). American Indian mental health service delivery: Persistent challenges and future prospects. In J. S. Mio & G. Y. Iwamasa (Eds.), *Culturally diverse mental health: The challenges of research and resistance* (pp. 211–229). New York: Brunner-Routledge.

Gone, J. P. (2003b, November). DSM dilemmas: Ruminations on the culture of classification and the discourse of diagnosis. In J. P. Gone & T. D. O'Nell (Co-Chairs), *Decolonizing psychiatry: Revisiting the role of DSM criteria in Native American mental health care*. Symposium conducted at the annual meeting of the American Anthropological Association, Chicago.

Gone, J. P. (2004a). Keeping culture in mind: Transforming academic training in professional psychology for Indian country. In D. A.

Mihesuah & A. Cavender Wilson (Eds.), *Indigenizing the academy: Transforming scholarship and empowering communities* (pp. 124–142). Lincoln: University of Nebraska Press.

Gone, J. P. (2004b). Mental health services for Native Americans in the 21st century United States. *Professional Psychology: Research and Practice*, *35*(1), 10–18.

Gone, J. P. (2006a). Mental health, wellness, and the quest for an authentic American Indian identity. In T. Witko (Ed.), *Mental health care for urban Indians: Clinical insights from Native practitioners* (pp. 55–80). Washington, DC: American Psychological Association.

Gone, J. P. (2006b). Research reservations: Response and responsibility in an American Indian community. *American Journal of Community Psychology*, *37*(3–4), 333–340.

Gone, J. P. (2007). "We never was happy living like a Whiteman": Mental health disparities and the postcolonial predicament in American Indian communities. *American Journal of Community Psychology*, *40*(3–4), 290–300.

Gone, J. P. (2008a). Introduction: Mental health discourse as Western cultural proselytization. *Ethos*, *36*(3), 310–315.

Gone, J. P. (2008b). The Pisimweyapiy Counselling Centre: Paving the red road to wellness in northern Manitoba. In J. B. Waldram (Ed.), *Aboriginal healing in Canada: Studies in therapeutic meaning and practice* (pp. 131–203). Ottawa, ON, Canada: Aboriginal Healing Foundation.

Gone, J. P. (2008c). "So I can be like a Whiteman": The cultural psychology of space and place in American Indian mental health. *Culture and Psychology*, *14*(3), 369–399.

Gone, J. P. (2009). Encountering professional psychology: Re-envisioning mental health services for Native North America. In L. J. Kirmayer & G. G. Valaskakis (Eds.), *Healing traditions: The mental health of Aboriginal peoples in Canada* (pp. 419–439). Vancouver: University of British Columbia Press.

Gone, J. P. (in press-a). A community-based treatment for Native American historical trauma: Prospects for evidence-based practice. *Journal of Consulting and Clinical Psychology*.

Gone, J. P. (in press-b). "I came to tell you of my life": Narrative expositions of "mental health" in an American Indian community. In M. Aber, K. Maton, & E. Seidman (Eds.), *Empowering settings and voices for social change*. New York: Oxford University Press.

Gone, J. P. (in press-c). Psychotherapy and traditional healing for American Indians: Exploring the prospects for therapeutic integration. *The Counseling Psychologist*.

Gone, J. P., & Alcántara, C. (2007). Identifying effective mental health interventions for American Indians and Alaska Natives: A review of the literature. *Cultural Diversity and Ethnic Minority Psychology*, *13*(4), 356–363.

Gone, J. P., Miller, P. J., & Rappaport, J. (1999). Conceptual self as normatively oriented: The suitability of past personal narrative for the study of cultural identity. *Culture and Psychology*, *5*(4), 371–398.

Griffiths, P. E. (1997). *What emotions really are: The problem of psychological categories*. Chicago: University of Chicago Press.

Griffiths, P. E. (2004). Is emotion a natural kind? In R. C. Solomon (Ed.), *Thinking about feeling: Contemporary philosophers on emotions* (pp. 233–249). New York: Oxford University Press.

Gross, D. M. (2006). *The secret history of emotion: From Aristotle's rhetoric to modern brain science*. Chicago: University of Chicago Press.

Hacking, I. (1995a). The looping effect of human kinds. In D. Sperber, D. Premack, & A. J. Premack (Eds.), *Causal cognition: A multidisciplinary debate* (pp. 351–383). Oxford, UK: Oxford University Press.

Hacking, I. (1995b). *Rewriting the soul*. Princeton, NJ: Princeton University Press.

Hacking, I. (1996). Memory sciences, memory politics. In P. Antze & M. Lambek (Eds.), *Tense past: Cultural essays in trauma and memory* (pp. 67–87). New York: Routledge.

Hacking, I. (1998). *Mad travelers: Reflections on the reality of transient mental illnesses*. Charlottesville: University Press of Virginia.

Hacking, I. (1999). *The social construction of what?* Cambridge, MA: Harvard University Press.

Harré, R. (Ed.). (1986). *The social construction of emotions*. Oxford, UK: Blackwell.

Healy, D. (2004). *Let them eat Prozac: The unhealthy relationship between the pharmaceutical industry and depression*. New York: New York University Press.

Henningsen, P., & Kirmayer, L. J. (2000). Mind beyond the net: Implications of cognitive neuroscience for cultural psychiatry. *Transcultural Psychiatry*, *37*(4), 467–494.

Herman, J. (1992). *Trauma and recovery*. New York: Basic Books.

Hinton, A. (Ed.). (1999). *Biocultural approaches*

to the emotions. Cambridge, UK: Cambridge University Press.

Hinton, D., & Hinton, S. (2002). Panic disorder, somatization, and the new cross-cultural psychiatry: The seven bodies of a medical anthropology of panic. *Culture, Medicine, and Psychiatry*, *26*(2), 155–178.

Hinton, D., Hinton, S., Pham, T., Chau, H., & Tran, M. (2003). "Hit by the wind" and temperature shift panic among Vietnamese refugees. *Transcultural Psychiatry*, *40*(3), 342–376.

Horwitz, A. V., & Wakefield, J. C. (2007). *The loss of sadness: How psychiatry transformed normal sorrow into depressive disorder*. New York: Oxford University Press.

Jackson, L. (2005). *Surfacing up: Psychiatry and social order in colonial Zimbabwe, 1908–1968*. Ithaca, NY: Cornell University Press.

James, W. (1890). *The principles of psychology*. New York: Holt.

Keller, R. C. (2007). *Colonial madness: Psychiatry in French North Africa*. Chicago: University of Chicago Press.

Kendell, R. E. (1989). Clinical validity. *Psychological Medicine*, *19*, 45–55.

Kilpatrick, D. G., Resnick, H. S., Freedy, J. R., Pelcovitz, D., Resick, P., Roth, S., et al. (1998). Posttraumatic stress disorder field trial: Evaluation of the PTSD construct—Criterion A through E. In T. A. Widiger, A. J., Frances, H. A. Pincus, R. Ross, M. B. First, W. W. Davis, et al. (Eds.), *DSM-IV sourcebook* (Vol. 4, pp. 803–838). Washington, DC: American Psychiatric Association.

Kinzie, J. D., & Goetz, R. R. (1996). A century of controversy surrounding posttraumatic stress-spectrum syndromes: The impact on DSM-III and DSM-IV. *Journal of Traumatic Stress*, *9*(2), 159–179.

Kirmayer, L. J. (1988). Mind and body as metaphors: Hidden values in biomedicine. In M. Lock & D. Gordon (Eds.), *Biomedicine examined* (pp. 57–92). Dordrecht, The Netherlands: Kluwer.

Kirmayer, L. J. (1994). Improvisation and authority in illness meaning. *Culture, Medicine, and Psychiatry*, *18*(2), 183–214.

Kirmayer, L. J. (1996). Confusion of the senses: Implications of ethnocultural variations in somatoform and dissociative disorders for PTSD. In A. J. Marsella, M. J. Friedman, E. T. Gerrity, & R. M. Scurfield (Eds.), *Ethnocultural aspects of post-traumatic stress disorders: Issues, research and clinical applications* (pp. 131–164). Washington, DC: American Psychological Association.

Kirmayer, L. J. (2005). Culture, context, and experience in psychiatric diagnosis. *Psychopathology*, *38*, 192–196.

Kirmayer, L. J. (2006). Beyond the "new cross-cultural psychiatry": Cultural biology, discursive psychology and the ironies of globalization. *Transcultural Psychiatry*, *43*(1), 126–144.

Kirmayer, L. J. (2007a). Cultural psychiatry in historical perspective. In D. Bhugra & K. Bhui (Eds.), *Textbook of cultural psychiatry* (pp. 3–19). Cambridge, UK: Cambridge University Press.

Kirmayer, L. J. (2007b). Psychotherapy and the cultural concept of the person. *Transcultural Psychiatry*, *44*(2), 232–257.

Kirmayer, L. J., Groleau, D., Guzder, J., Blake, C., & Jarvis, E. (2003). Cultural consultation: A model of mental health service for multicultural societies. *Canadian Journal of Psychiatry*, *48*(3), 145–153.

Kirmayer, L. J., & Jarvis, G. E. (2005). Depression across cultures. In D. Stein, A. Schatzberg, & D. Kupfer (Eds.), *Textbook of mood disorders* (pp. 611–629). Washington, DC: American Psychiatric Press.

Kirmayer, L. J., Lemelson, R., & Barad, M. (Eds.). (2007). *Understanding trauma: Integrating biological, clinical, and cultural perspectives*. New York: Cambridge University Press.

Kirmayer, L. J., & Minas, H. (2000). The future of cultural psychiatry: An international perspective. *Canadian Journal of Psychiatry*, *45*(5), 438–446.

Kirmayer, L. J., & Sartorius, N. (2007). Cultural models and somatic syndromes. *Psychosomatic Medicine*, *69*(9), 832–840.

Kirmayer, L. J., & Young, A. (1999). Culture and context in the evolutionary concept of mental disorder. *Journal of Abnormal Psychology*, *108*(3), 446–452.

Kitayama, S., & Markus, H. R. (1994). *Emotion and culture: Empirical studies of mutual influence*. Washington, DC: American Psychological Association.

Kleinman, A. (1977). Depression, somatisation, and the new "cross-cultural psychiatry." *Social Science and Medicine*, *11*, 3–10.

Kleinman, A. (1986). *Social origins of distress and disease: Depression, neurasthenia, and pain in modern China*. New Haven, CT: Yale University Press.

Kleinman, A. (1988). *Rethinking psychiatry*. New York: Free Press.

Klerman, G. L. (1978). The evolution of a scientific nosology. In J. C. Shershow (Ed.), *Schizophrenia: Science and practice* (pp. 99–121). Cambridge, MA: Harvard University Press.

Konner, M. (2007). Trauma, adaptation, and resilience: A cross-cultural and evolutionary perspective. In L. J. Kirmayer, R. Lemelson, & M. Barad (Eds.), *Understanding trauma: Biological, psychological and cultural perspectives* (pp. 300–338). New York: Cambridge University Press.

Leavitt, J. (1996). Meaning and feeling in the anthropology of emotions. *American Ethnologist*, *23*(3), 514–539.

Lee, S. (1998). Estranged bodies, simulated harmony, and misplaced cultures: Neurasthenia in contemporary Chinese society. *Psychosomatic Medicine*, *60*, 448–457.

Lerner, P. (2003). *Hysterical men: War, psychiatry, and the politics of trauma in Germany, 1890–1930*. Ithaca, NY: Cornell University.

Leys, R. (1996). Traumatic cures: Shell shock, Janet, and the question of memory. In P. Antze & M. Lambek (Eds.), *Tense past: Cultural essays in trauma and memory* (pp. 103–145). New York: Routledge.

Leys, R. (2000). *Trauma: A genealogy*. Chicago: University of Chicago Press.

Littlewood, R. (1990). From categories to contexts: A decade of the "new cross-cultural psychiatry." *British Journal of Psychiatry*, *156*, 308–327.

Lutz, C., & White, G. M. (1986). The anthropology of the emotions. *Annual Review of Anthropology*, *15*, 405–436.

MacCorquodale, K., & Meehl, P. E. (1948). On a distinction between hypothetical constructs and intervening variables. *Psychological Review*, *55*, 95–107.

McCulloch, J. (1995). *Colonial psychiatry and "the African mind."* Cambridge, UK: Cambridge University Press.

McHugh, P. R., & Slavney, P. R. (1998). *The perspectives of psychiatry* (2nd ed.). Baltimore: Johns Hopkins University Press.

McNally, R. (2003). Progress and controversy in the study of posttraumatic stress disorder. *Annual Review of Psychology*, *54*, 229–252.

Mezzich, J., Kirmayer, L. J., Kleinman, A., Fabrega, H., Jr., Parron, D. L., Good, B. J., et al. (1999). The place of culture in DSM-IV. *Journal of Nervous and Mental Disease*, *187*(8), 457–464.

Mezzich, J., Kleinman, A., Fabrega, H., Jr., & Parron, D. (Eds.). (1996). *Culture and psychiatric diagnosis*. Washington, DC: American Psychiatric Press.

Millon, T. (1991). Classification in psychopathology: Rationale, alternatives, and standards. *Journal of Abnormal Psychology*, *100*(3), 245–261.

Morey, L. C. (1991). Classification of mental disorder as a collection of hypothetical constructs. *Journal of Abnormal Psychology*, *100*(3), 289–293.

Oatley, K., & Jenkins, J. M. (1992). Human emotions: Function and dysfunction. *Annual Review of Psychology*, *43*, 55–85.

O'Nell, T. D. (1996). *Disciplined hearts: History, identity, and depression in an American Indian community*. Berkeley: University of California Press.

Pole, N., Gone, J. P., & Kulkarni, M. (2008). Posttraumatic stress disorder among ethnoracial minorities in the United States. *Clinical Psychology: Science and Practice*, *15*(1), 35–61.

Rabinow, P., & Sullivan, W. M. (Eds.). (1987). *Interpretive social science: A second look*. Berkeley: University of California Press.

Reddy, W. M. (2001). *The navigation of feeling: A framework for the history of emotions*. New York: Cambridge University Press.

Robert, J. S. (2007). Gene maps, brain scans, and psychiatric nosology. *Cambridge Quarterly of Health Care Ethics*, *16*, 209–218.

Robert, J. S., & Plantikow, T. (2005). Genetics, neuroscience and psychiatry. *Psychopathology*, *38*(4), 215–218.

Robins, E., & Guze, S. B. (1970). Establishment of diagnostic validity in psychiatric illness: Its application to schizophrenia. *American Journal of Psychiatry*, *126*, 983–987.

Rosaldo, M. Z. (1984). Toward an anthropology of self and feeling. In R. A. Shweder & R. A. LeVine (Eds.), *Culture theory: Essays on mind, self, and emotion* (pp. 137–157). Cambridge, UK: Cambridge University Press.

Sadowsky, J. H. (1999). *Imperial bedlam: Institutions of madness in colonial southwest Nigeria*. Berkeley: University of California Press.

Scott, W. (1990). PTSD in DSM-III: A case in the politics of diagnosis and disease. *Social Problems*, *37*(3), 294–310.

Shweder, R. A. (1988). Suffering in style. *Culture, Medicine, and Psychiatry*, *12*, 479–497.

Shweder, R. A. (1991). *Thinking through cultures: Expeditions in cultural psychology*. Cambridge, MA: Harvard University Press.

Shweder, R. A. (1993). The cultural psychology of the emotions. In M. Lewis & J. M. Haviland (Eds.), *Handbook of emotions* (pp. 417–431). New York: Guilford Press.

Silove, D. (1999). The psychosocial effects of torture, mass human rights violations, and refugee trauma. *Journal of Nervous and Mental Disease*, *187*(4), 200–207.

Trimble, M. R. (1985). Post-traumatic stress disorder: History of a concept. In C. R. Figley (Ed.), *Trauma and its wake* (pp. 5–14). New York: Brunner/Mazel.

Vergès, F. (1996). To cure and to free: The Fanonian project of "decolonized psychiatry." In L. R. Gordon, T. D. Sharpley-Whiting, & R. T. White (Eds.), *Fanon: A critical reader* (pp. 85–99). Oxford, UK: Blackwell.

Wexler, B. E. (2006). *Brain and culture: Neurobiology, ideology, and social change*. Cambridge, MA: MIT Press.

White, G. M. (1993). Emotions inside out: The anthropology of affect. In M. Lewis & J. M. Haviland (Eds.), *Handbook of emotions* (pp. 29–39). New York: Guilford Press.

Wierzbicka, A. (1999). *Emotions across languages and cultures: Diversity and universals*. Cambridge, UK: Cambridge University Press.

Wilson, M. (1993). DSM-III and the transformation of American psychiatry: A history. *American Journal of Psychiatry*, *150*(3), 399–410.

Woodruff, R. A., Goodwin, D. W., & Guze, S. B. (1974). *Psychiatric diagnosis*. New York: Oxford University Press.

Yehuda, R., & McFarlane, A. C. (1995). Conflict between current knowledge about posttraumatic stress disorder and its original conceptual basis. *American Journal of Psychiatry*, *152*(12), 1705–1713.

Young, A. (1991). Emil Kraepelin and the origins of American psychiatric diagnosis. In B. Pfleiderer & G. Bibeau (Eds.), *Curare: Vol. 7. Anthropologies of medicine: A colloquium on west European and North American perspectives* (pp. 175–181). Dordrecht: Association of Ethnomedicine.

Young, A. (1995). *The harmony of illusions: Inventing posttraumatic stress disorder*. Princeton, NJ: Princeton University Press.

Young, A. (1996a). Bodily memory and traumatic memory. In P. Antze & M. Lambek (Eds.), *Tense past: Cultural essays in trauma and memory* (pp. 89–102). New York: Routledge.

Young, A. (1996b). Suffering and the origins of traumatic memory. *Daedalus*, *125*(1), 245–260.

Young, A. (2007). Bruno and the Holy Fool: Myth, mimesis and the transmission of traumatic memories. In L. J. Kirmayer, R. Lemelson, & M. Barad (Eds.), *Understanding trauma: Integrating biological, clinical, and cultural perspectives* (pp. 339–362). New York: Cambridge University Press.

Zheng, Y. P., Lin, K. M., Takeuchi, D., Kurasaki, K. S., Wang, Y., & Cheung, F. (1997). An epidemiological study of neurasthenia in Chinese-Americans in Los Angeles. *Comprehensive Psychiatry*, *38*(5), 249–259.

CHAPTER 4

Cultural Issues in the Coordination of DSM-V and ICD-11

RENATO D. ALARCÓN

One of the most interesting epistemological developments in contemporary psychiatry is the increasing recognition of the role played by culture and cultural factors in all aspects (clinical, diagnostic, therapeutic, and prognostic) of the entities we know as mental disorders. This realization is even more impressive if we consider that the renewed acceptance of cultural components is taking place in the midst of an undeniable predominance of neurobiological research in the field (Lopez-Ibor, Gaebel, Maj, & Sartorius, 2002). Many reasons have been invoked to explain the phenomenon: the need for a multidisciplinary contribution to the delineation and expression of symptoms, syndromes, and nosological labels; the pervasive findings of differences in the actual experience, description, and expression of clinical manifestations across different societies, historical periods, or geographic regions; and the impact of globalization, both as a social occurrence with complex characteristics, and as a political and economic event requiring better explanations and fairer applications in today's world (Bauman, 1998; Stiglitz, 2002). Globalization is nourished by massive migrations and displacements of peoples and communities to and from entirely different milieus, for a variety of reasons (poverty, war, natural disasters, political exile, etc.). In turn, it leads to the realities of multiculturalism and of interfaces between cultures across the world—other sources of stress, as well as of hope (Simmons, 2002). The actual impact of globalization, migration, and multiculturalism is thus reflected in the unique context of the cultural background of each and every individual. If and when such experiences evolve into clinically detectable features, a cultural approach becomes essential in the description and characterization of the resulting diagnoses (Mastrogianni & Bhugra, 2003).

The need to study and systematize the cultural aspects of all psychiatric conditions has been one of the main driving forces in the field of "cultural psychiatry." Defined as the discipline that examines, among other topics, the context and meaning of morbid (abnormal or "pathological") emotional behaviors along the lines of a true biopsychosocial model (Engel, 1977), cultural psychiatry uses a set of unique variables (language, religion, traditions, beliefs, ethnicity, gender, sexual orientation, etc.) to ascertain

the real (as opposed to stereotyped) nature of mental suffering. Thus culture definitely shapes a variety of clinical dimensions and plays a variety of roles in the assessment of any given patient; it is an interpretive/explanatory tool, a pathogenic/pathoplastic factor, a diagnostic/nosological instrument, a therapeutic/protective intervention, and a service/management component (Alarcón, Westermeyer, Foulks, & Ruiz, 1999). Cultural psychiatry therefore aims at preserving the essential integration of the human condition. It is the search for a genuinely monistic, truly comprehensive clinical assessment of each patient.

The growth of cultural psychiatry, and its implications for the field in general, make it also necessary to ascertain what the discipline is not. Cultural psychiatry is not a psychiatric subspecialty; a "rehash" of old ideas; a political ploy; the study of just minorities, immigrants, or refugees; a collection of rhetoric; the observations of exotic people in distant lands; or an anti-biological-psychiatry movement (Alarcón, 1998, 2007). In parallel with its professed comprehensiveness, however, cultural psychiatry strives to define its boundaries, while maintaining that all human beings always show either hints or massive evidence of their cultural makeup—quite particularly while experiencing an illness, be that physical or mental (a distinction that unfortunately still permeates significantly the whole field of medicine). As such, it cannot evolve in isolation from other considerations of disease as a peculiar human status, especially those pertaining to mental or emotional conditions.

The preceding concepts are useful in the examination of the role of culture in psychiatric diagnosis. There are no doubt significant differences between the purely nosological and the cultural perspectives in regard to psychiatric diagnosis. The former perspective is disease-centered and individualized; the latter is person-centered but also focuses on social networks. Nosology is "essentialist," focused on specific, "purified" symptoms or diagnostic categories; culture is "contextualized," integrative, interconnected (Kleinman, 1988). The nosological perspective tends to be based on biomedical technology, whereas the cultural perspective may be more oriented toward psychosocial components and interventions. Furthermore, most of the medical specialties and subspecialties claim that in addition to specific symptomatology, they can count on generally reliable laboratory tests, technically known as "biomarkers," to confirm or rule out any given diagnostic option under consideration (Follette & Houts, 1996). Clinical psychiatry, as is well known, is lacking in pathognomonic symptoms, and is still looking for solid biological markers; therefore, its approach is still mostly based on a detailed, more or less rigorous gathering of chronological history, collection of information from sources other than the patient him- or herself, determination of main and collateral symptoms, examination of clinical course, and the use of some measurement instruments.

The concepts of "cultural competence" and "cultural fluency" must inform every attempt to introduce culture into the process of psychiatric diagnosis (Johnson, Saha, Arbelaez, Beach, & Cooper, 2004; Tseng & Strelzer, 2004). Although more frequently used in the field of treatment and provision of care, these terms point to skills and abilities that enhance communication, as well as to the context and background in which the diagnostic encounter takes place. Culturally oriented clinical inquiries lie behind clinical descriptions, biological measurements, and so-called "socioenvironmental" factors, all operating as well in the diagnostic arena.

Of all the areas of interaction between culture and psychiatry mentioned above, the one centered on diagnosis and nosology may be most in need of competent and serious attention from clinicians, researchers, and administrators alike. Although the notions of explanatory models of mental illness (Kleinman, 1988), behavioral styles influenced by both family microculture and broad environmental (cultural) factors, cultural competence on the part of providers (Tseng & Streltzer, 2004), and the presence of culturally based risk and protective indices (Kuh, Power, Blane, & Bartley, 1997) have been more or less accepted for a long time, the practical details of how culture affects diagnosis, and of how psychiatric diagnosticians can recognize and identify the cultural roots of their patients' ailments, have only recently become topics of interest. It is generally agreed that only with the inclusion of cultural considerations, the "cultural formulation," and the glossary of "culture-bound

syndromes" in the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV; American Psychiatric Association [APA], 1994) was the importance of cultural concepts in psychiatry duly recognized. Moreover, the actual utilization and implementation of these concepts since then have been insufficient, generating claims of "tokenism" (Kleinman, 2008) on the one hand, and misuse or plain neglect of a promising research and practice area on the other (Kirmayer & Young, 1999).

After briefly reviewing the historical sources of diagnostic endeavors in psychiatry, this chapter examines the evolution and current status of the connections between culture and psychiatric diagnosis, followed by an assessment of inevitable political and organizational aspects of the equation. The chapter then delineates the different aspects of the process in need of coordination for both the APA's DSM-V and the mental disorders section of the 11th revision of the World Health Organization's (WHO's) *International Classification of Diseases* (ICD-11). Outlining a diagnostic structure that includes a desirable cultural component, and key cultural issues that should be part of the overall diagnostic enterprise, is another objective of the chapter. The last section summarizes the main points of the preceding analyses.

Main Sources of the Psychiatric Diagnosis Process

Historically, psychiatric diagnosis has relied on different bodies of knowledge, or study approaches, at different times. This has resulted from both the dominance (some would call it "fashionability") of specific schools of thought at a given moment in history, and the political climate among leading national or international institutions in charge of this aspect of psychiatric work (Berrios, 1996). From this vantage point, it seems possible to identify up to five sources on which psychiatric diagnosis has relied to articulate different systems throughout the last 150–200 years—a period that most consider the "coming of age" of psychiatry as a well-defined field of study and clinical practice (Leighton, 1982). These epistemological sources are as follows:

1. *Phenomenology.* A primary reliance on descriptive narratives of what the patient reports as his or her personal experiencing of "strange," "unusual," or "different" subjective phenomena has been probably the oldest and most widely used approach to diagnosis. Initially an exclusively intuitive modality, this process began to evolve when clinicians started to ascribe technical names to these occurrences, based on the different areas of psychological or behavioral functioning seemingly affected: perception, mood, thought, instincts or drives, personality, and so forth (Delgado, 1967). This was then followed by the systematic grouping of the symptoms so described, leading to syndromes and eventual nosological categories. The phenomenological approach to diagnosis had its philosophical origin in Husserl's and other existential works (Spiegelberg, 1972), but its clinical application was ultimately free of theoretical links to this school. A naturalistic, neutral observation of the clinical picture was at the core of this type of diagnosis, first utilized in France, Italy, and Germany in the late 18th and early 19th centuries, and cogently promulgated by Jaspers's *General Psychopathology* (published in Germany in 1913, and translated into English only in 1968). It seems only fair to acknowledge the phenomenological flavor of the versions of APA's DSM from DSM-III onward.

2. *Psychodynamics.* The need to "explain" symptoms has always been a driving force of every clinical endeavor, and an important part of the diagnostic task. Explanations would confer a "scientific" seal on otherwise esoteric and often terrifying experiences, thus contributing to a relative assuaging of symptom-related fears. The failure of early explanatory attempts ranging from animistic or heavily religious to mechanistic ones (the latter were nourished by the strength of positivistic thinking during the "scientific revolution"; Delgado, 1947) shaped the psychodynamic approach of psychoanalytic authors inspired by Freud's findings, their speculative, unproven nature notwithstanding (Chessick, 2003). The historical context of the Freudian contribution, particularly its "liberating" nature in the middle of a rigid Victorian era, was a factor as important in its success as the brilliance of its author's literary style. It is

well known that Freud professed the hope that his explanations of subjective/unconscious phenomena would sooner or later be superseded by the findings of chemical and other biological methods of inquiry. In fact, psychodynamic "explanations" are, more precisely, interpretations of unexplained phenomena—a historically important ingredient of a diagnostic process with aspirations of true comprehensiveness.

3. *Epidemiology.* The search for a better and more solid methodological base for scientific inquiries led to the growth and variety of epidemiological tools, which carried the banner of quantitative (and therefore, irrefutable) precision. Diagnosis jumped from an individual focus to a massive, population-oriented scale in research attempting to quantify phenomenologically obtained data on symptoms and their frequency, to define combinations of symptoms delineating clinical patterns, and to provide more consistent characterizations of syndromes and categories. Such research also seriously attempted to eliminate subjectivity in the description of symptoms. Although it sometimes paid the price of potential simplifications and/or premature generalizations, epidemiology provided useful perspectives on such important diagnostic components as gender, socioeconomic status, educational background, and the like (Richerson & Boyd, 2005). One of its soundest accomplishments was the demonstration of symptomatic homogeneity of schizophrenia across cultures, as in the International Pilot Study of Schizophrenia (WHO, 1973), even though such findings were later subjected to solid critiques (Halliburton, 2004). As the "basic science" of public health, epidemiology also timidly but decisively hinted at the importance of context—a major aspect of the cultural approach (Van Ommeren, 2003). No diagnostic system or syndromic grouping nowadays can exist without substantial epidemiological justifications.

4. *Neurobiology.* With the spectacular advances in multiple areas of neurobiological research in the last four or five decades, many now do not seem to doubt that a psychiatric diagnosis will only be both reliable and valid if and when it has a laboratory test, a genetic profile, neuroimaging documentation, or all of these together, to substantiate it. Nevertheless, this has been an elusive objective so far, in spite of the overwhelming progress in genetics, biochemistry, pharmacology, neurophysiology, and other basic sciences, as well as the conceptual and heuristic elaborations of neuroplasticity, neurocircuitry, brain microbiology, and molecular biology (Guze, 1992; Kandel, 2006; Lopez-Ibor et al., 2002). There are no firmly established "biomarkers" in clinical psychiatry and psychiatric diagnosis so far, and the debates in the field reflect some of the frustrations as well as the high expectations generated by its undeniable progress. The field of genetics is touted as the one that will provide the information needed, but the search seems to be moving from candidate genes to a more or less broad compositum of genotypes whose precision will be based on better-defined phenotypes or on reemerging endophenotypes (Eisenberg, 2005; Hasler, Drevets, Manji, & Charney, 2004).

5. *Informatics.* With an extraordinary and ever-growing number of measurement instruments of different natures, versions, characteristics, and patterns of symptoms as its initial endowment, the field of "informatics" may be the youngest source of a modern, more sophisticated process of psychiatric diagnosis (World Health Organization, 2001). Defined as a massive set of analyzable data and data banks, working at an almost quantum-like velocity and precision, informatics can perform meta-analyses, correlating tasks, inferential associations, and many more operations. It utilizes both mathematical tools and operations, as well as logical deductive procedures, providing therefore both quantitative estimates and qualitative assessments. Although it cannot be used as the only source of diagnosis, informatics will be an essential component of diagnostic work in the not-so-distant future.

Reliance on the different sources for diagnosis outlined here entails richness and unmitigated promise. Nevertheless, they all have in common the omission of a clearly stipulated cultural component as part of psychiatric diagnosis. Although it may be tacitly included, or assumed to be included, the fact that it is not specifically dealt with may be a form of denial, a sign of arrogance, or a demonstration of ignorance or distraction. Let us hope that this neglect of cultural factors will be overcome in the diagnostic

and classification systems of the 21st century and beyond.

The Role of Culture in Psychiatric Diagnosis

The history and development of the two main diagnostic systems in the world—APA's DSM and WHO's ICD—have similarities and differences, primarily related to the structure, nature, and institutional dynamics of their sponsoring organizations. The APA is a national professional organization that (due to a number of sociopolitical, financial, and demographic factors inherent in the position of the United States in the world) enjoys global influence, shares in the country's scientific accomplishments, attracts the largest number of attendees to its annual meetings, leads the psychiatric publishing market, and has DSM as one of its most impressive signs of power. That English is the de facto scientific language of the world certainly does not hurt. The APA is politically and financially stable; its leadership changes in an organized fashion; it conducts its business in a transparent way; and, in spite of inevitable internal discrepancies and debates, it works within a predictable, homogeneous frame. The WHO, on its side, is an international institution. Its work spans the globe but must respond to regional, national, and local bureaucracies and diverse political ideologies; its decision-making processes are the results of complex and unavoidable negotiations; and its finances are supported by uneven contributions from its member nations. It has regional offices in different continents, and mental health in many cases is only part of broader divisional or sectional structures. Because of all these circumstances, the WHO frequently resorts to international consultants, advisory groups, or working committees.

With the development of diagnostic and classification systems being among the main responsibilities of both organizations, it is clear that the APA conducts a more focused, more consistent, and better-financed process. The WHO's work in this area covers all kinds of diseases, and diagnoses for mental disorders constitute only one ICD section. Furthermore, the public health perspective is closer to the core of the WHO's (and therefore ICD's) work, whereas DSM may have more clinical and actuarial purposes, including its attention to and use in legal and insurance-related areas. A paradoxical reality seems to be that for the last 20–30 years, DSM (a national manual) has been more widely recognized and utilized in most countries of the world, even though political obligations dictate that disease and diagnostic codes follow the international nomenclature embodied in ICD. This has made it necessary to work on equivalencies or compatibilities between the two systems—a process that is certainly beneficial, and may have led to closer collaboration between the APA and the WHO's Mental Health Division. Actually, a series of international conferences mostly funded by APA and sponsored by both organizations took place between 2005 and early 2008. The memberships of the APA's DSM-V Committee and Work Groups have also been announced (APA, 2004, 2008). Interestingly, both organizations have ultimately relied on "expert consensus" for the elaboration of their respective systems, even though the APA supported some field trials prior to the publication of DSM-IV.

A feature common to both nomenclatures is their "benign neglect" of the cultural perspective. It must be made clear that the WHO's international vantage point is not necessarily *cultural* in its scope, structure, and substance. International psychiatry is not cultural psychiatry—honest attempts to portray it as such, or political twists or spins, notwithstanding (Kirmayer, 2006). Incidence or prevalence data do not say much about cultural characteristics, explanatory models, or idioms of distress. On the APA's side, DSM-IV was the first version of the manual that included cultural concepts, over 40 years after the publication of DSM-I. And even in DSM-IV, the cultural formulation and culture-bound syndromes were relegated to Appendix I, at page 843 of the 886-page volume (APA, 1994). Under these circumstances, criticisms of both systems seem justified (Hughes, 1998; Kirmayer & Young, 1999; Kleinman, 2008).

What are the reasons for this "lip service" that two otherwise relevant classification systems pay to the cultural cause? Why are cultural factors seen as collateral or negligible in diagnostic systems that call them-

selves "comprehensive" or "integrating"? The reasons are as complex in nature and origin as the questions themselves. First, the aim of any diagnostic catalog is to seek and emphasize *homogeneity*, uniformization, generalization, and subsequent universal acceptance. Cultural approaches, epistemologically based on the principle of relativism, are seen as purposely searching for differences, uniqueness, or singularities in countries, regions, or societies across the world—therefore preventing generalizations perceived as necessary, and nourishing *heterogeneity*, disagreement, and conflict (Bains, 2005). Second, it is not easy to articulate what aspects of the complex cultural realities of patients, families, and communities should be included in a multiaxial, descriptive, narrative, or multidimensional diagnostic system. Third, culture is considered by some either as epiphenomenal and superficial (because "it is there")—more a motionless background curtain than a main actor in the diagnostic play—or as a politically inspired and therefore divisive issue. Fourth, research on cultural topics is regarded as "soft," more the province of social sciences such as anthropology or sociology, and therefore lacking in clinical or diagnostic relevance. Fifth, psychiatrists and other mental health professionals interested in cultural issues (and, more specifically, in culture and diagnosis) have not produced research as abundant as that in other fields; have published it in a limited number of specialized journals; and have only recently strengthened their professional presence through national and international organizations, scientific meetings, and dissemination of their research efforts (Tseng, 2007).

Some of the above-described views certainly have elements of truth; others stem from the same types of arrogance, misunderstanding, or ignorance mentioned earlier. Some are the results of others' actions or ideas; others, unfortunately, represent self-inflicted damage. There seems to be agreement, however, that DSM-IV's cultural formulation (APA, 1994) is the most satisfactory currently existing tool for the assessment of cultural topics in psychiatric diagnosis (Lewis-Fernandez, 1996). Because it includes concrete information about cultural characteristics, cultural assumptions, explanatory models, style of interpersonal transactions, and an overall evaluation of the patient's cultural identity and culturally based behaviors, the cultural formulation seems to be a valuable tool, conceived as clinically useful and valid. The unfortunate reality, however, is that its utilization in clinical and teaching settings has been minimal; research on it has been equally scarce; and its value and even its existence have (again) been denied, minimized, or plainly ignored (Lewis-Fernandez & Diaz, 2002). Its narrative (ethnography-based) structure and lack of quantifiable components have been pointed out as heuristic disadvantages, but the lack of consistency in its everyday use in clinical settings has been the main reason for this situation. Comparisons with the fate of the "psychodynamic formulation" of the 1960s and 1970s are not totally fair, since the latter's popularity was related to the dominance of the psychodynamic school of thought in American psychiatry at the time—something that cultural psychiatry has never sought or had. By the same token, the demise of the dynamic formulation was closely related to the emergence of DSM-III, which reintroduced a phenomenological, neo-Kraepelinian approach to psychiatric diagnosis (Ghaemi, 2003).

Although for entirely different reasons, both biological psychiatry and cultural psychiatry currently appear to be enjoying prominent positions in the preparation of new versions of DSM and ICD. Public pronouncements by leaders of both the APA and the WHO acknowledge the need to pay attention to cultural issues, and both organizations have in fact conducted international conferences to provide mutual input into both perspectives and the documents that will result from them (Phillips, First, & Pincus, 2003). It may be that DSM-V will become more deliberately "global" (i.e., culturally flexible) than its predecessors—especially in view of the growing diversity of the U.S. population—and that the WHO will agree to incorporate more DSM-like structural and terminological components into ICD-11. In 2002, the APA published a volume titled *A Research Agenda for DSM-V*, which expressly included a chapter on cultural issues (Alarcón et al., 2002). And the WHO is, by definition, naturally more receptive than a national organization to true cultural inputs.

Political Implications

The pertinent organizational issues in both the APA and the WHO have already been mentioned. Their audiences, decision-making rules, views, and perspectives may differ, but if the purpose is to produce compatible documents, negotiations and agreements will ideally favor this goal. For obvious reasons, the APA seems to be moving more decisively; as noted above, it has established a DSM-V Committee or Task Force, Work Groups, and Study Groups, and it has also announced a timetable for the publication of the manual (APA, 2008). With the assistance of the U.S. National Institute of Mental Health, and the collateral participation of the World Psychiatric Association, the APA is demonstrating vigor in this process. Its powerful networks and communication instruments (plus global electronic systems) assure rapid, agile exchange of information. This is indeed a promising process.

Political disagreements, ideological differences, and historical animosities will require both the APA and the WHO to make good-faith efforts at understanding each other's positions and constituencies, to exhibit flexibility in recognizing differences, to acknowledge diversity, to set aside ill-conceived or narrow "-isms" (including nationalisms), and to arrive at a consensus resulting from both clinical/research-based and value-based evidence (Baca-Baldomero & Lázaro, 2005; Ruiz, Alarcón, Lolas, Lázaro, & Baca-Baldomero, 2008). As noted earlier, the aims and roles of the two organizations are different (but ideally complementary). The APA provides a pragmatic vision, the WHO a social/public health perspective; the APA has substantial resources (both human and financial), while the WHO has a well-established global setting. The APA works in a country that still enjoys superpower status (with all its positive and negative implications), while dealing with the diversity generated by migration and globalization; the WHO deals with a complex and at times chaotic world audience in the unquestionably altruistic pursuit of public health in general and public mental health in particular. Both groups—again perhaps for different reasons—agree, however, that culture is important, and that it has to be accepted and included both conceptually and practically in any new psychiatric diagnostic classification.

Other factors to keep in mind include the phenomenon of globalization, as well as the political changes of the last 30–40 years throughout the world. Globalization includes, among its multiple implications, an emotional impact of drastic proportions related to massive displacements of people, acculturation processes, economic opportunities gained and lost, new notions of family structure and relationships, new communication patterns, and so forth (Bauman, 1998; Lacroix & Shrad, 2004; Stiglitz, 2002). Cultural factors cannot be ignored in the evaluation of individuals and communities showing psychiatric symptoms that adopt different shapes and expressions from those usually seen in mainstream Western societies (Kirmayer & Young, 1999; Kleinman, 1988; Kuh et al., 1997). Political changes determined by any of numerous factors—the rise or fall of ideologies, religious wars, neocolonialism, the wealth–poverty chasm in most countries of the world, natural disasters, or environmental conflicts—all have an undeniable cultural stamp as well: They are culturally based occurrences inducing, in turn, somewhat unpredictable cultural changes. And, once again, the psychological impact of these changes is undeniable.

The relativism inherent to a cultural assessment of everything human may be an excellent tool in the elaboration of the new diagnostic systems in psychiatry. If diagnoses are going to be accurate, integrated, and thorough, culture has to be present. If the diagnoses are going to include etiopathogenesis, cultural factors must be included. If the diagnoses are going to determine comprehensive treatment strategies, cultural settings must be anticipated. If the diagnoses are going to predict or intuit outcomes, cultural assertions have to be used. Finally, if the diagnoses are going to summarize the experience of human beings, culture—the most human of products—must be part of their texture.

Those responsible for the new systems will have to adjust their working calendars to these considerations. DSM-V and ICD-11 offer unique opportunities for a genuine inclusion of cultural factors in the generation of each and every psychiatric diagnosis. Together with a substantial dose of goodwill,

clinical and research strategies and verifications must be well coordinated with the scope and structure of the new nosological catalogs.

Coordination Areas and Issues

If two new diagnostic systems are going to exist (or start to coexist) within the next 4–5 years, an intense dialogue will have to continue between the two organizations involved in the process. In this section, I examine the main areas or topics in more or less urgent need of discussion and coordination, still from the cultural perspective. The main goals will be recognizing these areas; understanding their definitions, scope, and boundaries; manualizing and instrumentalizing these areas; and adapting them to the overall structure of the system. These areas and their related issues include the following:

1. *Settings*. The need for a universal scope for a diagnostic system cannot ignore the fact that countries, geographic regions, communities, and societies across the world differ in a variety of ways. Therefore, it is inevitable that different versions or adaptable arrangements will have to be considered. The differences in health structures and health care systems must be major factors in these considerations.

2. *Diversity*. The people in these different settings carry their cultural legacies through generations, and any diagnostic system looking for relevance and cogency has to address this human diversity. "People" refers, of course, to both patients and care providers—who either share a culture, or come from different cultures but attempt to find common ground in the description, explanation, and understanding of clinical realities.

3. *Clinical variables*. Issues of crucial clinical importance (other than the symptoms themselves) that are to be part of the process of diagnosis in psychiatry should include the following:

- Informant or informants (including relationship with the patient, educational level, reliability, etc.).
- Help-seeking pattern or modality (including level and manner of information regarding the health care setting, circumstances of contact, way of reporting clinical manifestations, expectations, body language, etc.) (Rogler, Malgady, & Constantino, 1987).
- Compliance history (in the case of previous clinical and treatment contacts) or compliance potential (in the case of a first visit).
- Perception of severity of symptoms and other clinical manifestations.
- Impact of clinical condition on the patient's family group or other surrounding relationships or settings (neighborhood, workplace, church, friendships, community organizations, etc.).
- Coping style (including resilience, vulnerabilities, individual and socially based risk and protective factors, characterological strengths and weaknesses, survival strategies, etc.).
- Overall organization of the clinical report, with emphasis on eventual cultural correlations of the gathered material.

4. *Specific cultural variables*. Coordinating efforts must be intense and sustained to make the primary cultural component of both DSM-V and ICD-11 a solid and well-accepted feature (Group for the Advancement of Psychiatry, 2002). In close connection with the above-described clinical variables (already embodying clearly defined cultural qualities), the efforts must put special emphasis on the following variables:

- *Demographics*. Age, gender, educational level, occupation, and socioeconomic status are demographic items of undeniable cultural relevance, as is the setting in which the evaluation encounter takes place. All of them should be adequately described and utilized in the diagnostic assessment and subsequent clinical steps.
- *Race and ethnicity*. Although also "demographic" in nature, these two items have a unique cultural value: race as an expression of inadequate groupings of human beings on the basis of variable, often ambiguous, and highly politicized physiognomic characteristics; ethnicity as cultural legacy, anchor of membership and participation in larger social systems, and core feature of individual and collective identities (Beneduce & Martelli, 2005).

- *Language and terminology.* Because language is the most distinctive tool of human communication, all its variations and components (dialects, jargon, rules, accent, inflections, complexity, expressiveness, meaning, etc.) offer the most direct route toward culturally charged information. In the clinical realm, there is sometimes a special terminology that clinicians must learn to understand in order to make thorough cultural sense out of clinical occurrences.
- *Religion.* Together with language, religion is probably the cultural variable with the deepest, most distinctive roots. It addresses collective concepts about transcending ideas and beliefs about the origin and end of life and nature, perception of virtue and vice, spiritual purity and sinfulness, a set of "explanations about the unexplainable," and management of intriguing or enigmatic situations or entities, including health and illness. It provides abundant information about the relationship between the individual and his or her environment, on the basis of rules and traditions that may be used to justify a variety of both normal and pathological behaviors. It also entails the "mentality" of groups and communities (Alarcón et al., 2002; Group for the Advancement of Psychiatry, 2002; Hutton, 1981).
- *History gathering.* Aspects of this include informants and their style of providing data, areas of emphasis, spontaneous causal explanations, levels of closeness and/or identification with the patient's plight, and so on.
- *Context.* This includes data on the physical and human/personal characteristics of the clinical setting, where and how the clinical history and symptoms evolved, sequence of events, initial reactions or management of the situation, and so forth. Context also covers the very important areas of locus of control and expressed emotions (Sontag, 1978).
- *Meaning.* Any culturally oriented clinical interview must inquire about the personal, unique, and intimate meaning of the experience of illness and its different manifestations, as well as the significance and implications of the illness for life, future, family, and personal fate. Symptoms as physical or subjective occurrences, and the words used to describe them, do have a potent cultural message to be deciphered.
- *Explanatory model(s).* In many cases, the patient and other informants provide their own versions of events, the reasons for the appearance of symptoms, and even opinions about what to do to correct them, on the basis of the causes invoked (which may range from the quite Westernized "chemical imbalance" to explanations based on exotic animistic phenomena or divine punishment) (Alarcón, 1990; Kleinman, 1988; Villaseñor, 2008). If explanatory models are not mentioned spontaneously, the clinician must ask specifically about them, as their cultural content may be decisive. Needless to say, a conflict in explanatory models between the provider and the patient or family should be avoided or negotiated, to prevent diminished rapport, lack of support, or stigmatization.
- *Dimensions of suffering.* These refer not only to areas of physical, emotional, or behavioral pain, but to the deeper and more meaningful dimensions of authentic human suffering (reflected in changes in self-image, self-perception, quality of life, relationship with family, God and religion, values and traditions, fate issues, etc.). A related concept is "social desirability," which is inversely related to individual suffering: The less desirable a given symptom or behavior is within an individual's culture, the more likely he or she is to be stigmatized and ostracized (Weatherhill, 1991).
- *Manuals and instruments.* As part of all clinical evaluations, the cultural assessment should be substantiated by the use of specific measurement tools and instruments. These measures can cover some of the above-described demographic/statistical variables, but should also include such areas as acculturation, stress levels, personality factors, and quality of life. Fortunately, various cultural measurement instruments are now available in today's mental health and clinical settings.
- *Treatment implications.* Like any other type of clinical evaluation, a culturally oriented psychiatric evaluation must include a variety of treatment implications. Information on race, ethnicity, language, religion, and other cultural variables will

lead to appropriate therapeutic indications that take them into account: individual, group, family, and/or couple therapy with cultural emphasis, and even biological modalities if newly developed techniques such as pharmacogenomic testing (with growing recognition of ethnic differences) are applied systematically and judiciously (Alarcón & Mrazek, 2007).

5. *Culture-bound syndromes.* The importance of this group of disorders—reflected in the vast existing literature, and even in the inclusion of an abbreviated list in DSM-IV—makes them deserving of special comment. "Culture-bound syndromes" are defined as unique clinical expressions of cultural idiosyncrasies, with mostly (if not exclusively) cultural determinants, playing a critical role in the context of individual, family, community, and social tapestries. The culture-bound syndromes have had a stormy history among official or institutional nosologists and diagnosticians, while being enthusiastically hailed by culturally oriented clinicians (Hughes, 1996; Tseng, 2006). Although such syndromes were initially restricted to non-Western societies, some authors now claim that they may indeed also exist in the West, particularly in the form of eating disorders or "new" somatoform disorders (Johnson et al., 2004; Littlewood & Lipsedge, 1986). Whether or not this is the case—and whether culture-bound syndromes can be absolutely (and smoothly) incorporated into existing or updated nomenclatures (without falling into what Kleinman [1988] calls "category fallacy"), or whether they still constitute a special class of disorders—they indicate how much work remains to be done in the field.

Overall Structure

Efforts to coordinate the two main diagnostic systems in the world will demand more than simply agreement on terms, categories, and criteria. Such efforts must also extend to the full integration of cultural aspects into the structures of DSM-V and ICD-11. Acceptance and recognition of cultural content across symptoms and syndromes, spectra, or well-defined clinical entities must go beyond the mere declarative nature of political or bureaucratic agreements. The inclusion of cultural elements in psychiatric diagnosis could be all too easily accomplished with the inclusion of a "cultural axis" in any future multiaxial or multilayered diagnosis. Nevertheless, objections to this type of addition are strong, growing, and well justified. The main one is the isolation in which cultural factors would find themselves if restricted to a single axis; the risks of neglect, minimization, or weakness would be quite realistic. Such a restriction would also go against the cardinal ecumenical feature of what is cultural: Everything human is cultural, and any symptom experienced by any human being in any part of the world has a cultural ingredient that is important to consider as part of the "whole" person, not just to note within a separate compartment. Furthermore, the risks of scarcity or excess of cultural data to be considered relevant for diagnosis would be ever-present; boundaries would be difficult to ascertain or reinforce.

A cultural dimension, if well delineated and quantifiable (or measurable), could serve better the stated purposes of recognition, value, and clinical usefulness of a diagnosis. As suggested earlier in this chapter, the interpretive/explanatory, pathogenic/pathoplastic, therapeutic/protective, and service/management roles of culture (Alarcón et al., 1999) can assist in consolidating a cogent diagnostic/nosological role for it. If agreements could be reached about key cultural variables—such as context, meaning, explanatory models, and impact of the morbid condition, as well as language, eventual migration or acculturative history, religion, and management implications—a dimension of cultural influence could then be measured in terms of length and depth, thus providing pragmatic information about treatment emphases and possible outcome predictions (Kraemer, 2007; Lopez, Compton, Grant, & Breiling, 2007). This would allow also an estimate of the cultural "weight" of specific symptoms, syndromes, or diagnostic categories (if a sort of mixed dimensional-categorical model ends up being accepted by the makers of the systems), with many positive implications for future classifications (Alarcón & Foulks, 1995). Although the assertion that "everything is cultural" still retains value, a dimensional approach would provide a realistic assessment of the

relevance of cultural factors in any individual diagnosis.

The suggestion of a cultural dimension does not mean that there are no objections to the dimensional approach. The same objectives can be made to this approach as to the idea of a single cultural axis, although less strongly. Furthermore, some commentators prefer using the term "environmental" to mean "cultural and everything else that is not biological"; however, this runs an even greater risk of neglect or oblivion. Conversely, the dimensional view can exaggerate, or even distort, the impact of cultural factors in psychopathology through excessive scoring or measurements, overemphasis on specific variables, or biasing of clinicians' perceptions. The old labels of "irrelevant," "generic," "too subjective," or "soft" can be applied to cultural research data.

There are good reasons to agree that different clinical conditions vary in terms of their "cultural load." Schizophrenia, bipolar disorders, and autism are justifiably considered the most biologically rooted disorders in modern psychiatry. This does not mean that cultural factors do not play a role in the pathogenesis, symptom expression, or clinical course of these conditions; actually, such factors are crucial in their management, outcome, and ultimate prognosis. On the other hand, it is fair to say at this point that most of the mental diagnostic conditions included in DSM-IV or ICD-10 (and, obviously, in their forthcoming versions) do include significant cultural components in their origins, and therefore must be subjects of culturally aimed diagnostic efforts. Such conditions as somatization disorder, and personality disorders, depression and its variants (e.g., bereavement), some anxiety disorders, and others have already been the topics of studies in different parts of the world; the results are revealing, rich, and varied, going beyond the simplified findings of large epidemiological inquiries (Gaw, 2001; Tseng, 2001).

Whatever the cultural content of DSM-V and ICD-11 turns out to be, it is important to keep in mind that the inclusion of cultural factors does not guarantee their universal acceptance or practical use in clinical work. For both conceptual and pragmatic reasons, it will be important to keep the cultural formulation of DSM-IV in the future editions of the manual, and to include some equivalent in ICD-11. Criticisms that the use of the formulation may result in fragmentation, dichotomization, simplification, stereotyping, and impersonalization have been, in a few cases, well justified. A few studies have attempted to demonstrate the cultural formulation's usefulness and relevance, while standardization, user-friendliness, and enlarged databases have been called for (Group for the Advancement of Psychiatry, 2002; Lewis-Fernandez, 1996; Lewis-Fernandez & Diaz, 2002). The text and content of the formulation could be enhanced, taking into account the rich variety of items examined here and in other publications; a quantitative version could also be devised. Above all, however, concerted efforts to publicize the formulation and to utilize it with all kinds of clinical populations (generating, in turn, concomitant useful research data sets) would be important steps toward guaranteeing a healthy presence of cultural factors in the field of psychiatric diagnosis.

Conclusions

There is ample evidence for a growing understanding and acceptance of the value of culture and cultural factors in the generation of a comprehensive and accurate psychiatric diagnosis. Even biologically oriented clinicians and researchers acknowledge this value and admit the need to add a cultural perspective to the diagnostic process. Indeed, there is a lot of truth in Kirmayer's (2006) assertion that psychiatric diagnosis is in fact a cultural interpretation of a person's (patient's) experience (symptoms). Despite this new level of respectability, however, the inclusion of cultural factors in diagnosis still encounters obstacles in areas of definition and terminology, efforts to operationalize such inclusion, and limited (until recently) coordination of initiatives by responsible organizations. The examination of all the political implications of such coordinated efforts should be followed by well-delineated work, paying attention to pervasive clinical and culturally proper variables and perspectives. Discussions have included (and should continue to include) addressing issues of cultural validity, threshold definitions, compatibility, and a consistent focus on cultural "fitness" of the forthcoming systems (Harper, 2001;

Kirmayer & Minas, 2000; Knapp & Jensen, 2006). In turn, cultural variables should be part of a more dimensional approach to clinical conditions; cultural factors should be measured in an objective manner; and tools such as DSM-IV's cultural formulation should be improved.

Although research in cultural psychiatry requires further and more vigorous development, the current state of knowledge is promising. In the area of psychiatric diagnosis, field studies should go beyond the descriptive or comparative *modus operandi* to utilize cultural approaches that embrace epidemiological and anthropological principles, while creating a descriptive account of the illness experience, its meaning, and the context and cultural determinants of occurrence, course, and outcome (Van Ommeren, 2003)—all of them diagnostic elements of the first order. Through the combined use of clinical, ethnographic, and even experimental studies, cultural case ascertainment and culturally standardized assessment instruments (including adapted psychological tests) would help in dimensionalizing a culturally based diagnosis. The coordinated efforts to make DSM-V and ICD-11 more culturally relevant—substantiated by joint research efforts with adequate cross-cultural methodology—will undoubtedly result in a better diagnostic process, and therefore in more appropriate treatments and solid outcomes for persons with mental illnesses across the world.

References

Alarcón R. D. (1990). *Identidad de la psiquiatría Latinoamericana: Voces y exploraciones en torno a una ciencia solidaria*. México City: Siglo XXI Editores.

Alarcón, R. D. (1998). What cultural psychiatry isn't. *Psychline*, *2*, 27–28.

Alarcón, R. D. (2007). Psiquiatría folklorica, etnopsiquiatría o psiquiatría cultural?: Examen crítico de las perspectivas de C. A. Seguín. *Revista Latinoamericana de Psiquiatria*, *5*, 8–15.

Alarcón, R. D., Bell, C. C., Kirmayer, L. J., Lin, K. M., Ustun, B., & Wisner, K. L. (2002). Beyond the funhouse mirrors: Research agenda on culture and psychiatric diagnosis. In D. J. Kupfer, M. B. First, & D. A. Regier (Eds.), *A research agenda for DSM-V* (pp. 219–289). Washington, DC: American Psychiatric Association.

Alarcón, R. D., & Foulks, E. F. (1995). Personality disorders and culture: Contemporary clinical views (Part B). *Cultural Diversity and Mental Health*, *1*, 79–91.

Alarcón, R. D., & Mrazek, D. A. (2007). Fármacogenomica psiquiátrica: Actualización y perspectivas. In H. Silva (Ed.), *Genética y farmacogenómica en psiquiatría* (pp. 59–79). Santiago de Chile: Ediciones SONEPSYN.

Alarcón, R. D., Westermeyer, J., Foulks, E. F., & Ruiz, P. (1999). Clinical dimensions of contemporary cultural psychiatry. *Journal of Nervous and Mental Disease*, *187*, 465–471.

American Psychiatric Association (APA). (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

American Psychiatric Association (APA). (2008). *Announcement of the establishment of the DSM-V Committee and Work Groups* [Press release)]. Washington, DC: Author.

Baca-Baldomero, E., & Lázaro, J. (Eds.). (2005). *Hechos y valores en psiquiatría*. Madrid: Triacastela.

Bains, J. (2005). Race, culture, and psychiatry: A history of transcultural psychiatry. *History of Psychiatry*, *16*, 139–154.

Bauman, Z. (1998). *Globalization: The human consequences*. New York: Columbia University Press.

Beneduce, R., & Martelli, P. (2005). Politics of healing and politics of culture: Psychiatry, identity, and migration. *Transcultural Psychiatry*, *42*, 367–393.

Berrios, G. E. (1996). *History of mental symptoms*. Cambridge, UK: Cambridge University Press.

Chessick, R. (2003). Psychoanalytic peregrination: VI. The effect on countertransference of the collapse of stabilization. *Journal of the American Academy of Psychoanalysis*, *31*, 541–562.

Delgado, H. (1947). Acerca de la psicopatología general de Jaspers. *Revista de Neuro-Psiquiatría*, *10*, 413–421.

Delgado, H. (1967). *Curso de psiquiatría* (6th ed.). Barcelona: Editorial Científico-Medica.

Eisenberg, L. (2005). Are genes destiny?: Have adenine, cytosine, guanine and thymine replaced Lachesis, Clotho and Atropos as the weavers of our fate? *World Psychiatry*, *4*, 3–8.

Engel, G. L. (1977). The need for a new medical

model: A challenge for biomedicine. *Science*, *196*, 129–132.

Follette, W., & Houts, A. (1996). Models of scientific progress and the role of theory in taxonomy development: A case study of the DSM. *Journal of Consulting and Clinical Psychology*, *64*, 1120–1132.

Gaw, A. C. (2001). *Concise guide to cross-cultural psychiatry*. Washington, DC: American Psychiatric Press.

Ghaemi, S. (2003). *The concepts of psychiatry: A pluralistic approach to the mind of mental illness*. Baltimore: Johns Hopkins University Press.

Group for the Advancement of Psychiatry, Committee on Cultural Psychiatry. (2002). *Cultural assessment in clinical psychiatry*. Washington, DC: American Psychiatric Press.

Guze, S. E. (1992). *Why psychiatry is a branch of medicine*. New York: Oxford University Press.

Halliburton, M. (2004). Finding a fit: Psychiatric pluralism in South India and its implications for WHO studies of mental disorder. *Transcultural Psychiatry*, *41*, 80–98.

Harper, G. (2001). Cultural influences on diagnoses in children and adolescents. *Psychiatric Clinics of North America*, *24*, 711–728.

Hasler, G., Drevets, W. C., Manji, H. K., & Charney, D. S. (2004). Discovering endophenotypes for major depression. *Neuropsychopharmacology*, *27*, 1–17.

Hughes, C. C. (1996). The culture-bound syndromes and psychiatric diagnoses. In J. E. Mezzich, A. Kleinman, H. Fabrega, & D. L. Parron (Eds.), *Culture and psychiatric diagnoses: A DSM-IV perspective* (pp. 289–305). Washington, DC: American Psychiatric Press.

Hughes, C. C. (1998). The glossary of "culture-bound syndromes" in DSM-IV: A critique. *Transcultural Psychiatry*, *35*, 413–421.

Hutton, P. H. (1981). The history of mentalities: The new map of cultural history. *History and Theory, 20*, 237–259.

Jaspers, K. (1968). *General psychopathology*. Chicago: University of Chicago Press. (Original work published 1913)

Johnson, R. L., Saha, S., Arbelaez, J. J., Beach, M. C., & Cooper, L. A. (2004). Racial and ethnic differences in patient perceptions of bias and cultural competence in health care. *Journal of General Internal Medicine*, *19*, 101–110.

Kandel, E. (2006). *In search of memory*. New York: Norton.

Kirmayer, L. J. (2006). Beyond the "new cross-cultural psychiatry": Cultural biology, discursive psychology and the ironies of globalization. *Transcultural Psychiatry*, *43*, 126–144.

Kirmayer, L. J., & Minas, H. (2000). The future of cultural psychiatry: An international perspective. *Canadian Journal of Psychiatry*, *45*, 438–446.

Kirmayer L. J., & Young, A. (1999). Culture and context in the evolutionary concept of mental disorder. *Journal of Abnormal Psychology*, *108*, 446–452.

Kleinman, A. (1988). *Rethinking psychiatry: From cultural category to personal experience*. New York: Free Press.

Kleinman, A. (2008). The normal, the pathological, and the existential [Editorial]. *Comprehensive Psychiatry*, *49*, 111–112.

Knapp, P., & Jensen, P. S. (2006). Recommendations for DSM-V. In P. S. Jensen, P. Knapp, & D. A. Mrazek (Eds.), *Toward a new diagnostic system for child psychopathology: Moving beyond the DSM* (pp. 162–181). New York: Guilford Press.

Kraemer, H. C. (2007). DSM categories and dimensions in clinical and research contexts. *International Journal of Methods in Psychiatric Research*, *16*(Suppl. 1), S8–S15.

Kuh, V., Power, C., Blane, D., & Bartley, M. (1997). Social pathways between childhood and adult health. In D. Kuh & Y. Ben-Shlomo (Eds.), *A life course approach to chronic disease epidemiology* (pp. 169–200). Oxford, UK: Oxford University Press.

Lacroix, M., & Shrad, G. E. (2004). Introduction: Globalization and community mental health. *Canadian Journal of Community Mental Health*, *23*, 5–21.

Leighton, A. H. (1982). *Caring for mentally ill people: Psychological and social barriers in historical context*. Cambridge, UK: Cambridge University Press.

Lewis-Fernandez, R. (1996). Cultural formulation of psychiatric diagnosis. *Culture, Medicine, and Psychiatry*, *20*, 133–144.

Lewis-Fernandez, R., & Diaz, N. (2002). The cultural formulation: A method for assessing cultural factors affecting the clinical encounter. *Psychiatric Quarterly*, *73*, 271–295.

Littlewood, R., & Lipsedge, M. (1986). The "culture bound syndromes" of the dominant culture: Culture, psychopathology and biomedicine. In J. L. Cox (Ed.), *Transcultural psychiatry* (pp. 253–273). London: Croom Helm.

Lopez, M. F., Compton, W. N., Grant, B. F., & Breiling, J. P. (2007). Dimensional approaches in diagnostic classification: A critical appraisal. *International Journal of Methods in Psychiatric Research*, *16*(Suppl. 1), S6–S7.

Lopez-Ibor, J. J., Gaebel, W., Maj, M., & Sartorius, N. (Eds.). (2002). *Psychiatry as a neuroscience*. New York: Wiley.

Mastrogianni, A., & Bhugra, V. (2003). Globalization, cultural psychiatry, and mental distress. *International Journal of Social Psychiatry*, *49*, 163–165.

Phillips, K. A., First, M. B., & Pincus, H. A. (Eds.). (2003). *Advancing DSM: Dilemmas in psychiatric diagnosis*. Washington, DC: American Psychiatric Association.

Richerson, P. J., & Boyd, R. (2005). *Not by genes alone: How culture transformed human evolution*. Chicago: University of Chicago Press.

Rogler, L. H., Malgady, R. G., & Constantino, G. (1987). What do culturally sensitive mental health services mean? *American Psychologist*, *49*, 565–570.

Ruiz, P., Alarcón, R. D., Lolas, F., Lázaro, J., & Baca-Baldomero, E. (2008). *Role of Spanish speaking psychiatry in a globalized world*. Manuscript under revision.

Simmons, A. (2002). *Mondialisation et migration international: Tendencies, interrogations et modeles theoriques*. Montréal: Cahiers Quebecois de Demographie.

Sontag, S. (1978). *Illness as a metaphor*. New York: Farrar, Straus & Giroux.

Spiegelberg, H. (1972). *Phenomenology in psychology and psychiatry: A historical introduction*. Evanston, IL: Northwestern University Press.

Stiglitz, J. (2002). *Globalization and its discontents*. London: Penguin Books.

Tseng, W. S. (2001). *Handbook of clinical psychiatry*. San Diego, CA: Academic Press.

Tseng, W. S. (2006). From peculiar psychiatric disorders through culture-bound syndromes to culture-related specific syndromes. *Transcultural Psychiatry*, *43*, 554–576.

Tseng, W. S. (2007). *Role of the World Association of Cultural Psychiatry*. Paper presented at the inaugural meeting of the World Association of Cultural Psychiatry, Beijing.

Tseng, W. S., & Streltzer, J. (Eds.). (2004). *Cultural competence in clinical psychiatry*. Washington, DC: American Psychiatric Publishing.

Van Ommeren, M. (2003). Validity issues in transcultural epidemiology. *British Journal of Psychiatry*, *182*, 376–378.

Villaseñor, S. J. (2008). *Apuntes para una etnopsiquiatria Méxicana*. Guadalajara, México: Imprenta Universitaria.

Weatherhill, R. (1991). The psychical realities of modern culture. *British Journal of Psychotherapy*, *7*, 268–277.

World Health Organization (WHO). (1973). *The international pilot study of schizophrenia*. Geneva: Author.

World Health Organization Commission on Macroeconomics and Health. (2001). *Macroeconomics and health: Investing in health for economic development*. Geneva: Author.

CHAPTER 5

A Sociocultural Conception of the Borderline Personality Disorder Epidemic

THEODORE MILLON

The interface between psychopathology and sociocultural contexts is extremely complex and variable. Numerous biological, psychological, and cultural factors give shape to the development of personality characteristics and general psychopathology. Symptomatological pictures presented by patients are multidetermined in an intricately interwoven pattern of causality. The ways in which social and cultural influences give rise to the traits and disorders of personality are many, complicated, and divergent.

Sociocultural processes can be sources of psychic stress themselves, as well as giving form and quality to the nature of life experiences. In addition, social influences furnish a framework for how individuals will learn to cope with the nature of their distress. The manner in which clinical symptoms will be interpreted is also a product of social and cultural value systems. No less relevant is the influence of culture in guiding how patients seek assistance therapeutically, as well as the modes of treatment that characterize the orientations of society's healers.

The value systems and nosological schemas generated in Western societies, such as those seen in the ICD-10 (World Health Organization, 1992) and the DSM-IV (American Psychiatric Association, 1994), reflect particular models of thought that may be at variance with the models of numerous cultures and subcultures around the world. More specifically, the Western schemas are oriented specifically to an individual's personal experiences and are largely grounded in a medical model of infectious disease—a model driven by contemporary biomedical technologies and, more particularly, those of pharmacological therapies. Western cultural perspectives contrast with numerous cultural orientations that are centered more on social contexts and relational networks, as well as interpersonal methods of intervention that reflect broad-ranging social health systems. Care must be taken, therefore, not to generalize findings generated in Western concepts and research to those of other cultures (Alarcón, Foulks, & Vakkur, 1998).

The mind is shaped by the institutions, traditions, and values that make up the cultural context of societal living. Methods by which social rules and regulations are transmitted between people and from generation to generation are often emotionally charged and erratic, entailing persuasion, seduction, coercion, deception, and threat. Feelings of anxiety and resentment are generated as a

function of environmental stress, leaving pathological residues that linger and serve to distort future relations. Bear in mind, however, that cultural and social conditions do not directly shape the mind or "cause" disorders; rather, they serve as a context for the more direct and immediate experiences of interpersonal and family life. They not only color but may degrade personal relationships, establishing maladaptive styles of coping and pathogenic models for imitation.

In this chapter, I address in part forces that characterize "society as the patient," as Frank (1948) suggested over six decades ago:

> Instead of thinking in terms of a multiplicity of so-called social problems, each demanding special attention and a different remedy, we can view all of them as different symptoms of the same disease. That would be a real gain even if we cannot entirely agree upon the exact nature of the disease. If, for example, we could regard crime, mental disorders, family disorganization, juvenile delinquency, prostitution and sex offenses, and much that now passes as the result of pathological processes (e.g., gastric ulcer) as evidence, not of individual wickedness, incompetence, perversity or pathology, but as human reactions to cultural disintegration, a forward step would be taken. (p. 42)

In much the same way as the paired strands of the DNA double helix unwind, and each strand then selects environmental nutrients to duplicate its discarded partner, so too does each culture fashion its constituent members to fit an existing template. Those societies whose customs and institutions are fixed and traditional will generally produce a psychically structured and formal citizenry, and those societies whose values and practices are highly fluid and inconsistent are likely to evolve so that their citizens develop deficits in psychic solidity and stability.

Though it would be naïve to state that sociocultural factors are directly responsible for individual pathology, responsible scientists would be lax if they ignored the role of cultural dynamics in the well-being of individuals. Whether or not one is inclined to attribute the increased incidence in youthful depressive disorders in Western countries, for example, to the waxing and waning of population parameters, it is both intuitively and observationally self-evident that sweeping cultural changes can affect innumerable social practices. Among those social practices most evidently affected by cultural dynamics are those of an immediate and personal nature, such as patterns of child nurturing and rearing, marital affiliation, family cohesion, leisure style, entertainment content, diminution of the role of organized religion, and so on.

Numerous demographic, political, and international changes, as well as advances in science and technology in recent decades, have stimulated a growing awareness of the role of sociocultural factors in public health and illness prevention (Brody, 1990; Kleinman, 1980, 1988a, 1988b; Littlewood & Lipsedge, 1987; Mezzich, Kleinman, & Fabrega, 1996). I focus in this chapter, however, on some of the sociocultural elements of experience affecting the lives of those who have been diagnosed as having borderline personality disorder (BPD).

Alternative Views Concerning the BPD Epidemic

Despite its exegetic brilliance, I am concerned that the Talmudic habit of intricate and abstruse argument, most notably within the psychoanalytic community, has drawn us into recondite intellectual territories, leading us to overlook the impact of palpable social forces generative of BPD. Though less labyrinthine and tortuous, the database for biological conjectures favoring constitutional origins is similarly equivocal, if not controvertible. Unfortunately, the riveting effects of internecine struggles between and within competing analytic and biological theories keep us fixated on obscure and largely unfalsifiable etiological hypotheses, precluding thereby serious consideration of sociocultural notions that may possess superior logic and validity. It is toward this latter goal that the present chapter is addressed.

By no means is the role of constitution or early experience to be dismissed. Nevertheless, the logical and evidential base for currently popular hypotheses is briefly examined and left wanting. Supplementary proposals that favor the deductive and probative primacy of social and cultural factors are posited. They point to a wide army of

influences that either set in place or further embed those deficits in psychic cohesion that lie at the heart of the disorder. Specifically, the view has been advanced that our contemporary epidemic of BPD can best be attributed to two broad sociocultural trends that have come to characterize much of Western life this past half century: first, the emergence of social customs that exacerbate rather than remediate early, errant parent–child relationships; and, second, the diminished power of formerly reparative institutions to compensate for these ancient and ubiquitous relationship problems. To raise questions about either the validity or adequacy of one or another theoretical interpretation is not to take issue with all aspects of its formulations; much of what has been proposed concerning the nature and origins of the BPD epidemic has both substantive merit and heuristic value. One may offer a compelling, if not persuasive, alternative and yet be entirely sympathetic to the major tenets of that which has been critically examined. Such is the case in what follows; the alternative proposed is more in the nature of an addendum than a supplantation. Hence I begin the discussion with references to views shared rather than disputed.

Before noting themes in common, I should say a few additional words of précis about the faddistic character of BPD. It is, in my judgment, overly diagnosed, having become a "wastebasket" for many patients who demonstrate the protean constellation of multiple symptoms that characterize the syndrome. Exhibiting almost all of the clinical attributes known to descriptive psychopathology, BPD and related conditions lend themselves to a simplistic if not perverse form of diagnostic logic: That is, patients who display a potpourri of clinical indices, especially where symptomatic relationships are unclear or seem inconsistent, must perforce have BPD.

Having stated my dismay over contemporary diagnostic fashions and fallibilities, let me record my firm belief in both the clinical existence and the significantly increased incidence of BPD and related conditions these past three or four decades. Whether they are voguish or not, I believe that the emergence of these disorders is a "real" phenomenon—albeit, in my view, not for the reasons advanced in contending analytic and biological theories.

Agreement on the Features of BPD

Overdiagnosed and elusive as the syndrome has been—approached from innumerable and diverse analytic perspectives (e.g., Eriksonian, Kernbergian, Mahlerian, Kohutian), as well as clothed in an assortment of novel conceptual terms (identity diffusion, self–object representations, projective identification)—there remain, nevertheless, certain shared observations that demonstrate the continued clinical astuteness and heuristic fertility of the analytic mindset. Whatever doubts one may have with respect to either the logical or methodological merit of conjectures posed by our modem cadre of third-generation analytic thinkers (Eagle, 1984), they deserve more than passing commendation, not only for their willingness to break from earlier and perhaps anachronistic etiological notions, but for their perspicacity in discerning and portraying the key features of a new and major clinical entity. Similarly, contemporary biogenic theorists (e.g., Akiskal & Akiskal, 1992; Klein, 1971) are to be congratulated for the care with which they have adduced empirical data favoring their views. Overlooking for the present seemingly intractable conflicts among and within analytic, biological, and social learning schools of thought (Millon, 1987), let me note what I believe to be the key features of BPD that contemporary theorists of each orientation appear to judge salient and valid (Millon, 1996).

There is a reasonable consensus that pervasive instability and ambivalence intrude constantly into the everyday lives of persons with BPD, resulting in fluctuating attitudes, erratic or uncontrolled emotions, and a general capriciousness and undependability. Such persons are impulsive, unpredictable, and often explosive; it is difficult for others to be comfortable in their presence. Both relatives and acquaintances feel "on edge," waiting for these patients to display a sullen and hurt look or become obstinate and nasty. In being unpredictably contrary, manipulative, and volatile, people with BPD often elicit rejection rather than the support they seek. Displaying marked shifts in mood, they may exhibit extended periods of dejection and apathy, interspersed with spells of anger, anxiety, or excitement.

Dejection, depression, and self-destructive acts are common. These patients' anguish and despair are genuine, but they are also means of expressing hostility, a covert instrumentality to frustrate and retaliate. Angered by the failure of others to be nurturant, persons with BPD employ moods and threats as vehicles to "get back" at them or to "teach them a lesson." By exaggerating their plight and by moping about, these individuals avoid responsibilities and place added burdens on others, causing their families not only to care for them, but to suffer and feel guilt while doing so. In the same way, cold and stubborn silence may function as an instrument of punitive blackmail, a way of threatening others that further trouble is in the offing. Easily nettled, offended by trifles, persons with BPD are readily provoked into being sullen and contrary. They are impatient and irritable, unless things go their way.

Cognitively capricious, these persons may exhibit rapidly changing and often antithetical thoughts concerning themselves and others, as well as the odds and ends of passing events. They voice dismay about the sorry state of their lives, their sadness, their resentments, their "nervousness." Many feel discontented, cheated, and unappreciated; their efforts have been for naught; they have been misunderstood and are disillusioned. The obstructiveness, pessimism, and immaturity that others attribute to them are only reflections, they feel, of their "sensitivity" and the inconsiderateness that others have shown. But here again, ambivalence intrudes; perhaps, they say, their own unworthiness, their own failures, and their own "bad temper" are the causes of their misery and the pain they bring to others.

The affective and interpersonal instability of persons with BPD may be traced in great measure to their defective psychic structures—their failure to develop internal cohesion and hierarchical priorities. Both a source and a consequence of this lack of inner harmony is their uncertain sense of self, the confusions they experience of either an immature, nebulous, or wavering sense of identity. Hence the deeper structural undergirding for intrapsychic regulation and interpersonal processing provides an inadequate scaffolding for both psychic continuity and self-integration. Segmented and fragmented, subjected to the flux of their own contradictory attitudes and enigmatic actions, their very sense of being remains precarious. Their erratic and conflicting inclinations continue as both causes and effects, generating new experiences that feed back and reinforce an already diminished sense of wholeness.

A Supplementary Sociocultural Thesis

Although the logic of biogenic conjectures justifies critical examination, the following comments are limited to the philosophical and empirical grounding of psychoanalytic theories, owing to their far greater currency among contemporary clinicians.

The premise that early experience plays a central role in shaping personality attributes is one shared by analytic and social learning theorists. To say the preceding, however, is not to say that these two groups agree on which specific factors during these developing years are critical in generating particular attributes; nor is it to note any agreement that known formative influences are either necessary or sufficient. Analytic theorists almost invariably direct their etiological attention to the realm of early childhood experience. Unfortunately, they differ vigorously among themselves (e.g., Kernberg, Kohut, Mahler, Masterson, Erikson) as to which aspects of nascent life are crucial to development.

The preceding critique of the epistemic and probative foundations of our discipline's theoretical assertions may seem unduly severe, or perhaps even discourteous to analytic colleagues whose views have been singled out for critical note. Despite appearances, however, I consider myself a loyal if peripheral "follower"—one not only sympathetic to both historic and contemporary psychoanalytic formulations, but one whose own views have been informed by and continue to rest on the foundations they provide. Thus, despite retaining aspects of my earlier conceptions of the "borderline levels" of personality pathology (Millon, 1969, 1981, 1996), I have restructured my ideas significantly in recent years, owing to the thoughtful contributions of numerous contemporary analytic writers.

What follows is not intended, therefore, to supplant the core notions proposed by pres-

ent-day analytic theorists concerning the experiential background of persons with BPD, despite these theorists' own divergences and the unfalsifiability of their propositions. Rather, it should be seen as an addendum—a proposal that societal customs that served in the past to repair disturbances in early parent–child relations have declined in their efficacy, and have been "replaced" over the past several decades with customs that exacerbate these difficulties, contributing thereby to what may be termed our contemporary BPD "epidemic."

Two central questions guide this commentary. First, what are the primary sources of influence that give rise to the symptoms distinguishing BPD—namely, an inability to maintain psychic cohesion in realms of affect, self-image, and interpersonal relationships? Second, which of these sources has had its impact heightened over the past few decades, accounting thereby for the rapid and marked increase in the incidence of the disorder?

The first question is not elaborated here, other than to note that well-reasoned yet contending formulations have been posited by constitutional, analytic, and social learning theorists, and that despite important divergences, there is a modest consensus that biogenic, psychogenic, and sociogenic factors each contribute in relevant ways.

It is the second question that calls for explication. It relates to which of these three etiological factors productive of the diffuse or segmented personality structure characteristic of BPD—constitutional disposition, problematic early nurturing, or contemporary social changes—has shown a substantial shift in recent decades. Is it some unidentified yet fundamental alteration in the intrinsic biological makeup of present-day youngsters? Is it some significant and specifiable change in the ways in which contemporary mothers nurture their infants and rear their toddlers? Or is it traceable to fundamental and rapid changes in Western culture that have generated divisive and discordant life experiences, while reducing the availability of psychically cohering and reparative social customs and institutions?

Despite the fact that tangible evidence favoring one or another of these possibilities is not accessible in the conventional sense of empirical "proof," it is my contention that the third "choice" is probatively the most sustainable and inferentially the most plausible. Toward these ends, two sociocultural trends generative of the segmented psychic structures that typify persons with BPD are elucidated. Although they are interwoven substantively and chronologically, these trends are separated for conceptual and pedagogical purposes. One adds to the severity of psychic dissonance; it appears to have been on the upswing. The other has taken a distinct downturn, and its loss also contributes to diminished psychic cohesion.

Increasingly Divisive Social Customs

We are immersed deeply in both our time and our culture. This immersion obscures our ability to discern many profound changes that may be underway in our society's institutions—changes often generative of unforeseen psychic and social consequences, Tom Wicker, a distinguished columnist for the *New York* Times, portrays sequential effects such as these in the following graphic passage:

> When a solar-powered water pump was provided for a well in India, the village headman took it over and sold the water, until stopped. The new liquid abundance attracted hordes of unwanted nomads. Village boys who had drawn water in buckets had nothing to do, and some became criminals. The gap between rich and poor widened, since the poor had no land to benefit from irrigation. Finally, village women broke the pump, so they could gather again around the well that had been the center of their social lives. (1987, p. 23)

Not all forms of contemporary change can so readily be reversed. "Progress" wrought by modem-day education and technology is too powerful to be turned aside or nullified, much less reversed, despite conservative efforts to revoke or undo their inexorable effects.

Over two decades ago, Klerman (1987), reviewing the increased incidence of depressive disorders among young people, contended that an as-yet-unidentified cohort effect (which he termed "Agent Blue") might be operative. Inclined to ascribe this pathological drift to Easterlin and Crimmins's (1985)

thesis that the baby boom had created a deterioration in economic possibilities among contemporary youth, Klerman noted that this group was raised in relative affluence and is physically the most healthy population in history; nevertheless, they paradoxically were (and are) suffering an epidemic of depressive and other mental disorders.

Whether or not one assigns the increased incidence of youthful depressive disorders to the waxing and waning of population parameters, it is both intuitively and observationally self-evident that sweeping cultural changes can affect innumerable social practices—including those of an immediate and personal nature, such as patterns of child nurturing and rearing, marital affiliation, family cohesion, leisure style, entertainment content, and so on. I turn next to contemporary changes such as these, narrowing the focus to those cultural transitions conducive to the formation of psychic diffusion and division.

Mirrored Social Discordance

It would not be too speculative to assert that the organization, coherence, and stability of a culture's institutions are in great measure reflected in the psychic structure and cohesion of its members. As noted earlier, in a manner analogous to the DNA double helix—in which each paired strand unwinds and selects environmental nutrients to duplicate its jettisoned partner—so too does each culture fashion its constituent members to fit an extant template. In societies whose customs and institutions are fixed and definitive, the psychic composition of its citizenry will likewise be structured; and where a society's values and practices are fluid and inconsistent, so too will its residents evolve deficits in psychic solidity and stability.

This latter, more amorphous cultural state, so characteristic of our modern times, is clearly mirrored in the interpersonal vacillations and affective instabilities that typify BPD. Central to our recent culture have been the increased pace of social change and the growing pervasiveness of ambiguous and discordant customs to which children are expected to subscribe. Under the cumulative impact of rapid industrialization, immigration, urbanization, mobility, technology, and mass communication, there has been a steady erosion of traditional values and standards. Instead of a simple and coherent body of practices and beliefs, children find themselves confronted with constantly shifting styles and increasingly questioned norms whose durability is uncertain and precarious.

No longer do youngsters find the certainties and absolutes that guided earlier generations. The complexity and diversity of everyday experience play havoc with simple, "archaic" beliefs, and render them useless as instruments to deal with contemporary realities. Lacking a coherent view of life, maturing youngsters find themselves groping and bewildered, swinging from one set of principles and models to another, unable to find stability either in their relationships or in the flux of events. At few times in history have so many children faced the tasks of life without the aid of accepted and durable traditions. Not only does the strain of making choices among discordant standards and goals beset them at every turn, but these competing beliefs and divergent demands prevent them from developing either internal stability or external consistency. And no less problematic in generating such disjoined psychic structures is the escalation of emotionally capricious and interpersonally discordant role models.

Schismatic Family Structures

Although transformations in family patterns and relationships have evolved fairly continuously over the past century, the speed and nature of transitions since World War II have been so radical as to break the smooth line of earlier trends. Hence children today typically no longer have a clear sense of either the character or the purpose of their fathers' work activities, much less a detailed image of the concrete actions constituting that work. Beyond the little they know about their fathers' daily routines to model themselves after, mothers of young children have shifted their activities increasingly outside the home, seeking career fulfillments or needing dual incomes to sustain family aspirations. Not only are everyday adult activities no longer available for direct observation and modeling, but traditional gender roles, once distinct and valued, have become blurred and questionable. Today, little that

is rewarded and esteemed by the larger society takes place where children can see and emulate it. What "real" and "important" people do cannot be learned from parents who return from a day's work too preoccupied or too exhausted to share their esoteric activities. Lost, then, are the crystallizing and focusing effects of identifiable and stable role models, which give structure and direction to maturing psychic processes. This loss contributes significantly to the maintenance of the undifferentiated and diffuse personality organization so characteristic of many persons with BPD.

With the growing dissolution of the traditional family structure, there has been a problematic increase in parental separation, divorce, and remarriage. Children subject to persistent parental bickering and family restructuring not only are exposed to changing and destructive models for imitative learning, but develop the internal schisms characteristic of BPD. The stability of life, so necessary for the acquisition of a consistent pattern of feeling and thinking, is shattered when erratic conditions or marked controversies prevail. There may be an ever-present apprehension that a parent will be totally lost through divorce; dissension may also lead to the undermining of one parent by the other, and a nasty and cruel competition for the loyalty and affections of children may ensue. Constantly dragged into the arena of parental schisms, a child not only loses a sense of security and stability, but is subjected to paradoxical behaviors and contradictory role models. Raised in such a setting, the child not only suffers the constant threat of family dissolution, but in addition is often forced to serve as a mediator to moderate conflicts between the parents. Forced to switch sides and divide loyalties, the child cannot be a coherent individual, but must internalize opposing attitudes and emotions to satisfy antagonistic parental desires and expectations. The different roles the child must assume to placate the parents are markedly divergent: As long as the parents remain at odds, the child persists with behaviors, thoughts, and emotions that are intrinsically irreconcilable.

For many children, divorce not only undermines their sense that they can count on things to endure, but it often dislodges formerly secure and crucial internalizations within their psychic selves, upsetting the fusions and integrations that evolved among once-incorporated parental models and standards. Because they are alienated from parental attachments, as well as often disillusioned and cynical, these internalized structures may now be totally jettisoned. Moreover, the confidence that they can depend in the future on a previously internalized belief or precept may now be seriously undermined. Devoid of stabilizing internalizations, such youngsters may come to prefer the attractions of momentary and passing encounters of high salience and affective power. Unable to gauge what to expect from their environment, how can they be sure that things that are true today will be there tomorrow? Have they not experienced capriciousness when things appeared stable? Unlike children who can predict their fate (good, bad, or indifferent), such youngsters are unable to fathom what the future will bring. At any moment, and for no apparent reason, they may receive the kindness and support they crave; at any other moment, and for equally unfathomable reasons, they may be the recipient of hostility and rejection. Having no way of determining which course of action will bring security and stability, such youngsters vacillate—feeling hostility, guilt, compliance, assertion, and so on, and shifting erratically and impulsively from one tentative action to another. Unable to predict whether their parents will be critical or affectionate, they must be ready for hostility when most might expect commendation, or assume humiliation when most would anticipate reward. Because they are eternally "on edge," their emotions build up and become raw to the touch. They are ready to react impulsively and unpredictably at the slightest provocation.

Capricious TV Models

Other "advances" in contemporary society have stamped deep and distinct impressions on children's psyches as well—ones equally effectively loaded, erratic, and contradictory. The rapidly moving, emotionally intense, and interpersonally capricious character of TV role models, displayed in swiftly progressing 30- or 60-minute vignettes that encompass a lifetime, add to the impact of disparate, highly charged, and largely dam-

aging value standards and behavior models. What is incorporated is not only a multiplicity of selves, but an assemblage of unintegrated and discordant roles, displayed indecisively and fitfully, especially among those youngsters bereft of secure moorings and internal gyroscopes. The striking images created by our modern-day flickering parental surrogate have replaced all other sources of cultural guidance for many; by age 18, the typical American child will have spent more time watching TV than in going to school or relating directly to his or her parents.

TV may be nothing but simple pablum for those with comfortably internalized models of real human relationships, but for those who possess a world of diffuse values and standards, or one in which parental precepts and norms have been discarded, the impact of these "substitute" prototypes is especially powerful, even idealized and romanticized. And what these TV characters and story plots present to vulnerable youngsters are the stuff of which successful "life stories" must be composed to capture the attention and hold the fascination of their audiences—violence, danger, agonizing dilemmas, and unpredictability, each expressed and resolved in an hour or less. These are precisely the features of social behavior and emotionality that come to characterize the affective and interpersonal instabilities of persons with BPD.

Mind-Blurring Drugs

Adding to this disorienting and cacophonous melange are aggravations consequent to drug and alcohol involvements. Although youth is a natural period for exploratory behaviors, of which many are both socially adaptive and developmentally constructive, experimental substance use entails high risks and can have severe adverse consequences in both the short and long run. However, from the perspective of youngsters who see little in life that has proven secure or desirable, the risks of using these all-too-accessible substances appear minimal. Although many adolescents may consider their substance use casual and recreational, the psychic effects can be quite hazardous, especially among youth who are already vulnerable. Thus for those prone to BPD, the impact of these substances will only further diminish the clarity and focus of their feeble internalized structures, as well as dissolving whatever purposefulness and aspirations they may have possessed to guide them toward potentially reparative actions. Together, these mind-blurring effects exacerbate already established psychic diffusions.

Decreases in Reparative Social Customs

As noted previously, the task of attaining personal integration is not an easy one in a world of changing events and practices. What is best? What is right? How shall I handle this or think about that? Questions such as these plague growing children at every turn. On top of these everyday perplexities, how can children subjected additionally to parental hostility or indifference acquire guidelines for a well-integrated and socially approved system of beliefs and actions? From what source can a consistent, valued, and effective set of internalized feelings, attitudes, and relationships be consolidated?

The fabric of traditional and organized societies not only comprises standards designed to educate and socialize the young; it also provides "insurance," if you will—backups to compensate for or repair system defects and failures. Extended families, church leaders, schoolteachers, and neighbors provide nurturance and role models through which children experiencing troubling parental relationships can find means of support and affection, enabling them thereby to become receptive to society's established body of norms and values. Youngsters subject to any of the diffusing and divisive forces described previously must find one or another of these culturally sanctioned sources of surrogate modeling and sustenance to give structure and direction to their emerging capacities and impulses. Without such bolstering, maturing potentials are likely to become diffuse and scattered. Without admired and stable roles to emulate, such youngsters are left to their own devices to master the complexities of their varied and changing worlds, to control the intense aggressive and sexual urges that well up within them, to channel their fantasies, and to pursue the goals to which

they may aspire. Many become victims of their own growth, unable to discipline their impulses or find acceptable means for expressing their desires. Scattered and unguided, they are unable to fashion a clear sense of personal identity, a consistent direction for feelings and attitudes, a coherent purpose to existence. They become "other-directed" persons who vacillate at every turn—overly responsive to fleeting stimuli, shifting from one erratic course to another. Ultimately, without the restitutive and remedial power of beneficent parental surrogates, they fail to establish internalized values to anchor themselves and to guide their future.

This aimless floundering and disaffiliated stagnation may be traced in part to the loss in contemporary society of various meliorative and reparative customs and institutions, which once "made sure" that those who had been deprived or abused would have a second chance by being exposed to compensatory sponsors and institutions exhibiting values and purposes around which social life could be focused and oriented. I next turn to these losses.

Decline of Consolidating Institutions

The impact of much of what has been described previously might be substantially lessened if concurrent or subsequent personal encounters and social customs were compensatory or restitutive—that is, if they repaired the intrapsychically destabilizing and destructive effects of problematic experiences. Unfortunately, the converse appears to be the case. Whereas the cultural institutions of most societies have retained practices that furnish reparative stabilizing and cohering experiences, thereby remedying disturbed parent–child relationships, my thesis in this chapter is that the changes of the past several decades have not only fostered an increase in intrapsychic diffusion and splintering, but have also resulted in the discontinuation of psychically restorative institutions and customs, contributing thereby to both the incidence and exacerbation of features that typify BPD pathology. Without the corrective effects of undergirding and focusing social mentors and practices, the diffusing or divisive consequences of unfavorable earlier experience take firm root and unyielding form, displaying their structural weaknesses in clinical signs under the press of even modestly stressful events.

One of the by-products of the rapid expansion of knowledge and education is that many of the traditional institutions of our society—such as religion, which formerly served as a refuge to many, offering "love" for virtuous behavior and caring and thoughtful role models—have lost much of their historic power as a source of nurturance and control in the contemporary Western world. Similarly, and in a more general way, the frequency with which families in our society relocate has caused a wide range of psychically diffusing problems. Families not only leave behind stability, but with each move jettison a network of partially internalized role models and community institutions, such as those furnished in church, school, and friendships. Undone thereby are the psychic structure and cohesion that could have been solidified to give direction and meaning to otherwise disparate elements of existence. Not only do children who move to distant settings feel isolated and lonely in these unfamiliar surroundings, and not only are they deprived of opportunities to develop a consistent foundation of social customs and a coherent sense of self, but what faith they may have had in the merits of holding to a stable set of values and behaviors can only have been discredited.

Disappearance of Nurturing Surrogates

The scattering of the extended family, as well as the rise of single-parent homes and shrinkage in sibling number, adds further to the isolation of families as they migrate in and out of transient communities. Each of these undermines the once-powerful reparative effects of kinship support and caring relationships. Contemporary forms of disaffection and parent–child alienation may differ in their particulars from those of the past. But rejection and estrangement have been and are ubiquitous, as commonplace as rivalry among siblings. In former times when children were subjected to negligence or abuse, they often found familial or neighborly parental surrogates—grandmothers, older sibs, aunts or uncles, or even a kind

childless couple down the street who would, by virtue of their own needs or identifications, nurture or even rear them. Frequently more suitable to parental roles, typically more affectionate and giving as well as less disciplinary and punitive, these healing surrogates have historically served not only to repair the psychic damage of destructive parent–child relationships, but to "fill in" the requisite modeling of social customs and personal values that youngsters so treated are receptive to imitate and internalize.

In the past several decades, however, estranged and denigrated children have much less often found nurturing older sibs, grandparents, or other relatives; nor are the once accessible and nurturing neighbors now available. With increased mobility, kinship separation, single parenting, and reduced sibling numbers, our society has few surrogate parents to pick up the pieces of what real parents may have fragmented and discarded, much less to restore the developmental losses engendered thereby.

Reemergence of Social Anomie

For some, the question is not which of the changing social values they should pursue, but whether there are *any* social values that are worthy of pursuit. Youngsters exposed to poverty and destitution; provided with inadequate schools; living in poor housing set within decaying communities; raised in chaotic and broken homes; deprived of parental models of success and attainment; and immersed in a pervasive atmosphere of hopelessness, futility, and apathy cannot help questioning the validity of the "good society." Children reared in these settings quickly learn that there are few worthy standards to which they can aspire successfully. Whatever efforts they may make to raise themselves above these bleak surroundings run hard against the painful restrictions of poverty, the sense of a meaningless and empty existence, and an indifferent if not actually hostile world. Moreover, and in contrast to earlier generations whose worlds rarely extended beyond the shared confines of ghetto poverty, the disparity between everyday realities and what is seen as so evidently available to others in enticing TV commercials and bounteous shopping malls is not only frustrating, but painfully disillusioning and immobilizing. Why make a pretense of accepting patently "false" values or seeking the unattainable goals of the larger society, when reality undermines every hope, and social existence is so pervasively hypocritical and harsh?

Nihilistic resolutions such as these leave youngsters bereft of a core of inner standards and customs to stabilize and guide their future actions, exposing them to the capricious power of momentary impulse and passing temptation. Beyond being merely "anomic" in Durkheim's sense of lacking socially sanctioned means for achieving culturally encouraged goals, these youngsters have incorporated neither the approved customs and practices nor the institutional aspirations and values of our society. In effect, they are both behaviorally normless and existentially purposeless—features seen in manifest clinical form among individuals with prototypal BPD.

Loss of Compelling Causes

As with the good fortunes that were anticipated upon the arrival of the Indian village's solar-powered water pump, so too have the "blessings" consequent to our modern-day standard of living produced their share of troublesome sequelae. Until a generation or two ago, children had productive, even necessary economic roles to fill within the family. More recently, when the hard work of cultivating the soil or caring for the home was no longer a requisite of daily life, youngsters were encouraged to advance their family's fortunes and status via higher education and professional vocations. Such needed functions and lofty ambitions were not only internalized, but gave focus and direction to a child's life—creating clear priorities among values and aspirations, and bringing disparate potentials into a coherent schema and life philosophy.

Coherent aspirations are no longer commonplace today, especially among the children of the middle classes. In contrast to disadvantaged anomic youngsters, they are no longer "needed" to contribute to their families' economic survival; on the other hand, neither can upwardly mobile educational and economic ambitions lead them to readily

surpass the achievements of already successful parents. In fact, children are seen in many quarters today as economic burdens, not as vehicles to a more secure future or to a more esteemed social status. Parents absorbed in their own lives and careers frequently view children as impediments to their autonomy and narcissistic indulgences.

But even among children who are not overtly alienated or rejected, the psychically cohering and energizing effects of being needed or of fulfilling a worthy family aspiration have been lost in our times. Without genuine obligations and real purposes to create intent and urgency in their psychosocial worlds, such youngsters often remain diffused and undirected. At best, they are encouraged to "find their own thing," to follow their own desires and create their own aims. Unfortunately, freedoms such as these translate for many into freedom to remain in flux, to be drawn to each passing fancy, to act out each passing mood, to view every conviction or ethic as being of equal merit—and ultimately to feel evermore adrift, lost, and empty. Satisfying each momentary wish, consuming pleasures once shrouded in mystery, today's youngsters have nonetheless been deeply deprived not of material wants, but of opportunities to fulfill both the minor daily chores now routinely managed by modern technology and the more distant goals that kept the minds of yesterday's children centered around a value hierarchy and oriented toward ultimate achievements.

Although many of this generation have their bearings in good order, some have submerged themselves in aimless materialism. Others remain adrift in disenchantment and meaninglessness, a state of disaffected malaise. Some have attached themselves to naive causes or cults that ostensibly provide the passion and purpose they crave to give life meaning, but even these solutions too often prove empty, if not fraudulent. In earlier times, the disenchanted and disenfranchised, often dislocated from burdened homes or cast out as unwelcome troublemakers, joined together in active protest and rebellion. Problematic economic, social, and political conditions were shared by many profoundly dissatisfied individuals, who formed philosophical movements such as the German *Sturm und Drang* of the late 18th century or the *Wandervogels* of the late 19th. The 20th century as well has witnessed similar though more benign movements stemming from an antipathy to parental ideals and cultural norms, such as the "beat" and "hippie" generations of the second half of the century.

Children in today's Western societies, however, are "rebels without a cause." Whereas earlier generations of disaffected youth were bound together by their resentments, opposed to economic or political oppression, or motivated by other discernible and worthy common causes that provided both group camaraderie and a path to action, today's middle-class youngsters have no shared causes to bring them together. Materially well nourished and clothed, unconstrained in an open society, they can follow their talents and aspirations freely. The purposelessness and emptiness they experience are essentially internal matters, private rather than collective affairs, with no external agents against whom or which they can join with others to take to the streets. I contend that these rebels without a cause—unable to forgo the material comforts of home and ineffective in externalizing their inner discontents upon the larger scene, yet empty and directionless—comprise a goodly share of today's population with BPD. Were it not for the general political, economic, and social well-being for many until society's very recent downturn, a good number would band together, finding some inspiration or justification to act out in concert.

With advances in modern education, we have seen a marked growth in our populace's psychological-mindedness, sufficient to encourage the parents of youngsters such as those described here to turn to our profession for guidance in solving the perplexing character of their children's emotional and social behaviors (e.g., "I don't understand him; he has everything a young person could want".) What in other times might have taken root as a social movement of disaffected young radicals has taken the form of an epidemic of materially prosperous youth who possess the freedom to pursue abundance and contentment, but who feel isolated, aimless, and empty, and whom we "treat" for a deeply troubling psychological disorder.

Conclusions

The preceding pages have described a number of the elements contributing to the broad mosaic of BPD-disposing influences in our times. Although I have largely bypassed their specifics, there are also salient elements of a biogenic nature in this multifactorial mosaic of determinants. Similarly, and prior critiques notwithstanding, this mosaic should be seen as encompassing the psychogenic role of adverse early nurturing and rearing.

What is troubling to those who seek an "ecumenical" synthesis among rival etiological models is not the observation that some biogenic and analytic authors view this constitutional proclivity or that ordeal of early life as crucial to the development of a particular personality disorder. Rather, it becomes troubling when claimants couple empirically unproven or philosophically untenable assumptions with the assertion that they alone possess the sole means by which such etiological origins can be revealed. Perhaps it is too harsh to draw parallels, but presumptions such as these are not unlike Biblical inerrantists who claim their construals of the Bible to be "divine" interpretations, or conservative jurists who assert their unenlightened views to correspond to the "original intent" of the U.S. Constitution's framers. So too do many of our more self-righteous interpreters practice blindly what some have judged our "hopelessly flawed craft" (Grunbaum, 1984)—one in which we demonstrably can neither agree among ourselves, nor discover either the data or methods by which a coherent synthesis may be fashioned among our myriad conjectures.

Accordingly, I hope the reader will recognize that the sociocultural thesis presented here assumes that individuals who will develop BPD in the future are likely to possess troublesome constitutional proclivities and/or to have been subjected to early and repetitive experiences of a psychically diffusing or divisive nature. By contrast, youngsters endowed with an emotionally sturdy disposition and/or reared in a uniform, dependable, and stable manner are not likely candidates for BPD, whatever their encounters may have been with the social forces described herein.

The present thesis is conceived best, then, as an addendum—one that seeks to describe the final set of elements in the trio of biopsychosocial influences that coalesce to form BPD.

Acknowledgment

Portions of this chapter have been adapted and updated from Millon (1987a). Copyright 1987 by The Guilford Press. Adapted by permission.

References

Akiskal, H. S., & Akiskal, K. (1992). Cyclothymic, hyperthymic and depressive temperaments as subaffective variants of mood disorders. In A. Tasman & M. B. Riba (Eds.), *Annual review of psychiatry* (Vol. 11, pp. 43–62). Washington, DC: American Psychiatric Press.

Alarcón, R. D., Foulks, E. F., & Vakkur, M. (1998). *Personality disorders and culture: Clinical and conceptual interactions*. New York: Wiley.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Brody, E. B. (1990). Editorial: Biomedical science and the changing culture of clinical practice. *Journal of Nervous and Mental Disease*, *178*, 279–281.

Eagle, M. (1984). *Recent developments in psychoanalysis: A critical evaluation*. New York: McGraw-Hill.

Easterlin, R. A., & Crimmins, E. M. (1985). *The fertility revolution*. Chicago: University of Chicago Press.

Frank, L. K. (1948). *Society as the patient: Essays on culture and personality*. New Brunswick, NJ: Rutgers University Press.

Grunbaum, A. (1984). *The foundations of psychoanalysis: A philosophical critique*. Berkeley: University of California Press.

Klein, D. F. (1971). Approaches to measuring the efficacy of drug treatment of personality disorders: An analysis and program. In *Principles and problems in establishing the efficacy of psychotropic agents* (Publication No. 2138, pp. 187–204). Washington, DC: U.S. Department of Health, Education and Welfare, Public Health Service.

Kleinman, A. (1980). *Patients and healers in the context of culture*. Berkeley: University of California Press.

Kleinman, A. (1988a). *The illness narratives:*

Suffering, healing and the human condition. New York: Basic Books.

Kleinman, A. (1988b). *Rethinking psychiatry: From cultural category to personal experience*. New York: Free Press.

Klerman, G. L. (1987, January). *The current age of youthful melancholia: Agent Blue*. Lecture presented to the Royal College of Psychiatrists, London.

Littlewood, R., & Lipsedge, M. (1987). The butterfly and the serpent: Culture, psychopathology and biomedicine. *Culture, Medicine, and Psychiatry*, *11*, 289–336.

Mezzich, J. E., Kleinman, A., & Fabrega, H. (Eds.). (1996). *Culture and psychiatric diagnosis: A DSM-IV perspective*. Washington, DC: American Psychiatric Press.

Millon, T. (1969). *Modern psychopathology*. Philadelphia: Saunders.

Millon, T. (1981). *Disorders of personality: DSM-III, Axis II*. New York: Wiley.

Millon, T. (1987a). On the genesis and prevalence of the borderline personality disorder: A social learning thesis. *Journal of Personality Disorders, 1*, 354–372.

Millon, T. (198b7). Social learning models. In R. Michels (Ed.), *Psychiatry: Personality disorders and the neuroses*. New York: Basic Books.

Millon, T. (with Davis, R. D.). (1996). *Disorders of personality: DSM-IV and beyond* (2nd ed.). New York: Wiley.

Wicker, T. (1987, September 24). The pump on the well. *New York Times*, p. 23.

World Health Organization. (1992). *The ICD-10 classification of mental and behavioural disorders: Clinical descriptions and diagnostic guidelines*. Geneva: Author.

PART II

Conceptual Issues in Classification

CHAPTER 6

Philosophical Issues in the Classification of Psychopathology

PETER ZACHAR
KENNETH S. KENDLER

Numerous approaches to a chapter on philosophical issues in classification are possible. Philosophers of mind might ask whether psychiatric disorders are best considered to be physical or mental entities. Epistemologists could analyze what it means for a diagnosis to be "evidence-based," and what kind of "evidence" counts. Philosophers of science might ask whether psychiatry and clinical psychology should aspire to discover natural and law-governed categories like those used in physics and chemistry. Political and moral philosophers might be interested in exploring how gendered norms are subtly written into some diagnostic categories (e.g., borderline personality disorder).

Like Dewey (1938) and Quine (1969), we construe philosophical inquiry as naturally integrated with, not separate from, science. We do not, therefore, claim some special authority to *inform* readers about what count as the "important" philosophical problems in the classification of psychopathology. Our own list of important issues consists of topics commonly encountered in the scientific literature. This disparate list includes questions about dimensional versus categorical approaches to nosology; problems about clinical significance; a query about the role that pathological processes should play in conceptualizing psychiatric disorders; and the exploration of validity. That these are important problems is a matter of widespread consensus. Our goal is to draw out and examine the underlying philosophical issues that are already there.

The potential advantage of a philosophically oriented approach to the contemporary classification problems of psychopathology is that those problems might be seen somewhat differently than they would be otherwise. A philosophically oriented approach might also make the failings of proposed solutions more evident, so that any solution is less likely to be taken literally. The appropriate philosophical framework could serve as a conceptual resource that functions as an "antidote" to literalism and one of its most important symptoms—the reification of diagnostic constructs. The framework we propose in this chapter has such a goal in mind.

Should Classification Be Dimensional or Categorical?

One of the more visible issues is whether psychiatric disorders should be modeled as categories or dimensions (First et al., 2002; Krueger, Watson, & Barlow, 2005; Regier, 2007). Three alternative perspectives are used to conceptualize the categories versus dimensions problem. Our descriptions are idealized, and a perspective's advocates will have varying degrees of commitment. The first is an "essentialist" perspective; the second is an "empiricist" perspective; and the third is the perspective of "pragmatism." Each of these perspectives has strengths and weaknesses, but we argue that the proponents of the empiricist and pragmatic perspectives are least vulnerable to the charms of diagnostic literalism.

The Essentialist Perspective

"Essentialism" is the view that true classifications optimally depict the inherent structure of the world in itself. How the world is, and not our interests, should guide classification (Kripke, 1980; Putnam, 1975). For example, the periodic table of the elements is often considered to represent a discovery of the inherent, true structure of the world.

Essentialism is often associated with the sorting of classifications into categories such as "natural" versus "artificial/arbitrary" (Ahn, Flanagan, Marsh, & Sanislow, 2006; Haslam, 2000). Scientific classifications are supposed to be natural. One form this perspective takes is to ask whether psychiatric disorders can be naturally segregated into real classes or whether they are really dimensional (Lilienfeld & Waldman, 2004; Livesley, 2003).

Real "classes" are groups for which there are definitive criteria for deciding membership. Examples include *even numbers*, *U.S. Senators*, and *people who are HIV-positive*. Categorical distinctions of this type are binary: One either is or is not a member of a class, and degree of membership is equally shared by all members (i.e., 4 is not more of an even number than 100).

In contrast to a class, a "dimension" refers to a construct of interest that can be measured and on which everyone has a value. Examples of dimensions are height and weight. Dimensions are also "graded," meaning that some people will be assigned lower and others higher values on the dimension. Categorical distinctions such as "tall" and "short" require dividing the dimension into discrete subgroups. From an essentialist perspective, such categorical distinctions are considered to be arbitrary with respect to the dimension itself. In philosophical parlance, they do not "carve nature at its joints."

One of the important virtues of the essentialist perspective is its insistence that we should classify the world based on what it is actually like, and not based on what we want it to be like. For example, if it could be demonstrated that psychiatric disorders really are dimensional rather than categorical, proponents of the essentialist approach would unequivocally state that they *should* be modeled dimensionally—because in accurately reflecting the way the world really is, both the science and the practice of psychiatry/psychology would benefit in the long run.

The Empiricist Perspective

The "empiricist" perspective on the categories versus dimensions issue focuses not on what is really there, but on what claims can be justified by the evidence. As a general rule, scientific empiricists adopt a skeptical stance, holding that our current beliefs and theories could turn out to be mistaken (van Frassen, 1980).

By long-standing tradition, empiricists are suspicious of unobservable (or latent) constructs, such as quarks, dark matter, implicit memory, and neuroticism. They consider unobservable constructs to be tools or *instruments* that can help us predict how the world will appear in the future, but that do not describe its essence. Empiricists claim that these instruments have no reality beyond the observations themselves. They do not believe that the world comes to us prepackaged in neat categories, but neither would they license the inferential leap from "Dimensional models fit the data better" to "The world is really dimensional." As far as they are concerned, all that can be legitimately said is that "Dimensional models fit the data better."

They also might add, " . . . fit the data better, considering what measurement instruments are currently available." From this skeptical perspective, empiricists believe that categorical and dimensional classifications each have advantages and disadvantages. They would be reluctant to endorse metaphysical claims about what is really there.

Empiricists espouse "antirealism." They argue that theoretical constructs such as neuroticism typically come to be defined with respect to other theoretical constructs (such as temperament, schema, and identity), and therefore inevitably become increasingly distant from observation. As this process of theoretical elaboration continues, metaphysical attributions about their "reality" become increasingly tenuous. This is as true for the five-factor model of personality as it is for the Freudian metapsychology. As a result, empiricists tend to be suspicious of the metaphor of carving nature at its joints, especially if it is used to support an essentialist perspective.

The categorical versus dimensional issue is an empirical question to the extent that theories about structure can be tested. Various mathematical models have been developed to test whether psychiatric disorders can be segregated into classes or dimensions, with factor analysis, latent-class analysis, and taxometrics being the best known (Goldberg & Velicer, 2006; Haslam & Williams, 2006; Markon & Krueger, 2006). Because the conditions listed in psychiatric diagnostic manuals represent a heterogeneous collection and do not form a single, homogeneous kind, it should not be surprising to find that the results of the various statistical methodologies vary (Haslam, 2007).

There is good evidence that major depressive disorder (MDD), generalized anxiety disorder (GAD), and the personality disorders have a dimensional structure (Krueger, 1999; Krueger, Caspi, Moffitt, & Silva, 1998; Widiger & Samuel, 2005). One can also find subtypes within dimensions (such as melancholia within depression) and dimensional structures within categories (such as the positive, disorganized, and negative symptom dimensions of schizophrenia). Categories can be nested in dimensions, and vice versa. Being concerned with all the evidence rather than with preferred models of reality, empiricism is consistent with the suggestion that diagnostic manuals should utilize both categorical and dimensional perspectives where appropriate, although how to integrate the two is a matter of some debate (Helzer, Kraemer, & Krueger, 2006; Kessler, 2002).

The Perspective of Pragmatism

A group of thinkers referred to as "pragmatists" also emphasize the instrumental or practical value of classifications. They contend that classifications should be chosen with respect to their ability to help us achieve our disciplinary goals (Brendel, 2006; James, 1907, 1909/1975). Pragmatists claim that the phenomena we classify are inherently complicated and that rich, and that any classification inevitably involves highlighting some features and deemphasizing others (Goodman, 1978). As a result, they are also suspicious of talk about carving nature at its joints (Zachar, 2006; Zachar & Bartlett, 2001).

An important philosophical difference between empiricists and pragmatists is that empiricists tend to be antirealists about theoretical constructs, whereas pragmatists need not be. For pragmatists, metaphysical inferences about "reality" are another set of philosophical tools, and it is sometimes informative to distinguish between "real" and "not real" (Dewey, 1929/1958; Hacking, 1999). For example, in biology it has not been possible to discover a single, optimal model of what counts as a species. There exist several species concepts, such as the "phenetic species" concept, the "biological species" concept, and the "phylogenetic species" concept (Mayr, 1988, 1991; Mishler & Brandon, 1987; Ridley, 1986). No single species concept works for all classificatory purposes. The phenetic species model can occasionally contradict evolutionary history; the biological species concept does not apply to many plant species; and the phylogenetic species concept classifies only branching, not divergence. This complication inclines empiricists to say that species categories are not real, while those with pragmatist leanings are willing to say that species are real, but there are multiple ways to define a good species (Ereshefsky, 1992; Mayr, 1982).

How does the categories versus dimensions issue look from the standpoint of pragmatists? To repeat, essentialist-leaning thinkers are willing to claim that psychiatric disorders really are dimensional, not categorical (or vice versa), whereas empiricists would claim only that dimensions fit the current data better (or vice versa). Pragmatists tend to be "pluralists." Pluralism is the belief that no single classification system can answer all the classificatory goals and purposes we adopt; therefore, different goals may require different classifications, each of which can have validity. As noted elsewhere (Zachar & Kendler, 2007), relevant goals for a classification system include selecting treatment, increasing etiological homogeneity, maximizing true positives and true negatives, being measurable, being consistent with genetics and physiology, being culturally congruent, and being clinically informative. No single kind (or dimension) is likely to achieve all the valid goals we can articulate.

For example, if we want to find out how many people in a country were diagnosed with schizophrenia in a single year or to learn about the age of onset and course of schizophrenia across different countries, some kind of a categorical model with inclusion and exclusion criteria will be important. If, on the other hand, we want to understand the relationship between level of disorganization and social functioning in the past 6 months, we will want to measure the full range of each variable (from low to high).

Pragmatists therefore ask what purposes are being served with either dimensional or categorical approaches to classification. Because they are more or less appropriate to specific kinds of goals, choosing dimensions or categories is not only about describing what is really there. In what follows, we explore some of these diverse goals.

Dimensions and Categories in Research

Many psychologists, especially those with a psychometric focus, advocate dimensional models as a function of being trained in correlational research designs. In correlational statistics, taking a continuous variable such as weight measured in kilograms, and transforming it into a dichotomous variable such as "light versus heavy," represents an important loss of information. The same is true for dichotomizing mood into "depressed versus not depressed." Classifying all the numerical information is a statistical virtue of dimensional models.

In contrast, those with backgrounds in epidemiology tend to have a greater appreciation of the virtues of categorical analyses. In practical settings, it may be more clinically informative to know that the Rorschach Perceptual Thinking Index (PTI) can accurately detect 85% of patients who are diagnosed with a psychotic disorder than to know that scores on the PTI explain 41% of the variance in psychotic symptoms (Dao, Prevatt, & Horne, 2007). Studying deaths from a disease, premorbid status, ages of onset, and number of recurrences requires categorization. Coding occurrences in terms of 1-month and lifetime prevalence is also categorical. Such concepts as "caseness," "sensitivity," "positive predictive value," and "base rate" are geared toward categorically defined outcomes. Needless to say, if true discontinuities exist, categorical approaches have even greater virtues.

Dimensions and Categories in Clinical Practice

Dimensions can also serve purposes more specific to practitioners. For example, dimensional models such as those proposed by Simms and Clark (2006) or Livesley (2006) should more comprehensively model the phenotypic space of personality disorders than do the current ICD and DSM categories. There will always be clinically important data not accounted for in the classification system, but the overreliance on the "not otherwise specified" diagnosis for Axis II conditions suggests that too many data are not being modeled. A more comprehensive model of the domain offers the possibility of identifying new targets for intervention, or distinguishing between those parts of the phenomena that are amenable to change and those that are not.

What purposes might categories serve for practitioners? For one, decisions about whether or not to treat are usually categorical. Such binary decisions will not be eliminated in a dimensional system. In DSM and ICD, describing conditions and stating the need for treatment is lumped into a single act of *diagnosis*. Dimensional models would

disentangle these descriptive and prescriptive tasks.

Another purpose of categories is to group together cases that share something in common. Groups of cases of this sort are called "kinds." In DSM and ICD, MDD, schizophrenia, and anorexia nervosa are kinds. Psychiatrists and clinical psychologists learn more about MDD, schizophrenia, and anorexia nervosa by studying what people with these conditions have in common. In theory, the same strategy is possible with groups sharing the same dimensional profile (e.g., the group that is high on narcissism and callousness, low on diffidence and compulsivity). The purpose of grouping cases is to make generalizations about group members, but for pragmatists there is nothing sacred about a grouping. If groups can be combined or decomposed in such a way as to enable new and better generalizations, then they should.

An important question about kinds is this: How good are the generalizations? With very homogeneous groups, such as "copper things," we can make robust generalizations (e.g., "All copper conducts electricity"). With very heterogeneous groups, such as "white things," few or no useful generalizations can be made. Groups of patients with similar profiles of psychiatric symptoms are neither as homogeneous as copper nor as heterogeneous as white things. The generalizations are good to the extent that they help us achieve a variety of disciplinary goals.

We conclude this section on dimensional versus categorical approaches by observing that the starkest choice for classifiers is between the realism of the essentialists and the antirealism of the empiricists. As noted, empiricists are no friends to the view that the world comes preorganized into a set of real categories waiting to be discovered, but neither do they seek to discover the true list of dimensions that are really out there. Among the various available perspectives, advocates of empiricism are the least vulnerable to the problems of reification.

Along with the pragmatists, empiricists caution against overexuberance regarding the nosological momentum that dimensional models clearly have. Like anything else, once a new conceptual strategy is institutionalized, it may expand the scope of psychiatric nosology, but then itself may become another barrier to progress because there is a tendency to take it too literally. The same kind of reification of diagnostic categories that occurred after the publication of DSM-III would be likely to occur with respect to any "official" dimensions included in DSM-V or ICD-11. Advocates of a more essentialist approach to dimensions, intentionally or not, subtly enable this unfortunate outcome.

What Counts as Clinically Significant?

An assumption of essentialism is that mental disorders are out there *in rerum natura*, and we just have to work hard to discover them. Diagnostic literalism signifies great respect for the authority of diagnostic manuals. This may refer to either a current manual or a future ideal manual. Attributing extensive authority to a current manual is associated with a tendency to believe that the inherent structure has been successfully classified (Kendler & Zachar, 2008). On the flip side of the same coin, evidence that a classification is flawed will incline others to say that the classification is scientifically primitive and needs to be eliminated in favor of a more "optimal" approach.

We agree that if they are appropriately classified, disorders are out there. However, we also believe that developing a good classification system requires more than discovery of facts; it also requires that we make decisions based partly on goals and values. Values can be defended, but are not easily classified as either true or false. The authority that accrues to a classification manual regarding issues of value is not the same as the authority that accrues to it with respect to statements of fact.

In this section, we argue that considerations of clinical significance highlight the role played by values in the classification of psychopathology. It turns out that very few thinkers claim that psychiatric nosology should be purged of value judgments. In agreement with the mainstream philosophical opinion on this issue, we claim that rational discussion and deliberation about values is not only possible; in some cases, it should be articulated more explicitly than it has been in the past.

The Role of Evaluation

"Values" refer to judgments of things as good or bad, beneficial or harmful. When the word "values" is used, people often think of moral values such as honesty, fairness, and concern for others, or loaded terms such as "family values," but values are not limited to the domain of the moral. Reliable cues to value terms include use of the words "should" and "ought." There are also political values, such as *habeus corpus* and freedom of speech. Objectivity, validity, and peer review are scientific values. Training in the mental health disciplines partly involves teaching students, interns, and residents what to value and to disvalue. For example, they learn to recognize compulsivity, emotional lability, and inflexibility as negatively valued (pathological) conditions.

Bentall's (1992) proposal to classify happiness as a mental disorder offers an amusing introduction to the role of values in classification. Bentall notes that happiness is a coherent syndrome with behavioral, affective, and cognitive components. As proponents of depressive realism might contend, happiness is also associated with impaired reality testing (Alloy, Albright, Abramson & Dykman, 1990). Bentall states that there is good anatomical and neurochemical evidence that happiness is biologically based, and may even have a genetic component. From an epidemiological perspective, persistent happiness is statistically deviant. It also has a higher prevalence in people of higher socioeconomic status (SES), suggesting that the high-SES population is more exposed to the "risk factors" for happiness. Furthermore, happiness seems to be better modeled as a dimensional construct, perhaps a subthreshold version of hypomania.

So why is happiness not a mental disorder? According to Bentall (1992), it is not a disorder because it is not negatively valued. There are two ways to be negatively valued. First, happiness is not a "negatively valued experience," whereas panic attacks are. Second, happiness is not negatively valued by being an "object of therapeutic concern." The two kinds of negative evaluations do not always coincide. Grief is a negatively valued experience, but it is not usually an object of therapeutic concern. Hypomania may not be a negatively valued experience, but it is typically an object of therapeutic concern.

The question is this: Should such values considerations bear on the objective facts of the matter? Bentall's conclusion is that yes, they should. In his own words, "only a psychopathology that openly declares the relevance of values to classification could persist in excluding happiness from the psychiatric disorders" (1992, p. 97).

A synonym for "object of therapeutic concern" is "clinical significance." About half of the disorders in DSM-IV require that there be clinically significant *impairment* or *distress* in social, occupational, or other important areas of functioning. The clinical significance criterion (CSC) was introduced because finding high prevalence rates in epidemiological studies that used strict translations of DSM criteria raised fears of a false-positive problem (Regier, 2007). A concern about the legitimacy of such disorders as social phobia and oppositional defiant disorder was also an issue (Spitzer & Wakefield, 1999). The CSC also allows professionals to attribute mental disorder status to conditions that do not reach diagnostic thresholds (i.e., to correct for false negatives). In many cases, the CSC is a strategy for resolving the boundary problems that occur at diagnostic thresholds.

Impairment, Distress, and Value

By what standards does one decide whether impaired social or occupational functioning is present? Is this a purely factual matter? In some cases, there is the observation of "decline in functioning," but how much decline counts as clinically significant? A related concept is "loss of control." The change in functioning is factual, but assessing clinically significant change in functioning requires making evaluative distinctions among "optimal," "normal," "compromised," and "impaired." Such evaluations will vary across individual patients, developmental stages, social roles, cultures, and historical epochs.

For example, is a professional baseball player in his prime who successfully competes but is no longer elected to the All-Star team significantly impaired because he has a level of drug and alcohol use that interferes with his training? What if the substance use

interferes with his friendships and parenting as well—even though he still has good friends and is a better parent than many? Even with something as observable as decline in functioning, to identify a disorder there must be a value judgment of clinically significant impairment (or inability to do what one normally should be able to do).

To some extent, clinical significance is a categorical judgment (present or absent); however, it is not only a matter of observing present-at-hand impairment because, according to DSM-IV, impairment need not be actually present. The manual allows the diagnosis of "threat to health" conditions, which are mental disorders that are analogous to hypertension. One could examine the baseball player, decide that there is a *potential impairment* if he continues his current level of substance use, and therefore diagnose a disorder. With potential impairment, a prediction is being made that "compromised" functioning could become "impaired," leading to the additional value judgment that preventative steps would be beneficial. Impairment is clearly a value-laden concept.

Next, we turn to distress. Distress or unpleasantness can be considered inherently negative. Even so, deciding whether distress is clinically significant distress is not a purely factual matter. Most of the heat generated by debates about medicalizing subthreshold conditions revolves around disagreements about when distress should be an object of therapeutic concern. Some mental health professionals would claim that the distress associated with active psychotherapy or the normal distress of loss is potentially healthy rather than pathological, even if it is intense (Horwitz & Wakefield, 2007). What if a person in grief were to feel better after being prescribed an antidepressant or an anxiolytic? Some suggest that an improvement in functioning does justify writing a prescription because a good deal of misery could be averted if subthreshold conditions were taken more seriously (Kendler, 2008b; Kessler et al., 2003; Pincus, McQueen, & Elinson, 2003).

Making evaluative distinctions among "impaired," "compromised," and "normal" has a palatable degree of ambiguity. The ambiguity about norms specifying what *should* and *should not* be suggests that the official listing of psychiatric disorders may be subject to modification and redecision over longer periods of time. The declassification of homosexuality as a mental disorder is an example of this process.

One of the problems with reification is that it takes complicated decisions about what conditions count as psychiatric disorders and translates those decisions into official lists that people take to be *only* matters of scientific fact. We suggest that accepting that evaluative complexities are inherent to good psychiatric classification is a more "realistic" strategy than seeking to discover a purely natural classification system.

Values, Psychiatric Classification, and Rational Deliberation

Thomas Szasz and his followers are generally considered to believe that values should play no role in demarcating true medical conditions. Szaszians tend to be out of the mainstream on this issue, however. Wakefield (1992, 1999a), Sadler (2005), and Fulford (1989) all argue that values are inevitable in nosology. As we discuss in the next section, Wakefield believes that classifying valid psychiatric disorders requires identifying the potential presence of an objective dysfunction and making value judgments regarding harm. In contrast, Sadler and Fulford do not favor a strict fact–value separation. They see values as fully penetrating psychiatric nosology, even where Wakefield sees facts. One of Sadler's and Fulford's main points is that that professionals can come to rational agreement on which set of values should guide both the science and practice of psychiatry/psychology.

In an earlier article, we identified "objectivism versus evaluativism" as one of the important philosophical issues inherent to psychiatric classification (Zachar & Kendler, 2007). We stated that a condition such as schizophrenia might be considered objective in the sense that describing what is broken (see below) could be considered more of a factual matter, but we also noted that its "objectivity" rests on the fact that there is widespread agreement about the relevant values issues. For example, hallucinations and delusions tend to violate epistemic norms. When there is intersubjective consen-

sus about "normality," medical evaluations can gain a degree of objectivity. Examples of the "minimally evaluative" include forestalling death when there is still potential quality of life and reducing intense physical pain.

The relevant value judgments needed for a psychiatric nosology will have varying degrees of consensus. There will be more concurrence with psychotic disorders, but less with personality disorders, sexual dysfunctions, and substance use disorders. Anxiety and depressive conditions will lie somewhere in between. Interestingly, although eating disorders are associated with death and serious physical deterioration and can be as medically relevant as any psychiatric disorder, members of the "pro-ana" movement assiduously define anorexia nervosa as a lifestyle choice and not a disorder (Udovitch, 2002). Dimensional models, which lack boundaries between normal and abnormal, are least able to avoid the value-laden problem of assessing clinical significance.

We are tempted to suggest that the more disagreement that is likely to exist about particular value judgments ("shoulds" and "oughts"), the more explicit the DSM and ICD committees should be in articulating the justifications for making them. At the same time, we urge caution. To give value judgments regarding impairment and distress a more explicit presence in DSM and ICD would increase the probability of subjecting those values to reification.

What Role Should Pathological Processes Play in Understanding Psychiatric Disorders?

In this section, we argue that psychiatrists and psychologists should seek to develop models of psychiatric disorders that help explain why these conditions come about in terms of underlying pathological processes. These processes can be more psychological or more biological—or, ideally, both. One of our main claims is that discovering underlying pathological processes is not the same as discovering what disorders are out there.

Natural Dysfunctions

Jerome Wakefield (1999a, 1999b) claims that a legitimate psychiatric disorder has two essential aspects: (1) the presence of an objective dysfunction, and (2) the evaluation that the dysfunction is harmful (impaired/maladaptive). Wakefield claims that a dysfunction is present whenever something is broken. "Broken" in this context means that part of an organism is not able to function as it was designed to function in evolution. For example, eukaryote cells were designed during evolution to use adenosine triphosphate (ATP) as a source of energy, and furthermore to obtain ATP from a type of prokaryote organism called mitochondria. Lack of mitochondria is a fatal dysfunction.

In order to conceptualize dysfunction, Wakefield and others have reintroduced the Aristotelian idea of natural function, which in the post-Darwin world now has a historical–evolutionary interpretation. "Natural function" refers to how the part or process in question contributed to a species's survival, and therefore also explains why that part/process was selected during evolution. For example, it is likely that detecting light increased one of our ancestors' ability to survive and reproduce, which in turn established a selection pressure for light detection that eventually resulted in eyes.

The important point is that if this historical story is true, then it is a matter of *fact* that eyes evolved because seeing conferred an adaptive advantage. Seeing counts as an eye's natural function. Whether or not an eye performs this natural function is also a factual matter. According to Wakefield, value judgments of the type "An eye should be able to see" can potentially be translated into factual statements about evolutionary history. As matters of fact and not matters of value, dysfunctions are supposedly things we can discover.

An advantage of Wakefield's harmful-dysfunction model is that it captures our common-sense notion of a disorder (i.e., that the attribution of a disorder means that something is broken and needs to be fixed). Dysfunctions can pertain not only to organs and organ systems, but to more abstract processes such as psychological functions (e.g., attention and emotional regulation).

The problem with the harmful-dysfunction model is that it is not amenable to empirical confirmation. In contrast to Wakefield, we claim that underlying "pathological" processes are discoverable, but attributions

of dysfunction in practice have an intuitive element. Nominating natural functions requires speculation that goes far beyond the available evidence because we cannot say for sure what selection pressures existed in human evolutionary history or what the targets of selection were. Other processes in addition to natural selection, such as genetic drift and molecular drive, may also have played roles in evolution. That is, not everything that has evolved had to have a natural function.

Let us explore this problem further by examining reading disorder. What is broken in an inability to read despite having a high level of intelligence and an appropriate education. Reading is not a natural function because the cognitive mechanisms used in reading were no more designed by natural selection for reading than three-dimensional visual–spatial abilities were designed for neurosurgery. Hunters and gatherers did not need to read.

Wakefield (1999a) claims that although reading is not a natural function, brains that are functioning as designed can typically read. Ability to read is a beneficial side effect of one or more other natural functions. Wakefield may be correct. It is, however, just speculation. No discovery of fact has been made.

It is possible that in a future scientifically advanced society, not being able to master calculus will be maladaptive. The inference that a normally functioning brain should be able to learn calculus will be likely to occur, and understandably so, but this inference will not occur independently of the perception of harm/impairment. Analogously, given its importance in modern society, and the very high base rate of people who can read, most will agree that being unable to read is an impairment. As with calculus, the inference that something is broken or not functioning properly may depend on a prior value judgment—in this case, that people *should* be able to read.

Here is the important point. If cognitive psychologists were to identify some underlying attentional mechanisms that helps explain reading difficulties, what would make those mechanisms objectively dysfunctional in Wakefield's sense, rather than normal variations in design that have recently become harmful? Looking at a targeted individual difference and claiming that it is a design failure rather than a maladaptive variation in design does not constitute a simple task.

Again, homosexuality provides a good example. Would a scientifically correct model of the biological genesis of an exclusively homosexual orientation describe a dysfunction or a natural variation? We suggest that one's intuitions about this issue will be correlated with one's previous value judgments about how sexuality *should* develop.

It is certainly true that some psychiatric disorders are objective dysfunctions that are harmful, especially when there is evidence of trauma or of a developmentally unexpected decline in functioning from a previous level of adaptation. Broken bones, autism, and disorganized schizophrenia are good examples. Impulsivity and executive function deficits contingent on a traumatic brain injury are also arguably objective dysfunctions. Other disorders, however, might be harmful variations. Narcissistic and obsessive–compulsive personality disorders may represent normal variations that are first judged to be maladaptive, and subsequently conceptualized as being broken.

As Wakefield (2006) has noted, one of the ways a culture works is to make its own values seem natural. For these reasons, we claim that nosologists should be cautious in speculating about what counts as an unnatural function (i.e., a design failure). Paul Meehl (1993) perceptively wrote that scientists should adopt the ethic of trying not to fool themselves and not to fool others. The high risk of fooling ourselves while speculating about what counts as unnatural is one of the reasons for being cautious about the medicalization of both subthreshold Axis I conditions and individual differences in personality. Few are more cautious in this respect than Wakefield.

Dysfunctions and Causal Roles

Let us assume for the moment that Wakefield's critics are correct regarding the difficulty or even impossibility of discovering natural functions and dysfunctions. Even if they are correct, we suggest that one should not conclude that a concept of dysfunction has no important role to play in psychopathology, even if it does not conform to Wakefield's stipulations.

In agreement with Ken Schaffner (1993), we claim that the concept of function does not have to be historical–evolutionary. Rather, it can refer to whatever role a particular part or process plays in the overall functioning of the organism. For example, a function of the heart is to pump blood; whether pumping blood really is the natural function is irrelevant. The same is true for the cognitive mechanisms that enable reading (or calculus). If reading is considered to be an important ability, whatever parts or processes can be used to explain failure to read can be identified as dysfunctional with respect to reading.

In concluding this section, we assert that the labeling of underlying mechanisms as dysfunctional is sometimes dependent on having already made a disorder attribution. Although the mechanisms are potentially objectively present, a complicated and shifting set of indicators have to be consulted in nominating disorders, and different indicators may be more or less relevant depending on the disorder in question.

The decline in functioning indicator is relevant in autism and schizophrenia, but not in attention-deficit/hyperactivity disorder (ADHD). It has also recently been claimed that liberalism is a mental disorder—a claim that some people take seriously (Rossiter, 2006; Savage, 2005). Similar claims have been made about racism (Bell, 2004). Justifying ADHD's status as a psychiatric disorder, while rejecting that of liberalism and racism, is a worthy conceptual problem—one that requires careful thinking. Discovery of fact is crucial, but such problems cannot be reduced to the kind of straightforward empirical questions favored by those inclined toward diagnostic literalism.

What Is Validity?

Rather than conceptualizing validity as a single quality, we advocate "validity pluralism." It is not just a question of whether a diagnosis is valid *per se*, but what kind of validity it has and how good the validity is. We review four perspectives on validity. Although the perspectives are distinguishable, many psychiatrists and psychologists will endorse them simultaneously. The four perspectives are "entity-based approaches," "information-based approaches," "construct validation," and "explanatory validity."

Entity-Based Approaches

It is well known in the classification literature that beginning with DSM-III, nosologists became concerned with reliability, yet paid limited attention to validity. But what is validity? Usually when mental health professionals ask, "Is schizophrenia a valid disorder?", they mean either "Is it a legitimate mental illness?" or "Is it really a disorder?" Some believe that legitimate mental illnesses are diseases (Kendell, 1975; Robins & Guze, 1970), whereas others claim that they are better considered disorders than diseases (Wakefield, 2000).

Kendell and Jablensky (2003) define valid psychiatric diagnoses as those for which natural boundaries between cases and noncases can be established. They state that valid disorders should be qualitatively different from normality and also should not overlap with other disorders. According to Kendell and Jablensky, valid diagnoses will eventually be shown to possess a combination of features that exist only when the disorder is present.

In highlighting the importance of discreteness, Kendell and Jablensky advocate the "disease realism" approach to validity. Its most famous proponents, Eli Robins and Samuel Guze (1970), proposed five criteria for confirming the presence of a disease, which they termed "validity indicators." These criteria are clinical description, laboratory tests, differential diagnosis, follow-up studies, and family studies. Later, Kendler (1980) expanded the number of potential validators and added a temporal dimension, distinguishing among antecedent, concurrent, and predictive validators.

Disease realists believe that mental illnesses have as-yet-undiscovered pathological processes. In the absence of a confirmed etiology, the goal is to look for integrated syndromes and fill in the etiologies as they are uncovered They astutely point out that one does not have to know the etiology of a mental illness for it to be valid, any more than 18th-century physicians had to know the etiology of tuberculosis for it to be valid. In both cases, diseases can be inferred if the appropriate indicators (validators) are present. To this view, Kendell and Jablensky (2003)

have added the validator of a qualitative difference between normal and abnormal.

Arguments that involutional melancholia is not a valid syndrome exemplify the disease realism approach. Involutional melancholia was a DSM-II syndrome that referred to first-episode psychotic depressions among women. It included an agitated presentation, as well as symptoms that were not supposed to be reactions to stress, but were contingent on menopause (i.e., the involutional period). In the 1970s, it was discovered that a large number of the identified cases did have precipitants and that an agitated presentation was related to past histories of depression. Furthermore, rates of depression for women are not higher during menopause (Becker et al., 2001; Berrios, 1991; Weissman, 1979). The obvious conclusion is that the syndrome referred to by the construct of involutional melancholia is not really there.

An important problem for entity-based approaches is that of systematics. In biology, "systematics" is the study of the relationship between species. As defined by Zachar (2008), "psychiatric systematics" studies what relationships, if any, exist between various disorders. A more absolute form of validity would ask whether a disorder is a coherent entity, but it is also important to understand a disorder relative to others disorders. For example, are MDD and GAD two disorders or one? If they are two overlapping disorders, how can they best be differentiated? If MDD segregates with GAD, then what is its relationship to bipolar disorder? Is there also a relationship between GAD and bipolar disorder? How should nosologists respond if the validators offer conflicting answers to such questions? The issues of overlap, differentiation, and hierarchical structure are complicated aspects of classification that deserve more empirical and conceptual attention.

Information-Based Approaches

Favoring a more data-based, quantitative conceptualization, proponents of empiricism reject the metaphysical predilections of disease realists. For empiricists, validity refers not to whether a disease is really there, but to what kind of inferences one legitimately can make about a patient on the basis of the diagnosis. Whereas for disease realists validity is a property that diagnoses have or do not have, for empiricists inferences, not diagnoses, are validated.

If someone is diagnosed with MDD, "validity" refers to how much evidence exists for the inferences that mental health professionals can make about etiology, treatment, and outcome. Inferential power will vary depending on a host of factors. A diagnosis of MDD would suggest some inferences about time course, but if the patient is also experiencing a third episode and there is a family history, different inferences can be legitimately made. In contrast to the validity of the disease realists, the validity of the empiricists is not an either–or phenomenon. It exists in degrees.

A reasonable approximation to a more empirical approach is the quantitative taxonomy of Achenbach and his colleagues (Achenbach, 1995, 2001; Achenbach, Bernstein, & Dumenci, 2005a, 2005b). Rather than using clinical observation to discover real syndromes with different natural histories, they use statistical procedures to detect syndromes defined as symptom patterns—or empirical regularities. The symptom patterns they have identified include anxious depression, withdrawn depression, rule breaking, and aggressive behavior. Instead of seeking to discover what is really there, they note that the observed patterns for any individual may differ depending on the data collected, such as self-report versus other-report. Variations in reporting may be weighted relative to each other, but not necessarily reduced to a single, true representation. Their multitaxonomic approach suggests that partialing the data into different patterns may increase the amount of information that is available to the clinician.

As noted previously, empiricists tend to more attracted to antirealism. For them the goal of science is to be able to make connections between observable phenomenon. They want to be able to make such claims as "If *X* happens, then we can predict *Y*," or "If you do *A*, then you can expect *B* to follow." Empiricists consider nonobservable entities such as electrons and diseases to be "inference tickets." In empiricist psychopathology, the kinds of inferences that are important include testable inferences about etiology, natural history, treatment response, outcome, and so on.

The difference between disease realism and empiricism can be seen in how they would conceptualize the historical shift from explaining phenomena such as hearing voices and thought insertion by reference to "demonic possession" to explaining them with respect to a syndrome named "schizophrenia." A disease realist will say that psychiatry has learned that demons do not exist. Instead of demonic possession, hearing voices is a psychotic episode that results from an underlying biopsychological dysfunction. For empiricists, arguments about whether demons exist are metaphysical superfluities. What is important is that the evidence for the inferences one would make with the demonic possession theory are not as valid as those one would make with the schizophrenia theory. Inferences about sinfulness as an etiological factor, the role of punishment from God, and the efficacy of exorcism all lack supporting evidence. In contrast, inferences about family history, time course, and response to medication have been better justified (supported by evidence).

It is important to emphasize that although the empiricists do not favor the validity of the disease realists, disease realists readily accept validity as it is defined by the empiricists. The information-based emphasis on the validity of inferences is a consensus view on validity among the scientific community, serving as the lowest common denominator. Whether scientific validity can or should refer to more than the validity of inferences is an important philosophical problem.

Construct Validation

Another approach to validity, introduced to psychology by Paul Meehl in the 1950s, is called "construct validation" (Cronbach & Meehl, 1955). Construct validation represents an evolution of the rigorous empiricism that dominated the philosophy of science in the early 20th century. The proponents of the revised empiricism, such as Herbert Feigl, were close colleagues of Meehl. The early empiricists were committed operationalists. That is, they believed that abstract concepts had to be explicitly defined in terms of observable data, and that the methods used to make the observations had to be explicit. For example, in psychological testing, a score of 65 or greater on a depression scale operationally defines "depression." For strict operationalists, what is meant by "depression" is the measurement.

Meehl and his colleagues questioned such a firm commitment to operationalism. As we learn more about the correlates of the depression scale, we may discover that "depression" is associated with other constructs, such as "anxiety" (Kendler, 1996); that it involves negative thoughts about the self, the world, and the future (Beck, 1976); or that being depressed leads to interpersonal rejection, which sets up a feedback loop that maintains the depression (Joiner & Coyne, 1999). A construct is explicitly defined in term of observables, but implicitly defined in terms of other constructs. The bearer of this *surplus meaning* above and beyond the operational definition is a theoretical construct—termed "depression." In Meehl's evolved empiricism, depression is construed realistically, not instrumentalistically.

Let us focus on the differences between the strict operationalist approach of the classical empiricists and Meehl's approach based on theoretical constructs/latent variables. For operationalists, a theoretical term such as "depression" is a verbal label for referring to what is being measured. The goal is to use the measure to infer observable consequences. In contrast, for Meehl the primary object of scientific interest is the latent variable called "depression," and our measure is considered to be a fallible indicator of that latent construct. Different measures of depression are conceptualized to be assessing the same thing, but each one is at best a partial measure. Observable evidence is used to triangulate on the construct and better understand its full nature.

Applied to psychiatric classification, the diagnostic criteria for MDD are also fallible indicators. Each indicator samples only a part of the domain of depression, and they all work better together to represent the whole domain. These indicators can be assessed for diagnostic validity, but the construct of depression cannot be reduced to its indicators. For example, in the development of DSM and ICD, some potentially relevant characteristics of MDD (such as increased experience of panic, somatic complaints, and irritability) were considered poor diagnostic criteria because they muddled differential diagnosis with the anxiety disorders, but from

a construct validity framework they are still indicators of depression. An indicator can be sensitive but not specific. A complete description of depression at the criteria level is sacrificed in order to facilitate differential diagnosis. The intricacies of such tradeoffs are another reason not to be too literalist about official diagnostic criteria.

Construing a stripped-down DSM or ICD category literally also has the unfortunate consequence of limiting what kind of features are considered relevant in developing models of underlying pathological processes. For these reasons, psychologists, who are schooled in the construct validity tradition, are uncomfortable using DSM or ICD as a textbook of psychopathology.

Validation, Theory, and Law

As noted earlier, the construct validation approach was inspired by an evolution within the empiricist philosophy of science. One of the things the revised empiricism focused on was laws (Feigl, 1970; Hempel, 1966). Laws describe patterns or regularities in nature that must occur. Knowing about the regularities allows one to predict what will occur, and also to explain why events did occur.

An example of a law is "All copper conducts electricity." If the law is valid, then whenever presented with an individual piece of copper, we know that it has to conduct electricity. This law-like or "nomological" approach to science would seek to confer on "Bill will respond to treatment *X* because he has schizophrenia" the same degree of certainty that we attribute to statements such as "This metal will conduct electricity because it is copper." Conducting electricity and responding to treatment *X* would be lawful consequences of the physical nature of copper and schizophrenia.

In this tradition, a theory is considered to be an interconnected network of theoretical terms and observations. Laws and hypotheses relate observations to theoretical terms. Laws also relate theoretical terms to each other. The whole edifice is called a "nomological network."

For example, observable behaviors related to facial, vocal, and postural cues might be associated with theoretical constructs such as anger, sadness, and fear, which are then combined to infer an even more abstract construct—namely, "neuroticism." A vulnerability relationship between neuroticism and other theoretical constructs, such as phobias and depression, can be formulated. The construct of depression has also been correlated with loss. By patiently tracking all the regularities, we can use constructs to predict and explain observable phenomena. To illustrate, we might predict imminent depression if a person with neuroticism experienced a major loss or humiliation in the past year, or explain a current depressive episode by referring to that prior loss.

Validating the construct consists in looking for evidence that either supports or refutes the various connections. As Meehl himself observed, his notion of validating a construct was based on the logical empiricists' notion of validating theories (Cronbach & Meehl, 1955). The different kinds of psychometric validity, such as concurrent and predictive validity, are strategies for testing theories in a logical empiricist framework.

Developments in the philosophy of science subsequent to Cronbach and Meehl's (1955) proposal have cast doubt on the logical empiricists' views of these issues, as also acknowledged by Meehl (1990). For example, the relationship between observation/evidence and a theory is surprisingly complicated. In testing theories, we make a conjecture about the implications that the evidence will have for the theory, but it turns out that it is often possible to reinterpret those implications. The idea that scientific theories bear a complex relationship with the evidence is called the "Duhem–Quine thesis." When the evidence does not correspond to our predictions, we know something is wrong, but we do not know where in the network the error lies. The theory could be mistaken or incomplete; the evidence could be unreliable; or one of the background assumptions on which the theory rests could be mistaken. In Meehl's terms, how we cope with this problem is a matter of scientific strategy.

Kendler's (1990) discussion of validation in light of constructs can also be understood from this perspective. On the basis of their disease theory, Robins and Guze (1970) listed a set of validators or evidence that would confirm disease status. For example, hypothesizing that schizophrenia is a major mental

illness with a genetic etiology indicates that increased family prevalence would be a good validator.

What happened when biological psychiatrists discovered that what runs in the families of patients diagnosed with schizophrenia is not only the Feighner criteria's major mental illness "schizophrenia," but a wider range of conditions (including certain personality disorders)? Did they conclude that schizophrenia does run in families as predicted, but that it is actually a broad category similar to the DSM-II concept? They could have adopted this decision-making strategy, but did not. Instead, they revised their conjecture about the relationship between evidence and theory by altering an auxiliary genetic hypothesis, rather than altering the "Schizophrenia is a major mental illness" hypothesis. The revised auxiliary hypothesis says that there is a genetic *vulnerability* to schizophrenia, and that schizophrenia is the most pathological outcome of that vulnerability, but not the only possible outcome. A category called the "schizophrenia spectrum" was introduced, and with it the construct of "schizotypal personality." The biological psychiatrists protected the hypothesis that schizophrenia is a major mental illness. A theory consists of a network of assumptions, and a number of those assumptions can be modified to make the theory consistent with the evidence.

Let us give another example. Diseases were once construed as altered states of functioning associated with pain and/or impairment. Did discovering hypertension and then not being able to confirm the presence of pain or impairment mean that hypertension was not a disease? No. Instead, physicians added a new component hypothesis to the disease theory—specifically, that a disease may raise the probability of pain or impairment in the future. Similar conditions, such as coronary artery disease, could subsequently be called "diseases." It is often possible to modify our conceptualizations about what implications empirical findings have for a theory being tested. Kendler (1990) has stated that these fundamentally nonempirical issues cannot be eliminated from classification.

Meehl (1986; reprinted in Chapter 8, this volume) initially touched upon such complications with respect to his notion of "open concepts" (not fully validated). When we try to validate a particular measure of schizophrenia, there is no gold standard against which we can evaluate it because any other standard (such as an ICD diagnosis) is also an open concept. Modifying the nomological network is also a possible response to a failed hypothesis, but Meehl was skeptical of the Duhem–Quine thesis because he believed that over time we can triangulate on the roughly *more correct* theory—by which he meant we would have good evidence for the basic laws and/or generalizations that make up the theory.

With a similar focus, the philosopher Charles Peirce (1878/1968) said that "the opinion which is *fated* to be ultimately agreed to by all who investigate, is what we mean by the truth, and the object represented in this opinion is the real" (p. 77; original emphasis). Inquiry will cease when we come to a point at which there are no longer any disagreements.

Everyone should agree that a good fit between theory and observation is quite difficult to achieve. Histories of past successes and abilities to predict the unexpected provide theories such as the central dogma in genetics and natural selection in evolution with a level of gravitas. The Duhem–Quine thesis, however, is quite radical. One of its consequences is that no matter how solid it appears, any construct is *potentially* revisable in the light of new evidence, and it is impossible for all the evidence ever to be in. It also claims that constructs can sometimes be saved from invalidation by altering another part of the theoretical network. Whenever there are multiple options for achieving a good fit, the facts alone cannot prescribe how the fit is to be decided. Therefore, we should not be too literal about any fit on which we settle.

We are left with a difference in emphasis between Meehl's rigorous approach (which focuses on constructs' being temporarily open, with the goal being to make them incrementally tighter) and the Duhem–Quine-inspired view (in which constructs will always be potentially open). We suggest that Meehl's approach articulates an important practical goal on which progress can be made; yet the grain of truth in Duhem–Quine radicalism is not likely ever to be eliminated from the classification of the complicated, multilevel constructs of psychopathology.

Explanatory Validity

Philosopher Dominic Murphy (2006) is critical of the construct validation perspective because he claims that it focuses on discovering "lawful regularities" rather than pathological processes. The notion that science explains with respect to laws is widely believed by philosophers to be inappropriate for biology, or even physics. Cronbach and Meehl's (1955) law-based account of theories is somewhat outdated. Murphy claims that validity should refer to a model's ability to explain successfully how it is that putative psychiatric disorders come about. It is not *disease entities*, *inferences*, or *constructs* that are valid; rather, *models* are valid when they track genuine causal structures.

There is nothing new about focusing on causality, as both disease realists and logical empiricists did so. The disease realists took their inspiration from the infectious-disease model in biology, whereas the logical empiricists were inspired by the explanatory laws of physics. What is new about model-based approaches is that they have a more pluralistic view of cause, and they also undermine the descriptive–etiological dualism that has driven a good deal of nosological bickering for 30 years.

The infectious-disease approach motivated psychopathologists to search for "necessary causes" (Guze, 1992). A necessary cause is one that must occur for the target effect to occur. If one can prevent or alter such a cause, then one can also eliminate the effect. Many thinkers have claimed that valid psychiatric disorders should also be *sorted* with respect to etiology, implicitly understood to be some kind of necessary cause (Andreasen, 1984; Kendell & Jablensky, 2003).

Infectious diseases and Mendelian genetic conditions have both casual and sorting value, but they have not been fruitful models for understanding other conditions. One reason given for this failure is to claim that the causes are there, but our basic science has not yet advanced enough for us to discover them. Some also claim that the syndromes identified in the current nosology are causally heterogeneous entities, and as a result function as barriers to the search for causes (Charney et al., 2002; Livesley, 2003; Parshall & Priest, 1993).

Another strategy is to adopt a broader notion of "cause"—one that includes risk factors and vulnerabilities. Risk factors include biological, psychological, and sociocultural variables that each contribute to producing an effect, but none of which is sufficient. Disorders are explained as a result of multifactorial packages, but any one of the factors by itself has limited causal force. As argued by Kendler (2005) with respect to genetics, the problem is not that psychiatric disorders lack a genetic basis, or that most of the important genes remain hidden; the problem is that any single gene on average accounts for only a small proportion of the variance of most disorders. Many different genes raise the risk of developing psychiatric disorders, but usually not by much. Genes also have nonspecific effects, raising the risk for several psychiatric disorders rather than having a 1:1 relationship with a particular disorder. Other genes may have protective effects, lowering one's risk.

New philosophical approaches to understanding scientific explanations that emphasize models offer good reasons for believing that the conventional causal theory of explanation in psychiatric nosology can be improved upon (Giere, 1988; Harré, 1986). Models are simplified and idealized representations of phenomena that are not literally true in all respects, but that still tell us what phenomena are like. A famous example of an idealized model is Freud's model of id, ego, and superego. Other examples of models include the central dogma in molecular genetics, the model of allopatric speciation in evolutionary theory, and neural network models in cognitive science.

Models only represent the information that is deemed relevant. To make this point, philosophers such as Ronald Giere (1999) compare models to maps. A road map is a model of a geographical area that is realistic to a greater or lesser extent, but usually a partial representation. It does not model the vegetation, the diversity of animal life, the uphill and downhill sections of the road, the style of the buildings, or the population density.

Although they are not intended to be literally true, models should have what philosophers call "verisimilitude," or approximate truth. An example of approximate truth is Carnot's explanation of heat engines with

reference to a fluid called "caloric." Although caloric fluid does not exist, Carnot's description can be considered the first formulation of the second law of thermodynamics.

According to Murphy (2006), DSM and ICD syndromes are like maps as well. They are more or less realistic, but, like maps, they represent information relative to a particular set of interests, rather than being literally true. Instead of viewing diagnostic categories as "natural kinds," Murphy thinks they should be seen as idealized representations called "exemplars." In doing so, he tentatively joins others (e.g., Ghaemi, 2003; Zachar, 2000a, 2000b, 2008) who are suspicious of using the philosophical concept of natural kind to understand psychiatric classification. Murphy notes that an exemplar should itself be mapped with a typical causal profile, or even families of profiles. The casual profiles will themselves be idealizations that become more or less realistic as details are added and subtracted, depending on one's explanatory purposes.

In psychopathology, models can potentially provide coarse-grained accounts of a disorder in general, or can be made so fine-grained that they attempt to model the development and maintenance of a disorder in a single individual. One model of MDD for 3 million patients risks being a gross approximation, but 3 million idiographic models of depression would be impractical, and neither is remotely the best model for all purposes.

Hesse (2000) notes that philosophers have proposed a range of model-based views for understanding scientific explanation. An approach that is particularly relevant for psychiatric nosology, and is often used in biology, is called the "mechanistic model." Rather than basing explanations on general laws and nomological networks, mechanistic models seek to explain by understanding how systems work, usually by decomposing them into parts and proposing theories about how those parts work together. They are often described as attempts to understand how causal processes are actually implemented in the world.

Constructing such models involves more than the search for necessary causes. For example, in developing a model of an internal combustion engine, we would not say that the fuel injector causes the car to go. Instead, the model seeks to describe what role the fuel injector plays in the overall functioning of the system. Whether these roles will be construed causally depends on the question being asked. In asking about why a car is overemitting pollutants, if we find out that the injector is delivering too much fuel to the cylinders, then we would assign the injector a causal role. Most of the time, however, a fuel injector is just seen as a part of the engine with a certain functional role. Most of the modeling work involves understanding functional roles rather than seeking causes.

As Kendler (2008a) notes, mechanistic models begin as proposals or sketches of how a system might work. Over time, the details are filled in and the sketch is updated. Models are tested until things start to fit together; in this way, they are evidence-based. Once the description reaches a certain level of detail, it can be used to do explanatory work. Bechtel and Abrahamsen (2005) say that mechanistic models explain *why* by describing *how*.

One final aspect about models is that they can be formulated at different levels of generality. Murphy (2006) believes that the primary explanatory level for a scientific psychopathology will be that of cognitive neuroscience, but he and most others assume that the better explanations will be multilevel. The genetic, computational, psychological, social-psychological, and cultural levels will all work together, and no single model will fill in all the details. What is new is an expectation that the details of how cultures and brains interact should be modeled in terms of empirically supported causal structures, rather than left at the level of slogans.

Literalism and Science

We bring this chapter to a close with some thoughts about diagnostic literalism. It may have occurred to readers that one of the goals of science is to offer literally true rather than metaphorical accounts of natural phenomena. For example, scientists believe that the evolutionary account of organic diversity is literally true, but that the creation story in Genesis is not. They also believe that the Copernican theory is literally true, whereas the Ptolemaic theory was not. Shouldn't nosology aspire to be like Darwin and Copernicus? Our response is "Yes, of course it should."

The current scientific understanding of the solar system is a good choice for exploring literalism. The literalist story is that the sun occupies the center of the solar system, and the planets revolve around the sun. The Copernican model is well supported. There are no good competitors to it, nor does it seem likely that there will ever be. From the sun in the center outward, the planets are in this order: Mercury, Venus, Earth, Mars, Jupiter, Saturn, Uranus, Neptune, and Pluto. This model has been scientific orthodoxy for over 75 years.

But wait. Are there literally 9 planets? Or are there 8, or 10? One of the amazing things about Pluto is that its existence and location were predicted by Percival Lowell in the early 1900s. Astronomers discovered that the observed orbit of Uranus was not what the scientific calculations predicted, but deduced that if there were another planet beyond Neptune, the predicted orbit would more closely match the observed orbit (Sobel, 2005). Lowell made some calculations, and Clyde Tombaugh found the planet Pluto in 1930, just where Lowell said it would be.

In 2005, astronomers at the Palomar Observatory discovered an object beyond Pluto that later appeared to be a 10th planet, now named Eris. One of the reasons for considering Eris a 10th planet is that it is 20–30% larger than Pluto and also has at least one moon (Ridpath & Tirion, 2008).

An interesting feature of Eris is that it has a highly eccentric orbit. Other planets orbit the sun in roughly the same plane; their orbits mostly differ by being farther away from the sun (Millennium House, 2007). The orbit of Eris, on the other hand, is tilted 44 degrees relative to the orbital plane of Earth. This made some astronomers reluctant to call Eris a real planet.

It turns out that Pluto has a similar problem. Its orbit is tilted 17 degrees relative to that of Earth. Furthermore, for 20 years of its 250-year orbit around the sun, Pluto is actually closer to the sun than is Neptune (Sobel, 2005). It also seems to be a member of a large swarm of objects that orbit the sun beyond Neptune, called the "Kuiper belt." Many "Kuiper belt objects" (KBOs) also have their own moons.

Echoing the American Psychiatric Association's 1973 decision to declassify homosexuality as a mental disorder, in 2006 the International Astronomical Union voted to demote Pluto from a real planet to a dwarf planet (Millennium House, 2007). At the same time, Ceres, which lies between Mars and Jupiter, was promoted from an asteroid to a dwarf planet. An alternative proposal would have made Ceres a planet.

There is apparently continuing disagreement about these classifications, especially regarding the scientific criteria for "valid" planets. For example, there was a concern that if Pluto and Eris were accepted as planets, several other KBOs would also be planets, and 30 or more planets is too many. Compare this to claims that a 30–50% lifetime prevalence for experiencing a mental disorder is too high (Mechanic, 2003; Narrow, Rae, Robins, & Regier, 2002). A typical solution to such conundrums is to choose a polythetic set of distinctions that also respect our intuitions—for example, defining "planet" so that Mercury through Uranus are included but Pluto is excluded. Some believe that such nonempirical considerations are scientifically unseemly, but such complexities are less of a problem for those who construe classification as an ongoing deliberative activity.

Quite likely, there are astronomical thinkers with tendencies toward literalism who want to keep Pluto a planet. We would hope that there are not any who want to keep the number of planets at 9.

Should we call Pluto a planet, a dwarf planet, or a KBO? Closer to home, is chronic dysthymia better termed depressive personality disorder? Is social phobia really avoidant personality disorder? Should we call minor depression a psychiatric disorder, or is it a subthreshold condition that is occasionally a focus of clinical attention? Some of the arguments about minor disorders in psychiatry and minor or dwarf planets in astronomy are quite similar.

Reasons can be given for making classificatory distinctions, and facts about the world are among those reasons. There is a close relationship between facts (such as the nature of orbits or the presence of moons) and nonempirical preferences about what role astronomers want planets to play in classification. The same is true for facts about genetics and cognitive–emotional processes and what mental health professionals want a diagnosis to do. In psychopathology,

once there is an agreement on what we want a diagnosis to do (e.g., to identify etiologically homogeneous groups), then science can address that problem. Science can, however, address multiple diagnostic purposes, and there is no good reason to believe that all scientifically supported classifications will be consistent with each other.

Studying history also helps occasionally. For example, when Pluto was discovered and named a planet, it was assumed that in order to influence Uranus's orbit, it had to be much larger than it was eventually discovered to be. More importantly, the astronomers had made a mistake when predicting the orbit of Uranus. In their calculations, they plugged in an incorrect size for Neptune's mass. If they had plugged in the correct size, the difference between the predicted and the observed orbits of Uranus would not have been so great (Sobel, 2005). It was mere accident that Tombaugh found a big object where Lowell said it should be.

So are there literally 8, 9, or 10 planets? As noted, if there are 10, the number of planets is likely to be greater than 10 because there are a lot of big spherical objects with moons out there. Many readers are probably thinking that there are 8, but what if Earth-like "planets" in other solar systems have orbits like those of Pluto or Eris? We hope it has occurred to readers that the taxonomic problem in astronomy is not about developing either a literal or a metaphorical classification of the solar system. The 9-planet model was not a metaphor, and a commitment to its literal truth would be scientifically harmful.

Returning to the more specific topic of philosophical problems in the classification of psychopathology, taxonomists should reject the assumption that in order to be as scientific as the classification systems of the natural and the biological sciences, psychiatric nosology should be construed literally. Seeking the literal truth can be an important motivator, but also, as just noted, has the potential to do harm.

Conclusion

In deciding which philosophical issues to explore in this chapter, we have selected conceptual problems that are internal to the scientific study of psychopathology. With respect to dimensional and categorical models, we have advocated a pragmatic perspective that prefers to utilize the strengths of both approaches. We have argued that conceptualizing scientific classification as an attempt to "carve nature at its joints" helps the field aspire to classify the world objectively, but also makes diagnostic literalism and reification of classifications harder to avoid. With respect to the concept of "clinical significance," we have stated that making distinctions among "optimal," "normal," "compromised," and "impaired" requires not just the discovery of facts, but also evaluative judgments. In some cases these judgments can be rationally articulated, although the level of consensus on values will vary from disorder to disorder.

We have further argued that it would be better if disorders were conceptualized in terms of pathological processes, rather than being solely descriptive. Wakefield's harmful-dysfunction model provides a quite helpful conceptual definition of "disorder," but we have suggested that objective dysfunctions may not be subject to empirical confirmation in many cases. Describing four different perspectives on the validity of psychopathological constructs, we do not believe that any of those perspectives should be considered to be the correct approach to validity. In our view, a "validity-of-inferences model" represents a consensus view on validity. We have suggested that a philosophically updated version of the construct validity approach emphasizing local explanatory models—models that are calibrated to the purposes one has for classifying—offers a promising way forward.

Acknowledgment

Andrea Solomon and Steve LoBello provided helpful commentary on an earlier version of this chapter.

References

Achenbach, T. M. (1995). Empirically based assessment and taxonomy: Applications to clinical research. *Psychological Assessment*, *3*, 261–274.

Achenbach, T. M. (2001). Empirically based assessment and taxonomy across the life span. In J. E. Helzer & J. J. Hudziak (Eds.), *Defining psychopathology in the 21st century* (pp. 155–168). Washington, DC: American Psychiatric Press.

Achenbach, T. M., Bernstein, A., & Dumenci, L. (2005a). DSM-oriented scales and statistically based syndromes for ages 18 to 59: Linking taxonomic paradigms to facilitate multitaxonomic approaches. *Journal of Personality Assessment*, *84*, 49–63.

Achenbach, T. M., Bernstein, A., & Dumenci, L. (2005b). A snark or a boojum?: Exploring multitaxonomic possibilities and building on Widiger's commentary. *Journal of Personality Assessment*, *84*, 66–69.

Ahn, W., Flanagan, E. H., Marsh, J. K., & Sanislow, C. A. (2006). Beliefs about essences and the reality of mental disorders. *Psychological Science*, *17*, 759–766.

Alloy, L. B., Albright, J. S., Abramson, L. Y., & Dykman, B. M. (1990). Depressive realism and nondepressive optimistic illusions: The role of the self. In R. E. Ingram (Ed.), *Contemporary psychological approaches to depression: Treatment, research, and theory* (pp. 71–86). New York: Plenum Press.

Andreasen, N. C. (1984). *The broken brain: The biological revolution in psychiatry*. New York: Harper & Row.

Bechtel, W., & Abrahamsen, A. (2005). Explanation: A mechanistic alternative. *Studies in History and Philosophy of the Biological and Biomedical Sciences*, *36*, 421–441.

Beck, A. T. (1976). *Cognitive therapy and the emotional disorders*. New York: International Universities Press.

Becker, D., Lomranz, J., Pines, A., Shmotkin, D., Nitza, E., BennAmitay, G., et al. (2001). Psychological distress around menopause. *Psychosomatics*, *42*, 252–257.

Bell, C. (2004). Racism: A mental illness. *Psychiatric Services*, *55*, 1343.

Bentall, R. P. (1992). A proposal to classify happiness as a psychiatric disorder. *Journal of Medical Ethics*, *18*, 94–98.

Berrios, G. E. (1991). Affective disorders in old age: A conceptual history. *International Journal of Geriatric Psychiatry*, *6*, 337–346.

Brendel, D. (2006). *Healing psychiatry*. Cambridge, MA: MIT Press.

Charney, D. S., Barlow, D. H., Botteron, K., Cohen, J. D., Goldman, D., Gur, R. E., et al. (2002). Neuroscience research agenda to guide development of a pathophysiologically based classification system. In D. J. Kupfer, M. B. First, & D. A. Regier (Eds.), *A research agenda for DSM-V* (pp. 31–83). Washington, DC: American Psychiatric Press.

Cronbach, L. J., & Meehl, P. E. (1955). Construct validity in psychological tests. *Psychological Bulletin*, *52*, 281–302.

Dao, T. K., Prevatt, F., & Horne, H. L. (2007). Differentiating psychotic patients from nonpsychotic patients with the MMPI-2 and Rorschach. *Journal of Personality Assessment*, *90*, 93–101.

Dewey, J. (1938). *Logic: The theory of inquiry*. New York: Holt.

Dewey, J. (1958). *Experience and nature*. New York: Dover. (Original work published 1928)

Ereshefsky, M. (1992). Eliminative pluralism. *Philosophy of Science*, *59*, 671–690.

Feigl, H. (1970). The "orthodox" view of theories: Remarks in defense as well as critique. In M. Radner & S. Winokur (Eds.), *Minnesota studies in the philosophy of science* (Vol. 4, pp. 3–16). Minneapolis: University of Minnesota Press.

First, M. B., Cuthbert, B., Krystal, J. H., Malison, R., Offord, D. R., Reiss, D., et al. (2002). Personality disorders and relational disorders. In D. J. Kupfer, M. B. First, & D. A. Regier (Eds.), *A research agenda for DSM-V* (pp. 123–129). Washington, DC: American Psychiatric Association.

Fulford, K. W. M. (1989). *Moral theory and medical practice*. Cambridge, UK: Cambridge University Press.

Ghaemi, S. N. (2003). *The concepts of psychiatry*. Baltimore: Johns Hopkins University Press.

Giere, R. N. (1988). *Explaining science: A cognitive approach*. Chicago: University of Chicago Press.

Giere, R. N. (1999). *Science without laws*. Chicago: University of Chicago Press.

Goldberg, L. R., & Velicer, W. F. (2006). Principles of exploratory factor analysis. In S. Strack (Ed.), *Differentiating normal and abnormal personality* (pp. 209–237). New York: Springer.

Goodman, N. (1978). *Ways of worldmaking*. Indianapolis, IN: Hackett.

Guze, S. (1992). *Why psychiatry is a branch of medicine*. New York: Oxford University Press.

Hacking, I. (1999). *The social construction of what?* Cambridge, MA: Harvard University Press.

Harré, R. (1986). *Varieties of realism*. Oxford, UK: Blackwell.

Haslam, N. (2000). Psychiatric categories as natural kinds: Essentialist thinking about mental disorder. *Social Research*, *67*, 1032–1058.

Haslam, N. (2007). The latent structure of mental disorders: A taxometric update on the categorical vs. dimensional debate. *Current Psychiatry Reviews*, *3*, 172–177.

Haslam, N., & Williams, B. (2006). Taxometrics. In S. Strack (Ed.), *Differentiating normal and abnormal personality* (pp. 284–308). New York: Springer.

Helzer, J. R., Kraemer, H. C., & Krueger, R. F. (2006). The feasibility and need for dimensional psychiatric diagnoses. *Psychological Medicine*, *36*, 1671–1680.

Hempel, C. G. (1966). *Philosophy of natural science*. Englewood Cliffs, NJ: Prentice-Hall.

Hesse, M. (2000). Models and analogies. In W. H. Newton-Smith (Ed.), *A companion to the philosophy of science* (pp. 299–307). Oxford, UK: Blackwell.

Horwitz, A. V., & Wakefield, J. C. (2007). *The loss of sadness*. New York: Oxford University Press.

James, W. (1975). *Pragmatism* and *The meaning of truth*. Cambridge, MA: Harvard University Press. (Original works published 1907, 1909)

Joiner, T., & Coyne, J. C. (Eds.). (1999). *The interactional nature of depression*. Washington, DC: American Psychological Association.

Kendell, R. (1975). The concept of disease. *British Journal of Psychiatry*, *127*, 305–315.

Kendell, R., & Jablensky, A. (2003). Distinguishing between the validity and utility of psychiatric disorders. *American Journal of Psychiatry*, *160*, 4–12.

Kendler, K. S. (1980). The nosologic validity of paranoia (simple delusional disorder): A review. *Archives of General Psychiatry*, *37*, 699–906.

Kendler, K. S. (1990). Toward a scientific psychiatric nosology. *Archives of General Psychiatry*, *47*, 969–973.

Kendler, K. S. (1996). Major depression and generalised anxiety disorder: Same genes, (partly) different environments—revisited. *British Journal of Psychiatry*, *168*(Suppl. 30), 68–75.

Kendler, K. S. (2005). "A gene for . . . ": The nature of gene action in psychiatric disorders. *American Journal of Psychiatry*, *162*, 1243–1252.

Kendler, K. S. (2008a). Explanatory models for psychiatric illness. *American Journal of Psychiatry*, *165* , 695–702.

Kendler, K. S. (2008b). Review of *The loss of sadness*: How psychiatry transformed normal sorrow into depressive disorder. *Psychological Medicine*, *38*, 148–150.

Kendler, K. S., & Zachar, P. (2008). The incredible insecurity of psychiatric nosology. In K. S. Kendler & J. Parnas (Eds.), *Philosophical issues in psychiatry: Explanation, phenomenology and nosology* (pp. 368–383). Baltimore: Johns Hopkins University Press.

Kessler, R. C. (2002). The categorical versus dimensional assessment controversy in the sociology of mental illness. *Journal of Health and Social Behavior*, *43*, 171–188.

Kessler, R. C., Merikangas, K. R., Berglund, P., Eaton, W. W., Koretz, D. S., & Walters, E. E. (2003). Mild disorders should not be eliminated from the DSM-V. *Archives of General Psychiatry*, *60*, 1117–1122.

Kripke, S. (1980). *Naming and necessity*. Cambridge, MA: Harvard University Press.

Krueger, R. F. (1999). The structure of common mental disorders. *Archives of General Psychiatry*, *56*, 921–926.

Krueger, R. F., Caspi, A., Moffitt, T. E., & Silva, P. A. (1998). The structure and stability of common mental disorders (DSM-III-R): A longitudinal–epidemiological study. *Journal of Abnormal Psychology*, *107*, 216–227.

Krueger, R. F., Watson, D., & Barlow, D. H. (2005). Introduction to the special section: Toward a dimensionally based taxonomy of psychopathology. *Journal of Abnormal Psychology*, *114*, 491–493.

Lilienfeld, S. O., & Waldman, I. D. (2004). Comorbidity and Chairman Mao. *World Psychiatry*, *3*, 26–27.

Livesley, W. J. (2003). Diagnostic dilemmas in classifying personality disorder. In K. A. Phillips, M. B. First, & H. A. Pincus (Eds.), *Advancing DSM* (pp. 153–189). Washington, DC: American Psychiatric Association.

Livesley, W. J. (2006). Dimensional assessment of personality pathology (DAPP) approach to personality disorder. In S. Strack (Ed.), *Differentiating normal and abnormal personality* (pp. 401–429). New York: Springer.

Markon, K. E., & Krueger, R. F. (2006). Latent variable modeling. In S. Strack (Ed.), *Differentiating normal and abnormal personality* (pp. 239–256). New York: Springer.

Mayr, E. (1982). *The growth of biological*

thought: Diversity, evolution, and inheritance. Cambridge, MA: Harvard University Press.

Mayr, E. (1988). *Toward a new philosophy of biology: Observations of an evolutionist*. Cambridge, MA: Harvard University Press.

Mayr, E. (1991). *One long argument: Charles Darwin and the genesis of modern evolutionary thought*. Cambridge, MA: Harvard University Press.

Mechanic, D. (2003). Is the prevalence of mental disorders a good measure of the need for services? *Health Affairs*, *22*, 8–20.

Meehl, P. E. (1986). Diagnostic taxa as open concepts: Metatheoretical and statistical questions about reliability and construct validity in the grand strategy of nosological revision. In T. Millon & G. Klerman (Eds.), *Contemporary directions in psychopathology* (pp. 215–231). New York: Guilford Press.

Meehl, P. E. (1990). Appraising and amending theories: The strategy of Lakatosian defense and two principles that warrant it. *Psychological Inquiry*, *1*, 108–141.

Meehl, P. E. (1993). Philosophy of science: Help or hindrance. *Psychological Reports*, *72*, 707–733.

Millenium House. (2007). *Astronomica*. Elanora Heights, Australia: Author.

Mishler, B. D., & Brandon, R. N. (1987). Individuality, pluralism, and the phylogenetic species concept. *Biology and Philosophy*, *2*, 397–414.

Murphy, D. (2006). *Psychiatry in the scientific image*. Cambridge, MA: MIT Press.

Narrow, W. E., Rae, D. S., Robins, L. N., & Regier, D. A. (2002). Revised prevalence estimates of mental disorders in the United States. *Archives of General Psychiatry*, *59*, 115–123.

Parshall, A. M., & Priest, R. G. (1993). Nosology, taxonomy and the classification conundrum of the functional psychoses. *British Journal of Psychiatry*, *162*, 227–236.

Peirce, C. S. (1968). How to make our ideas clear. In P. Kurtz (Ed.), *American philosophy in the twentieth century* (pp. 62–79). New York: Macmillan. (Original work published 1878)

Pincus, H. A., McQueen, L. E., & Elinson, L. (2003). Subthreshold mental disorders: Nosological and research recommendations. In K. A. Phillips, M. B. First, & H. A. Pincus (Eds.), *Advancing DSM* (pp. 129–144). Washington, DC: American Psychiatric Association.

Putnam, H. (1975). The meaning of "meaning." In H. Putnam, *Philosophical papers: Vol. 2. Mind, language and reality* (pp. 215–271). Cambridge, UK: Cambridge University Press.

Quine, W. V. (1969). *Ontological relativity and other essays*. New York: Columbia University Press.

Regier, D. A. (2003). Mental disorder diagnostic theory and practical reality: An evolutionary perspective. *Health Affairs*, *22*, 21–27.

Regier, D. A. (2007). Dimensional approaches to psychiatric classification: Refining the research agenda for DSM-V: An introduction. *International Journal of Methods in Psychiatric Research*, *16*(Suppl. 1), S1–S5.

Ridley, M. (1986). *Evolution and classification: The reformation of cladism*. New York: Longman.

Ridpath, I., & Tirion, W. (2008). *Stars and planets*. Princeton, NJ: Princeton University Press.

Robins, E., & Guze, S. (1970). Establishment of diagnostic validity in psychiatric illness: Its application to schizophrenia. *American Journal of Psychiatry*, *126*, 983–987.

Rossiter, L. H. (2006). *The liberal mind: The psychological causes of political madness*. St. Charles, IL: Free World Books.

Sadler, J. Z. (2005). *Values and psychiatric diagnosis*. Oxford, UK: Oxford University Press.

Savage, M. (2005). *Liberalism is a mental disorder*. Nashville, TN: Nelson Current.

Schaffner, K. F. (1993). *Discovery and explanation in biology and medicine*. Chicago: University of Chicago Press.

Simms, L. J., & Clark, L. A. (2006). The Schedule of Nonadaptive and Adaptive Personality (SNAP): A dimensional measure of traits relevant to personality and personality pathology. In S. Strack (Ed.), *Differentiating normal and abnormal personality* (pp. 431–450). New York: Springer.

Sobel, D. (2005). *The planets*. New York: Penguin.

Spitzer, R. L., & Wakefield, J. W. (1999). DSM-IV diagnostic criteria for clinical significance: Does it help solve the false positive problem? *American Journal of Psychiatry*, *156*, 1856–1864.

Udovitch, M. (2002, September 8). The way we live now: 9-08-02: Phenomenon; A secret society of the starving. *New York Times*. Retrieved May 30, 2008, from *www.nytimes.com*

Wakefield, J. C. (1992). The concept of mental disorder: On the boundary between biological

facts and social values. *American Psychologist*, *47*, 373–388.

Wakefield, J. C. (1999a). Evolutionary versus prototype analyses of the concept of disorder. *Journal of Abnormal Psychology*, *108*, 374–399.

Wakefield, J. C. (1999b). Mental disorder as a black box essentialist concept. *Journal of Abnormal Psychology*, *108*, 465–472.

Wakefield, J. C. (2000). Aristotle as sociobiologist. *Philosophy, Psychiatry, and Psychology*, *7*, 17–44.

Wakefield, J. C. (2006). Personality disorder as harmful dysfunction: DSM's cultural deviance requirement reconsidered. *Journal of Personality Disorders*, *20*, 157–169.

Weissman, M. M. (1979). The myth of involutional melancholia. *Journal of the American Medical Association*, *242*, 742–744.

Widiger, T. A., & Samuel, D. B. (2005). Diagnostic categories or dimensions? A question for the *Diagnostic and Statistical Manual of Mental Disorders*–Fifth Edition. *Journal of Abnormal Psychology*, *114*, 494–504.

van Frassen, B. C. (1980). *The scientific image*. Oxford, UK: Oxford University Press.

Zachar, P. (2000a). Folk taxonomies should not have essences either. *Philosophy, Psychiatry, and Psychology*, *7*, 191–194.

Zachar, P. (2000b). Psychiatric disorders are not natural kinds. *Philosophy, Psychiatry, and Psychology*, *7*, 167–182.

Zachar, P. (2006). The classification of emotion and scientific realism. *Journal of Theoretical and Philosophical Psychology*, *26*, 120–138

Zachar, P. (2008). Real kinds, but no true taxonomy: An essay in psychiatric systematics. In K. S. Kendler & J. Parnas (Eds.), *Philosophical issues in psychiatry: Explanation, phenomenology and nosology* (pp. 327–355). Baltimore: Johns Hopkins University Press.

Zachar, P., & Bartlett, S. (2001). Basic emotions and their biological substrates. *Consciousness and Emotion*, *2*, 189–221.

Zachar, P., & Kendler, K. S. (2007). Psychiatric disorders: A conceptual taxonomy. *American Journal of Psychiatry*, *164*, 557–565.

CHAPTER 7

Classification Considerations in Psychopathology and Personology

THEODORE MILLON

In thoughts I expressed over 40 years ago concerning the character of theory (Millon, 1969), I voiced my chagrin that nature was not made to suit our need for a tidy and well-ordered universe. Quite evidently, the complexity and intricacy of the natural world make it difficult not only to establish clear-cut relationships among phenomena, but to find simple ways in which these phenomena can be classified or grouped. In our desire to discover the essential order of nature, we find it necessary to concern ourselves with only a few of the infinite number of elements that could be chosen; in this selection, we narrow our choice only to those aspects of nature that we believe best enable us to answer the questions we have posed. Moreover, the elements we choose are labeled, transformed, and reassembled in a variety of ways, but we must bear in mind that these labels and transformations are not "realities." The various concepts and categories that we construct as scientists are only optional tools to guide our observation and interpretation of the natural world; different concepts and categories may be formulated as alternative approaches to the understanding of the same subject of inquiry. These tools are especially necessary when the terrain we face is as uncharted as the taxonomy of psychopathology, and the materials of which it is composed are as intractable as they are.

The subject areas which subdivide the natural world differ in the degree to which their phenomena are inherently differentiated and organized. Some areas are "naturally" more articulated and quantifiable than others. To illustrate, the laws of physics relate to highly probabilistic processes in many of its most recondite spheres, but the features of our everyday physical world are highly ordered and predictable. Theories in this latter realm of physics (e.g., mechanics, electricity) serve largely to *uncover* the lawful relationships that do in fact exist in nature: it was the task of physicists at the turn of the last century to fashion a network of constructs that faithfully mirrored the universal nature of the phenomena they studied. By contrast, probabilistic realms of physical analysis (e.g., short-lived elementary particles) or systems of recent evolutionary development (e.g., human interactions) are inherently weakly organized, lacking either articulated or invariant connections among their constituent elements. In knowledge domains that relate to these less ordered spheres of nature (the "softer" sciences), classifiers and theorists

find it necessary to *impose* a somewhat arbitrary measure of systematization; in so doing, they construct a degree of clarity and coherence that is not fully consonant with the "naturally" unsettled and indeterminate character of their subject. Rather than equivocate strategically, or succumb to the "futility of it all," these workers make noble or pretentious efforts to arrange and categorize these inexact and probabilistic elements so that they simulate a degree of precision and order transcending that which they intrinsically possess. For instance, in fields such as economics and psychopathology, categories and classifications are in considerable measure splendid fictions, compelling notions, or austere formulas devised to give coherence to their *inherently imprecise* subjects.

The logic, substance, and structures created or imposed as a means of giving order to the phenomena of psychopathology are the principal topics of this chapter. It may serve as either a pedagogical introduction or a comprehensive review, depending on the reader's starting point.

Humans developed reliable and useful classifications long before the advent of modern scientific thought and methods. Information, skill, and instrumentation were achieved without "science" and its symbolic abstractions and techniques of research. If useful classifications could be acquired through intelligent observation and common sense alone, what special values are derived by applying the complicated and rigorous procedures required in developing explicit criteria, taxonic homogeneity, and diagnostic efficiency? Are rigor, clarity, precision, and experimentation more than compulsive and picayunish concerns for details, more than the pursuit of the honorific title of "science"? Are the labors of differentiating attributes or exploring optimal cutoff scores in a systematic fashion worth the time and effort involved?

There is little question in the "age of science" that the answer would be yes. But why? What are the distinguishing virtues of precision in terminology, the specification of observable conceptual referents, the analysis of covariant attribute clusters? What sets these procedures apart from everyday methods of categorizing knowledge?

Because the number of ways we can observe, describe, and organize the natural world is infinite, the terms and concepts we create to represent these activities are often confusing and obscure. For example, different words are used to describe the same behavior, and the same word is used for different behaviors. Some terms are narrow in focus, others are broad, and some are difficult to define. Because of the diversity of events to which we can attend, or the lack of precision in the language we employ, different processes are confused and similar events get scattered in hodgepodge fashion across a scientific landscape; as a consequence, communication gets bogged down in terminological obscurities and semantic controversies.

One of the goals of formalizing the phenomena that constitute a scientific subject is to avoid this morass of confusion. Not all phenomena related to the subject need be attended to at once. Certain elements may be selected from the vast range of possibilities because they seem relevant to the solution of a specific question. And to create a degree of reliability or consistency among the efforts of those interested in a subject, its elements are defined as precisely as possible and classified according to their core similarities and differences (Dougherty, 1978; Tversky, 1977). In a subject such as psychopathology, these classes or categories are given specific labels, which serve to represent them. This process of definition and classification is indispensable for systematizing observation and knowledge.

Are conceptual definition and classification possible in psychopathology? Can these most fundamental of scientific activities be achieved in a subject that is inherently inexact, of only modest levels of intrinsic order—one in which even the very slightest variations in context or antecedent conditions (often of a minor or random character) produce highly divergent outcomes (Bandura, 1982)? Because this "looseness" within the network of variables in psychopathology is unavoidable, are there any grounds for believing that such endeavors could prove more than illusory? Persuasive answers to this question of a more philosophical nature must be bypassed in this all-too-concise chapter; those who wish to pursue this line of analysis would gain much by reading, among others, Pap (1953), Hempel (1965), and Meehl (1978). Let us touch, albeit brief-

ly, on a more tangible and psychologically based rationale for believing that formal classification in psychopathology may prove to be at least a moderately fruitful venture.

Why May Formal Classification Be Useful?

There is a clear logic to classifying "syndromes" in medical disorders. Bodily changes wrought by infectious diseases and structural deteriorations repeatedly display themselves in reasonably uniform patterns of signs and symptoms that "make sense" in terms of anatomical structures' and physiological processes' alterations and dysfunctions. Moreover, these biological changes provide a foundation not only for identifying the etiology and pathogenesis of these disorders, but also for anticipating their course and prognosis. Logic and fact together enable us to construct a rationale to explain why most medical syndromes express themselves in the signs and symptoms they do, as well as the sequences through which they unfold.

Can the same be said for psychopathological classifications? Is there a logic, perhaps evidence, for believing that certain forms of clinical expression (e.g., behaviors, cognitions, affects, mechanisms) cluster together as medical syndromes do—that is, not only covary frequently, but "make sense" as coherently organized and reasonably distinctive groups of characteristics? Are there theoretical and empirical justifications for believing that the varied features of personality display a configurational unity and expressive consistency over time? Will the careful study of individuals reveal congruency among such attributes as overt behavior, intrapsychic functioning, and biophysical disposition? Are this coherence and stability of psychological functioning valid phenomena—that is, not merely imposed upon observed data by virtue of clinical expectation or theoretical bias?

There are reasons to believe that the answer to each of the preceding questions is yes. Stated briefly and simply, the observations of covariant patterns of signs, symptoms, and traits may be traced to two facts: People possess relatively enduring biophysical dispositions that give a consistent coloration to their experience; and the range of experiences to which people are exposed throughout their lives is both limited and repetitive (Millon, 1969, 1981). Given the limiting and shaping character of these biogenic and psychogenic factors, it should not be surprising that individuals develop clusters of prepotent and deeply ingrained behaviors, cognitions, and affects that clearly distinguish them from others of dissimilar backgrounds. Moreover, once several components of a particular clinical pattern are identified, knowledgeable observers are able to trace the presence of other, unobserved, but frequently correlated features seen in that pattern.

If we accept the assumption that most people do display patterns of internally consistent characteristics, we are led next to the question of whether groups of patients evidence commonality in the patterns they display. The notion of clinical categories rests on the assumption that there are some such shared covariances—for example, regular groups of diagnostic signs and symptoms that can confidently be used to distinguish certain classes of patients. (However, this assumption does not negate the fact that patients classified into categories display considerable differences as well—differences we routinely observe with medical diseases.)

Although grievances itemizing the inadequacies of both our current and historic systems of psychopathology have been voiced for years, as have suggestions that endeavors to refine these efforts are fussy and misdirected, if not futile and senseless pretensions that should be abandoned, the presence of categorical systems is both unavoidable (owing to our human linguistic and attribution habits) and inevitable (owing to our need to differentiate and to record, at the very least, the most obvious of dissimilarities among psychologically impaired individuals). Given the fact that one or another set of categories is inevitable—or, as Kaplan (1964, p. 279) once phrased it, "it is impossible to wear clothing of no style at all"—it would appear both sensible and fitting that we know the explicit basis upon which such distinctions are to be made, rather than have them occur helter-skelter in nonpublic and nonverifiable ways. Furthermore, if psychopathology is to evolve into a true science, its diverse phenomena must be subject to

formal identification, differentiation, and quantification procedures. Acts such as diagnosis and assessment presuppose the existence of discernible phenomena that can be recognized and measured. Logic necessitates, therefore, that psychopathological states and processes be distinguished from one another, being thereby categorizable in some degree *before* they can be subjected to identification and quantification.

The number of categories that can be distinguished in a classification schema will depend in part on the incisiveness with which diagnosticians make their clinical observations and the creative inferences they draw from them. As has been discussed in earlier pages, classification data may legitimately be derived both from concrete observations and from abstract inferences.

In spite of a long history of brilliant cogitations, psychopathological nosology still resembles Ptolemy's astronomy of over 2,000 years ago: Our diagnostic categories describe, but they do not really explain. Like so many crystalline spheres, each lies in its own orbit, for the most part uncoordinated with the others. We do not know why the universe takes its ostensible form. There is no law of gravity that undergirds and binds our psychopathological cosmos together. In fact, the word "cosmos" implies an intrinsic unity, a laudable ideal, which is not appropriate in its usage here: Our "star charts," our DSMs and ICDs, remain aggregations of taxa, not true taxonomies. Because of their reliability but dubious validity, our field possesses the illusion of science but not its substance. Such a state of affairs is simply unscientific.

Our most radical (albeit reactional) alternative would be to discard taxonomies altogether. This, of course, would be impossible, as a taxonomy serves indispensable clinical and scientific functions. Clinically, it provides a means of organizing pathological phenomena—the signs and symptoms or manifestations of mental disorder. By abstracting across persons, a taxonomy formalizes certain clinical commonalities and relieves the clinician of the burden of conceptualizing each patient sui generis, as an entity so existentially unique it has never been seen before, nor ever will be seen again. For psychopathology to be practiced at all, there cannot be as many groups as individuals. Even if the formal categories that constitute a taxonomy are but convenient fictions of dubious reality, some groups are better than no groups at all.

Despite any existential disenchantment, personological taxonomists may take a lesson from mystics, both as a point of contrast and as a point of departure. This particular metaphor helps us realize and assert our goals: In short, we want what mystics have (or say they have). We want a clear vision; we want freedom from confusion. Our greatest dream is one of almost mystical insight, wherein our representational blinders are removed and the inner essences of reality are revealed—for the purposes of this chapter, the substantive structural and functional variables that constitute personology and its nexus with psychopathology.

Our scientific sensibilities, however, inform us that the actual mystical experience may not be all that we wish for. It almost invariably resists representation, perhaps actively so, proving ultimately too numinous and ineffable to articulate. Science, of course, cannot afford the luxury of being numinous and ineffable. Science depends on self-conscious knowledge. What is numinous to mystics is vague to scientists. Whereas mystics comprehend nature in its totality as a radically open system of seamless unity, scientists must create artificially closed relational systems. We are wedded to representational systems, including taxonomies—so wedded, in fact, that the abandonment of all representational schemas would be an abandonment of knowledge itself. In the best of all possible worlds, of course, we would have *both* the experience of true seeing *and* a representational system with which to articulate it. Such is the holy grail of a taxonomy of psychopathology or personology, and only such a taxonomy "should be viewed as having objective existence in nature" (Hempel, 1965)—that is, as carving nature at its joints or affording a sense of communion that goes beyond intervening variables and construct systems. Such a sense of communion is the only true validity, that which comes from an intuition of nature as it is. A scientist born thinking within such a taxonomy might never become conscious of the representational aspect; the taxonomy would be completely transparent. Whether such a taxonomy exists, or

whether it must remain an ideal which all actual taxonomies will fall short of in various degree, only just such a taxonomy will prove ultimately scientifically satisfying for psychopathology and personology, and ultimately satisfying to researchers and clinicians who must work inside it.

The metatheoretical logic and scientific study of classification are of relatively recent origin and have been given their most important impetus in several major works, notably those by Simpson (1961) and by Sokal and Sneath (1963). Both these volumes were oriented to the application of quantitative methods in biological taxonomies by setting forth explicit principles and procedures to achieve scientific goals, such as interjudge reliability and external validity. Of no less importance was a seminal article by Hempel (1961) in which he specifically addressed psychopathologists; with unerring logic, it served not only to raise conceptual considerations involved in developing productive taxonomies, but to alert clinicians to the key role that theoretical clarity and empirical synthesis must play. Before these splendid and influential contributions, psychopathological classification reflected belief systems that were based on impressionistic clinical similarities; most were not grounded in quantifiable data, used unrepresentative populations, and were devoid of a cohering theory.

Psychopathology is an outgrowth of both psychology and medicine. As such, efforts to construct a taxonomy must contend with the goals, concepts, and complications inherent in both disciplines (e.g., context moderators, definitional ambiguities, overlapping symptomatologies, criterion unreliabilities, multidimensional attributes, population heterogeneities, instrument deficits, and ethical constraints).

As already noted, the current state of psychopathological nosology and diagnosis resembles that of medicine a century ago. Concepts remain overwhelmingly descriptive. To illustrate, the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III; American Psychiatric Association, 1980) was not only formulated to be atheoretical, but addressed itself exclusively to observable phenomena. It is not that inferences and theory have failed in the past to provide useful knowledge, but that segments of the mental health profession have not been convinced of their scientific utility, at least not sufficiently to use this knowledge for a nosology. On the other hand, despite the fact that much observation and experimentation have been done, the significance of their products lacks an adequate consensus. Thus we remain unsure today whether to conceive depression as a taxon (category) or an attribute (symptom), whether to view it as a dimension or as a set of discrete types, or whether to conceive it as a neuroendocrinological disease or as an existential problem of life. Although debates on these issues often degenerate into semantic arguments and theoretic hairsplitting, it is naive to assume that metaphysical verbiage and philosophical quibbling about words are all that these debates involve. Nevertheless, the language we use, and the assumptions it reflects, are very much a part of our scientific disagreements.

In this chapter, I hope to illustrate at least one point—namely, that philosophical issues and scientific modes of analysis must be considered in formulating a psychopathological taxonomy. Arguably, the following considerations will not in themselves reveal clear resolutions to all nosological quandaries. Their more likely role will be to unsettle prevailing habits and thereby to force us to progress, if for no other reason than because our cherished beliefs and assumptions have been challenged. Logic necessitates that psychopathological states and processes be distinguished from one another and be thereby categorizable to some degree before they can be subjected to identification and quantification.

Conceptual Issues

What exactly is a clinical attribute? That is, what constitutes a taxon, and on what grounds shall we decide that it exists? To pose this question is not mere sophistry. For example, a serious issue demanding the attention of responsible philosophers, psychiatrists, and psychologists (Medin, Altom, Edelson, & Freko, 1982; Smith & Medin, 1981) is the very nature of the construct "disease entity." Noting the complexities inherent in conceiving so universal a concept as "disease," Kendell (1975) wrote that

> contemporary writers still make frequent reference to "disease entities"—almost invariably without defining their meaning. Like "disease" itself, "entity" has become one of those dangerous terms which is in general use without ever being defined, those who use it fondly assuming that they and everyone else knows its meaning. (p. 65)

Feinstein (1977) noted the circular nature of many definitions in the following illustration:

> The complex situation we have been contemplating can be greatly simplified if only we are allowed a bit of circular reasoning. At the root of the difficulty is the problem of deciding what is a diagnosis. We can dispose of that difficulty by defining a diagnosis as the name for a disease. We are now left with defining disease, which we can call a state of abnormal health. Abnormal health is readily defined as a departure from normal health. And then, completing the circle, normal health can be defined as the absence of disease. (p. 189)

As Feinstein went on to say, "disease" is an abstract and multifaceted concept that remains so even if one prefers to represent it with another term, such as "ailment," "sickness," "illness," or "disorder."

Those who have more than a tangential interest in the nature of psychopathological classification cannot help being apprehensive over the profound problems that arise in merely defining its constituents, no less in their specification, criteria, and measurement. I now survey the extent to which constructs such as "taxa" and "attributes" have tangible empirical referents. The basic issue is whether these concepts are anchored to observable or to inferred phenomena.

The constituents of a nosological classification are represented by a set of terms or labels—that is, a language by which members of a clinical group communicate about a subject. These terms serve two functions. First, they facilitate the manipulation of ideas. Clinical or theoretical concepts are systematically (if implicitly) linked; it is through their interplay that meaningful clinical ideas are formulated and deductive scientific statements are proposed. Second, most concepts possess an empirical significance; that is, they are linked in some way to the observable world. Although some may represent processes or events that are not apparent, they can be defined by or anchored with reference to the explicit and tangible. It is this translatability into the empirical domain that allows nosologists to test their schemas in the clinical world.

Ideally, all of the concepts that constitute a nosology ought to be empirically anchored (i.e., to correspond to observable properties in clinical practice); this minimizes confusion about the attributes of which a taxon is composed (Schwartz, 1991). Furthermore, nosological labels must be more precise and clinically descriptive than the words of ordinary language. Although everyday language has relevance to significant real-world events, it gives rise to ambiguity and confusion because of the varied uses to which conventional words are often put. Taxonic concepts must be defined as precisely and with as much clinical relevance as possible, in order to assure that their meaning is clear and pertinent.

Empirical precision can be achieved only if every defining feature that distinguishes a taxon is anchored to a single and observable phenomenon; that is, a different datum will be used for every difference that can be observed in the clinical world. This goal is simply not feasible, nor is it desirable (for reasons to be noted shortly). Classificatory terms do differ, however, in the extent to which they achieve empirical precision. There are points along a gradation from conceptual specificity to conceptual openness that may be identified; doing so may aid readers to recognize certain features that distinguish among both attributes and taxa.

Some concepts are defined literally by the procedures that measure observable events, and they possess no meaning other than the results obtained in this manner. They reflect what Bridgman (1927) termed "operational definitions." The meaning of an operationally defined concept becomes synonymous with how we measure it, not with what we say about it. Its scientific advantage is obvious: Taxa and the attributes of which they are composed are unambiguous, and diagnostic identifications associated with these taxa are translatable directly into the clinical attributes they represent.

Useful as these definitions may be, they present several problems (Schwartz & Wig-

gins, 1986; Spitzer, 1990). Diagnostic terms must be generalizable; that is, they ought to enable clinicians to include a variety of measures and observations as gauges of a taxon. Operational definitions are too restrictive. They preclude extensions to new situations that are even slightly different from the original defining condition. One of the primary goals of a classification is to integrate diverse observations with a minimum number of terms. A strict operational approach floods clinicians with an infinite number of attributes and taxa, and clutters their thinking with largely irrelevant distinctions.

Intrapsychic processes and dispositional traits are nebulous and concealed, hidden from the observable world, and hence only to be inferred. These unobservable mediating structures and processes are not merely useful, but may be necessary elements in constructing an effective psychopathological nosology. Because of their abstract and hypothetical character, these indeterminate constructs are known in philosophy as "open concepts" (Pap, 1953). Some are defined by, and largely reducible to, a set of diverse empirical events. For example, the concept of "projection" may be gauged by observing persons ascribe their own traits to others, by the presence of certain scores on a psychological test, by a history of litigious actions, and so on. Although the term "projection" implies a concealed intrapsychic process within a person that cannot itself be observed, its existence may be inferred from a variety of observables.

Many open and fully speculative concepts are formulated with minimal or no explicit references. Their failure to be anchored to the realm of observables has led some to question their suitability in scientific contexts. No doubt clarity gets muddled, and deductions are often tautological, when a diagnosis is explained in terms of a series of such constructs. For example, such statements as "In an individual with borderline personality disorder, the mechanisms of the ego become diffused when libidinous energies overwhelm superego introjections" are at best puzzling. Postulating connections between one set of open concepts and another may lead to facile but often confusing clinical statements, as any periodic reader of contemporary psychoanalytic literature can attest. Such use results in formulations that are difficult to decipher because one cannot specify observables by which the formulations can be anchored or evaluated.

Open concepts usually assume their meaning with reference to a theoretical network of variables and constructs within which they are embedded. The significance they derive thereby accounts for both their weaknesses and their strengths as components of taxonomy (Spitzer, 1990). Because classification is a human artifact, not every one of its terms needs to be linked to observable events, especially if its purpose is to extend the generalizability of knowledge (Dougherty, 1978). Unrealistic standards of empirical anchorage, particularly in the early stages of taxonomic construction, often discourage the kind of imaginative speculation necessary to decode and to integrate elusive phenomena. Vague and risky as open concepts may be, they prove among the most useful tools available in developing a productive classification.

Clinical Attributes

What data from the stream of ongoing clinical events and processes ought to be selected to serve as the basic units of taxa? Must we restrict ourselves only to observables, or will inferred processes be permitted? Ought the data to consist of behaviors only, or will self-reports or physiological signs be admissible? What of past history or of socioeconomic or situational factors? Is everything grist for the taxonic mill? Must the attributes of diagnostically comparable syndromes be uniform (i.e., consist of the same class of data), or will biological indices be included only in some, cognitive processes only in others, and so on? What structural framework shall we use to organize the components of the taxonomy, and what rules will govern the taxa into which its defining features will be placed? Shall its overall architecture be horizontal, vertical, or circular in format, and shall its elements constitute categories or dimensions? Shall we construct or select its elements on the basis of formal statistical techniques of analysis, such as numerically derived clusters, or shall we turn to theory and choose an element on the basis

of logically deduced constructs? Such questions as these, and the issues and alternatives they raise, are central to the taxonomic enterprise—considerations of a formal character that have only recently been examined in constructing psychopathological nosologies.

Psychopathology has been studied from many vantage points; it has been observed and conceptualized in legitimately different ways by behaviorists, phenomenologists, psychodynamicists, and biochemists. No point of observation or conceptualization encompasses all of the complex and multidimensional features of psychopathology. Clinical processes and events have been described in terms of conditioned habits, reaction formations, cognitive expectancies, or neurochemical dysfunctions. These domains cannot be arranged in a hierarchy, with one level viewed as reducible to another (Millon, 1990; Sartorius, 1990). Neither can they be compared in terms of some objective truth value. Alternative substantive domains are merely different; they facilitate the observation and conceptualization of different clinical attributes, and lead therefore to different taxa. The point is that taxa may be differentially composed in accord with the kinds of clinical data (e.g., etiology, symptoms, and treatment response) they include as their basic constituents. Choices are often pragmatic, and questions of comparative utility cannot be determined a priori. However, irrelevant controversies and needless confusions can be avoided if the class of attributes from which taxa are composed has been specified clearly. When this has been done properly, clinicians and researchers can determine whether two taxonomies are comparable, whether the same diagnostic label refers to different clinical phenomena, whether different taxa encompass the same attributes, and so on. Panzetta (1974) stated:

> As one begins to consider the variety of starting-off points then we quickly appreciate that the first step in the nosologic process is inherently arbitrary. I would insist that we begin by acknowledging that the arbitrary focus is not, per se, a deficiency but rather a reality which flows naturally from the tremendous complexity of human behavior. It is useless to try to develop the "correct" initial focus. There is no correct focus, only several alternatives. (p. 155)

Sneath and Sokal (1973) recorded the wide array of attributes available in every discipline that can be potentially useful in forming a taxonomy. The task is that of identifying which are likely to be most relevant and optimally productive. The history of psychopathology provides guidelines that may prove fruitful—for example, the well-established and important distinction between longitudinal and concurrent attributes. The former represent the progression of various clinical phenomena across time and circumstance; the latter represent the diverse ways in which these phenomena manifest themselves contemporaneously and simultaneously across a number of expressive dimensions.

Longitudinal Attributes

Data that concern causal factors as clinical attributes for taxa would be extremely useful, if only such knowledge were available. Unfortunately, etiological data are scanty and unreliable. Moreover, they are likely to remain so because of the obscure, complex, and interactive nature of influences that shape psychopathological phenomena. In great measure, etiological attributes are conjectures that rest on tenuous empirical grounds; most reflect schools of thought that emphasize their developers' favorite hypotheses. These speculations are best construed as questions that deserve empirical testing on the basis of taxa formed by less elusive data.

Among the few etiological schemas that lend themselves to taxonomic goals is Zubin's (1968) differentiation of six categories: sociocultural (ecological), developmental, learning, genetic, internal environment, and neurophysiological. Systematic and orderly though models such as Zubin's may be, they do not fully address issues associated with alternate levels of analysis; nor do they trace the intricate and varied causal chains that unfold ultimately into a clinical state.

Beyond the issue of identifying which of several levels of an interactive causal system may be selected as etiological attributes, there are questions of a more philosophical and methodological nature concerning what exactly is meant by "etiology" and in what precise way it may be gauged. Meehl (1972) addressed this matter succinctly:

> A metatheoretical taxonomy of causal factors and a metataxonomy of causal relations (such as "necessary but not sufficient condition," "interaction effects," "threshold effects," and the like) are badly needed. In medicine, we recognize several broad etiologic classes such as deficiency diseases, autoimmunity diseases, disease due to microorganisms, hereditary-degenerative diseases, developmental anomalies, diseases due to trauma. . . . The concept "specific etiology" . . . appears to have half a dozen distinguishable and equally defensible meanings (e.g., sine qua non, critical threshold, uniformly most powerful factor) that might be useful under various circumstances. (p. 22)

The yearning among taxonomists for a neat package of etiological attributes simply cannot be reconciled with the complex philosophical and methodological issues and the difficult-to-disentangle networks of both subtle and random influences that shape our mental disorders. It also makes understandable the decision of the DSM-III Task Force to set etiological and course variables aside as clinical grist for its taxonic mills.

When we turn from the antecedent to the consequent side of the clinical course, logic argues that the nature of a mental disorder must be at least partially revealed by its response to treatment. The data available on this matter, however, provide little that goes beyond broad generalizations. This contrasts with medicine at large, in which a variety of interventions are specific to particular disorders. Notable in medicine are a variety of challenge or stressor tasks that serve to identify patient vulnerabilities (e.g., introducing allergens to uncover susceptibilities or to elicit reaction sensitivities). Considerations of ethics and human sensibilities preclude adopting parallel strategies in psychopathology. Even where the intent is clearly beneficial, as in psychological treatment interventions, the problems encountered in discerning and differentiating optimal clinical attributes are many—for example, the inevitability of spontaneous remissions and unanticipated life events. Even if we could partial out the effects of these confounding events, could we identify the ingredients that account for beneficial reactions in a heterogeneous patient population? Furthermore, what conclusions can be drawn about a therapy whose efficacy is wide-ranging (i.e., demonstrable among disorders whose origins and expression are highly diverse)? Complicating matters further is the fact that every technique of therapy claims high efficacy across a wide band of sundry diagnostic classes. We may then conclude that treatment response, as with etiology, may prove ultimately to be a useful taxonic attribute; for the present, however, reliable data are not in hand.

Concurrent Attributes

Given the problematic nature of longitudinal attributes, what remain to constitute psychopathological taxa are coexistent attributes of a contemporaneous nature—notably, objective signs on the one hand, and subjectively reported symptoms on the other. To these two classical indicators of disorder may be added the essentially inferred attributes of personality traits. Information about these clinical features may be derived from four conceptually and methodologically distinct data sources: namely, the biophysical, intrapsychic, phenomenological, and behavioral.

Signs

"Signs" consist of more or less objectively recorded changes in state or function that indicate both the presence and character of clinically relevant processes or events. Two sources provide the main body of clinical signs: biophysical markers and behavioral acts.

As for the first data set, biophysical markers, there are few anatomical, biochemical, and neurophysiological gauges among the standard diagnostic taxa, despite their tangible, objective, and quantitative nature. This failure is not only surprising but disconcerting, given the vast number of psychopathological studies over the years that have hypothesized and investigated potential biophysical markers obtained from a multitude of measures (urine or blood analysis, diverse muscle appraisals, skin gauges, cardiac assessments, eye movements, metabolic indices, electroencephalographic rhythms, etc.).

That so few of these biophysical measures have aided the definition of clinical attributes can be traced to a number of factors. Most cannot be arranged into clearly discriminable categories, and with few exceptions, normative distributions on relevant clinical populations are either unavailable or incon-

sistent. For many, reliability measures are lacking or indicate high levels of variability over time and across settings. To complicate interpretive efforts, low intercorrelations are typically found among measures that ostensibly represent the same basic functions. Moreover, the expense of technical equipment is high, and the availability of needed expertise is often so scarce as to place many of these procedures out of reach for all but well-funded research investigators.

As for explicit behavioral acts, the second of the major objective indices, it has been the methodological goal of behavioral purists to avoid drawing inferences about internal or subjective processes. Hence they seek to use techniques that ostensibly bypass explicit dependence on subjective symptomatic data. As promising as overt behavior may be as a source for clinical attributes, numerous concerns about its utility have been registered, not the least of which is its unpredictability—that is, its high variability across setting and time (e.g., the recording of verbal behaviors fails to generate reliable normative data across diverse circumstances). This is in contrast with data produced by well-constructed rating instruments. Most of these are reasonably reliable, succeed in discriminating among relevant patient groups, and possess adequate normative data for differential or comparative purposes.

Rather commonplace behaviors of potential diagnostic significance have come under systematic scrutiny and analysis. Buss and Craik (1983, 1987) and Livesley (1985, 1991) have attempted to provide a descriptive (i.e., nonexplanatory) basis for diagnostically relevant features; these investigators have developed lists of familiar acts observed in the course of everyday life that may typify certain clinical characteristics. Despite these promising advances, behavioral methods are long on neat measures of a rather trivial character, with tangential or limited substantive significance in the realm of psychopathological taxonomy (Block, 1989).

Symptoms

In contrast to clinical signs, "symptoms" are subjective in nature, represented by reports from patients of their conscious recollections and recorded experiences (e.g., moods, feelings, perceptions, memories, attitudes). Together with clinical signs, symptoms focus on phenomenological processes and events that relate directly to diagnostic matters. It is here where the psychopathologist has an advantage over the physicist or the biologist, for neither can ask the objects of their study to reflect on their experience, no less to communicate it in articulate or meaningful ways. Strauss (1986) framed it thus:

> To some extent, the field has been discouraged by previous claims and promises regarding the understanding of subjective experiences such as these. Although the claims were often overstated, and the assessment of these processes is complex, neither reason is adequate for avoiding a major attempt to develop creative ways for looking at the role of such subjective experiences in the course of psychopathology and their relevance to diagnosis. (p. 262)

Contributing to the utility of symptomatic data is their ease of evocation. Moreover, structured assessments, such as interview schedules and self-report inventories, minimize potential sources of distortion. Nevertheless, these methods are subject to difficulties that can invalidate data (e.g., one cannot assume that subjects will interpret questions in the same way, that they possess sufficient self-knowledge to respond informatively, or that they may not be faking or dissembling).

Although the substantive contents of phenomenological symptoms are elusive and often unreliable and are fraught with philosophical and methodological complexities, taxonomists cannot afford the luxury of bypassing them. Symptoms lie at the very heart of all psychopathological inquiries. The events they portray are real and represent facets of experience far richer in scope and diversity than concrete observables.

Traits

In contrast to signs, which represent objective biological measures or behavioral acts, and symptoms, which are phenomenologically reported recollections and experiences, "traits" include inferred psychological habits and stable dispositions of broad generality and diverse expression. This long-established psychological construct has been used in two ways. First, it encompasses various characteristic habits, moods, and attitudes; second,

through inference it identifies dispositions to act, feel, and think in certain ways. Traits can be considered to be both more and less than signs and symptoms. For example, several traits can coalesce to form the expression of a single behavioral sign. Conversely, several different specific symptoms may be the upshot of a single trait. Furthermore, each trait may express itself in diverse signs and symptoms. Clearly, there is no one-to-one correspondence between traits and signs or symptoms.

Traits are often inferred rather than observed, generalized rather than specific, and dispositional rather than consequential. They are assumed to be enduring and pervasive. However, only certain traits of a person display this durability and pervasiveness; that is, only some of them prove to be resistant to the influences of changing times and circumstances. Other forms of behavior, attitude, and emotion are presumably more transient and malleable. It is noteworthy that the traits exhibiting consistency and stability in one person may not be the same as those in others. These qualities are most prominent among characteristics that are central to maintaining a person's overall psychological balance and style of functioning. To illustrate, the interpersonal conduct trait of significance for some is that of being agreeable, never differing or having conflict; for others, it may be interpersonally important to maintain one's distance from others so as to avoid rejection or humiliation; for a third group, the influential interpersonal trait may be that of asserting one's will and dominating others.

The sources used to identify clinical traits are highly diverse. They range from methods designed to uncover intrapsychic processes, such as free association, dream analysis, hypnosis, and projective techniques, to such phenomenological methods as structured interviews and self-report inventories, and to behavioral methods of observation and rating (be they systematic or otherwise).

It is no understatement to say that the rich vein of clinical attributes uncovered by dispositional and intrapsychic traits has been a boon to clinical theory, but a source of perplexity and despair to taxonomists. More than any other domain, dispositional data and methods produce information fraught with complexities and obscurities that can bewilder the most sophisticated of classifiers. Part of the difficulty stems from the fact that the identification of hidden traits is highly inferential. Because the dispositional structure and processes that make up traits can be only partially observed and take different manifest forms in different contexts, it is difficult to identify them reliably, and hence to assign them a standard place in a taxonomy. Matters are made more difficult by the absence of intrapsychic normative and base rate data, as well as by the lack of strong validational support.

Structural Models

Whatever features are chosen to provide the substantive body of a taxonomy, decisions must be made about the structural framework into which the taxonomy will be cast, the rules that will govern the taxa into which its clinical attributes and defining features will be placed, and the compositional properties that will characterize these attributes and features. These are problems of the essential architecture of the taxonomy: whether it should be organized horizontally, vertically, or circularly; whether all or only a limited and fixed subset of features should be required for taxonic membership; whether its constituents should be conceived as categories or dimensions; and which of a host of other differentiating characteristics one should choose. I discuss several modern structural designs, and the options available among them—a task of no simple proportions, given that nothing is logically self-evident and that there is no traditional format or contemporary consensus to guide selections among these alternatives.

Taxonomic Structure

Ought the various attributes that make up the substantive data of psychopathology to be listed more or less randomly, or should they be ordered into a series of logical or functional groups that attempt to mirror the inherent nature of psychopathology? The obvious answer is the latter.

Several frameworks for structuring psychopathology have been formulated in recent years, and they are not mutually exclusive. From a design viewpoint, they can

be described as having vertical, horizontal, or circular structures. The vertical or "hierarchical" framework organizes the various taxa of psychopathology (e.g., depressive disorders or schizophrenic disorders) in a series of echelons in which lower tiers are subsumed as subsets of those assigned higher ranks. The second or "horizontal" framework is known as the "multiaxial" schema; it orders different classes of attributes (e.g., symptoms or etiologies) in a series of aligned or parallel categories. Since the publication of DSM-III, the DSMs have encompassed both hierarchical and multiaxial structural forms, albeit with modest logic and success. The circular framework is referred to as the "circumplical" model. It has not received official recognition, although it has gained considerable currency among theorists who emphasize the role of interpersonal attributes.

Hierarchical Models

Hierarchical models are typically arranged in the form of taxonic decision trees. Once a particular branch (i.e., a higher-order diagnosis) has been chosen, subsequent taxonic choices are limited to the several branches and twigs that constitute subdivisions. To illustrate, once it has been decided with DSM-IV-TR (American Psychiatric Association, 2000) criteria that a patient is exhibiting a mood disorder, the clinician may further differentiate the disturbance as either a bipolar disorder or a depressive disorder. If the choice is bipolar, the clinician may move further down the hierarchy to select among bipolar I disorder (manic, hypomanic, mixed, or depressed episode), bipolar II disorder (hypomanic or depressed episode), cyclothymic disorder, or bipolar disorder not otherwise specified.

A consequence of so carefully fashioned a sequential chain of categories is that successive taxa in the classification are invariably more specific and convey more precisely differentiated information than those that precede them. This increasing distinctness and exactitude—necessary ingredients in a successful hierarchical schema—assures that each successive category possesses authentic clinical features not found in categories previously listed.

Sequential patterns of the decision tree type would be a remarkable achievement for any hierarchical nosology, if they were naturally or logically justified (Millon, 1983). Not only is there no inherent structure to psychopathology that permits so rigorous an arrangement, but the various DSMs, for instance, impose only a modest degree of sequential rigor on the taxonomic organization. The problems of pursuing a hierarchical method for differential diagnosis are compounded by the fact that modern DSMs not only permit but encourage multiple diagnoses—a problem aggravated further by the manuals' standard multiaxial framework. Not only does the hierarchical goal of orderly and successive diagnostic choice points run hard against the structural character of DSM taxa, but its formalism and sequential requirements are undermined repeatedly by the multidiagnostic aims and intrinsic multiaxial schema of the DSMs.

Multiaxial Models

The multiaxial format, the second of the overarching structural models, encounters few of the logical difficulties and assumptions found in hierarchical systems. The formal adoption of the multiaxial schema in DSM-III and ICD-10 (World Health Organization, 1992) approached a paradigm shift (Millon, 1983). It reflected a distinct turn from the traditional infectious-disease model, in which the clinician's job is to disentangle distracting symptoms and to clear away confounding situational problems so as to pinpoint the underlying or true pathophysiological state. By contrast, the multiaxial model (Essen-Moller & Wohlfahrt, 1947; Mezzich, 1979; Williams, 1985a, 1985b) not only recognizes that distracting and confounding circumstances are aspects worthy of attention, but encourages recording them on their own representative axes as part of an interactive complex. The multiaxial structure aligns many of the potentially relevant factors that can illuminate the nature of a clinical condition, and it provides a means of registering their distinguishing attributes. In contrast to the more traditional hierarchical model, in which a single class of attributes (signs or etiologies) is differentiated, the multiaxial format per-

mits multiple classes of data (again, signs and etiologies) and thereby encourages diagnostic formulations that include several facets of information relevant to clinical decision making.

The very comprehensiveness of the multiaxial model can prove to be its undoing, however. Such systems provide a more thorough picture than do schemas of unitary axes—but they are therefore also more complicated and demanding to implement, and require a wider band of data and a greater clarity and diversity of judgments than clinicians are accustomed to performing. They often impose a procedural complexity on an otherwise expedient process. From a pragmatic view, a fully comprehensive multiaxial assessment may be an unnecessary encumbrance in routine diagnostic work, impractical for everyday decision making, and abhorrent to clinicians accustomed to the diagnostic habit of intuitive synthesis.

Circumplical Models

Circumplical models have been used in the arrangement of both taxa and attributes. In neither case have they been recognized in formal psychopathological taxonomies; rather, their primary use has been as a structural tool for ordering interpersonal traits (Benjamin, 1974, 1986, 2006; Lorr, 1966), most notably in conjunction with personality processes and disorders (Kiesler, 1983, 1996; Leary, 1957; Pincus & Wiggins, 1990; Plutchik & Platman, 1977; Sim & Romney, 1990; Strack, 2005; Strack, Lorr, & Campbell, 1990).

Circumplical models are structured so as to locate similar taxa in adjoining or nearby segments of a circle; taxa located diametrically on the circle are considered psychologically antithetical. Plutchik and Conte (1985) provided evidence that emotions, traits of personality, and personality disorders line up in parallel ways on a circumplex, which suggests that this structure can arrange diverse concepts into a common framework and lead thereby to the identification of relations that may otherwise not be recognized. As interesting as this formulation may be for organizing conceptual categories, the circumplex appears at present to be essentially an academic tool of theoretical rather than clinical value, despite indications that promise the latter as well.

Taxonic Structure

Taxonic units in psychopathology may be monothetic or polythetic in structure. All of the attributes that constitute a monothetic taxon must be in evidence for a diagnosis to be correctly made. In polythetic taxa, various and different optional subsets of the full attribute list can suffice to justify a diagnosis.

Classical versus Prototypal Taxa

Classical taxa comprise categories made up of discrete entities that are homogeneous with respect to their defining features—that is, arranged in a restrictive, monothetic format (Cantor & Mischel, 1979; Rosch, 1978). Failures to identify all of the attributes of a taxon can result from obscuring and confounding conditions and/or from deficits in observational technology and skill.

Frances and Widiger (1986) characterized the major features of and difficulties with classical taxa as follows:

> The classical model of categorization conceives of disorders as qualitative, discrete entities and assumes that the defining features are singly necessary and jointly sufficient, that the boundaries between categories are distinct, and that members are homogeneous with respect to the defining features. . . . Although the classical model works well for abstract categories (e.g., "square"), it fails to do justice to the complexity of naturally occurring taxonomic problems. All squares share the features of having four equal sides joined at right angles . . . [but] actual objects, plants, animals, and persons, however, often fail to share a set of singly necessary and jointly sufficient features. . . . If a classical typology is an inappropriate model for classification of objects, birds, and plants, it is clearly inappropriate for psychiatric diagnosis. (p. 392)

Horowitz, Post, French, Wallis, and Siegelman (1981) described the contrasting prototypal construct succinctly:

> A prototype consists of the most common features or properties of members of a category and thus describes a theoretical ideal or stan-

> dard against which real people can be evaluated. All of the prototype's properties are assumed to characterize at least some members of the category, but no one property is necessary or sufficient for membership in the category. Therefore, it is possible that no actual person would match the theoretical prototype perfectly. Instead different people would approximate it to different degrees. The more closely a person approximates the ideal, the more the person typifies the concept. (p. 575)

The prototypal structure assumes a measure of taxonic heterogeneity, and hence is likely to require a polythetic format. Its open and permissive taxonic structure is more consonant with the natural fuzziness of conceptual boundaries (Cantor & Genero, 1986; Osherson & Smith, 1981; Schwartz, Wiggins, & Norko, 1989; Wittgenstein, 1953), as well as the inherent inexactness of natural reality (Meehl, 1978; Millon, 1969, 1987, 1990). Referring to the classical approach, Widiger and Frances (1985a, 1985b) have written that once patients are placed in the same taxon, there is a tendency to exaggerate their similarities, ignore their differences, and focus on the stereotypic features that distinguish the category, all at the expense of bypassing disconfirming traits and downplaying idiosyncratic behaviors. By contrast, they have stated that polythetically constructed taxa limit stereotyping, permit diagnostic flexibility, and encourage within-groups variability. On the other hand, no pathognomonic signs will be present; persons similarly diagnosed will vary in their degree of prototypicality; and defining features will differ in their diagnostic efficiency.

Categorical versus Dimensional Taxa

My discussion of structural alternatives has progressed from choices among three taxonomic configurations (hierarchical vs. multiaxial vs. circumplical) to a selection between two taxonic structures (classical vs. prototypal). I continue this progression toward increasing specifics, addressing the choices that need to be made with concern for the compositional character of taxa and attributes. Here the issue is raised as to whether taxa and their attributes ought to be conceived qualitatively (categorically) or quantitatively (dimensionally). Thoughtful papers on the issue will be found in Chapters 15, 17, and 18 later in this book.

For monothetic (classical) taxa, authors have asked whether clinical syndromes (e.g., dysthymia) ought to be conceived as qualitatively discrete categories, or whether they ought to be conceived on a quantitative dimension of severity (Frances, 1982). The issue among those who arrange polythetic rules (usually prototypal) is whether different combinations of defining features can be conceived as forming quantitative variations of the same qualitative category. This latter approach focuses not on the taxa themselves, but on their clinical attributes and defining features. For this issue, each attribute is conceived as a quantifiable dimension along such lines as salience or severity. What is categorized is not the taxon, but the several variants of a clinical attribute (e.g., if interpersonal conduct is a relevant attribute for diagnosing personality disorder taxa, then choices may be made first among the several interpersonal defining feature options, such as aversive, seductive, or secretive). Second, once the interpersonal options have been chosen (qualitative categorization), each may be given a score to represent its degree of salience or severity (quantitative dimensionalization).

The first of the two approaches to the issue is related to categorical versus dimensional taxa. The issue applies especially to clinical attributes, for which the trend toward prototypal models and polythetic definitions is more central. The taxonic issue may be stated in the form of a question: Ought taxa to be conceived and organized as a series of dimensions that combine to form distinctive profiles for each person, or should certain characteristics found commonly in clinical populations be selected to exemplify and classify taxa (Livesley, 1991)?

Dimensional conceptions emphasize quantitative gradations among persons, rather than qualitative, discrete, all-or-none class distinctions. To illustrate, Kendell (1968) proposed that a single dimension might suffice to represent the continuum he found between neurotic and psychotic depressions. By contrast, and consistent with the clear boundaries between taxa expected with a categorical schema, Paykel (1971) found minimal overlapping among four classes—

those of psychotic, anxious, hostile, and depression in young persons.

Several advantages to dimensional models may be noted. First, they combine several clinical attributes (or their defining features) in a single configuration. This comprehensiveness results in a limited loss of information, and no single attribute is given special significance, as is the case when only one distinctive characteristic is brought to the forefront. Dimensional profiles also facilitate the assignment of unusual or atypical cases. In categorical formats odd or mixed conditions are often excluded because they fail to fit the prescribed criteria. Given the idiosyncratic character of many clinical conditions, a dimensional system permits representation and assignment of interesting and unique cases without forcing them into Procrustean categories for which they are ill suited. A major advantage of the dimensional model is that the strength of its constituent features is gauged quantitatively, wherein each characteristic extends into the normal range. As a consequence, normality and abnormality are construed as points on a continuum rather than as distinct and separable phenomena.

Despite these advantages, dimensional taxa have not fared well in psychiatric classifications (Gunderson, Links, & Reich, 1991). Numerous complications have been noted in the literature; for example, there is little agreement among theorists about the number of dimensions necessary to represent psychopathological phenomena. Menninger (1963) contended that a single dimension suffices; Eysenck (1960) asserted that three are needed; Cattell (1965) claimed to have identified as many as 33 and believed there to be many more. In fact, theorists appear to invent dimensions in accord with their expectations, rather than to discover them as if they were intrinsic to nature, merely awaiting scientific detection. The dimensions required to assess psychopathological phenomena appear to be determined not by the ability of research to disclose some inherent truth, but rather by researchers' predilections for conceiving their investigations and interpreting the findings.

Categorical models are the traditional form used to represent clinical conditions. There are several reasons for this preference. First, most taxa neither imply nor are constructed to be all-or-none categories. Certain features are given prominence, but others are not overlooked; rather, they are merely assigned lesser significance.

The success of categorical taxa may be traced to the ease with which clinicians can use them in making rapid diagnoses with numerous briefly seen patients. Although clinical attention is drawn to only the most salient patient attributes, other, less conspicuous characteristics are often observed, suggested, or inferred. The quality of intimating characteristics beyond the immediately observed contributes to the value of established categorical taxa. The categorical approach's power of extending its scope to associated attributes contrasts with the tendency of dimensional schemas to segment, if not fractionate, persons and disorders into separate components. Categories restore the unity of a patient's pathology by integrating seemingly diverse elements into a single, coordinated configuration. Well-established categorical taxa often provide a standard of reference for clinicians who are otherwise faced with reconstructions or de novo diagnostic creations (Gunderson et al., 1991).

There are objections to the use of categorical taxa. For example, they contribute to the fallacious belief that psychopathological processes constitute discrete entities, even medical diseases, when in fact they are merely concepts that help focus and coordinate our observations. Moreover, categories often fail to identify or include significant aspects of behavior because of the decision to narrow their list to a set of predetermined characteristics. This discarding of information is not limited to categories; dimensional schemas also select certain attributes to the exclusion of others. The problem, however, is that certain categorical schemas give primacy only to one or two attributes. A related criticism is that both the number and diversity of categories in most taxonomies are far less than the clinically significant individual differences observed in everyday practice. Not only are there problems in assigning many patients to the limited categories available, but clinicians often claim that the better they know patients, the greater the difficulty they have in fitting them into a category.

Issues of categoricality versus dimensionality are more properly the province of

attributes than of taxa. For example, the Axis I taxon of depressive disorders really represents a clinical attribute; the distinction between its two major subcategories, major depression and dysthymia, may essentially be a matter of quantitative severity and hence may reflect dimensionality. Furthermore, the distinction between bipolar disorders and depressive disorders may be best conceived as variations in two clinical attributes, not taxa. The former reflects the operation of two mood attributes, mania and depression—each of which may vary as a single dimension, although both may be found in certain persons (bipolar disorders), whereas only one may be exhibited in others (depressive disorders). The fact that some taxa are composed essentially of a single clinical attribute, whereas others encompass several distinct attributes, has not only confounded discussions of categoricality versus dimensionality; it has contributed a share of confusion to theory, research, and practice as well.

To restore order to some aspects of the problem, Skinner (1986) elaborated several hybrid models that integrate elements of these ostensibly divergent schemas. In what Skinner termed the "class–quantitative approach," efforts are made to synthesize quantitative dimensions and discrete categories. I described an endeavor of this nature in a couple of articles (Millon, 1984, 1986). I proposed that an essential aspect of integrating a mixed categorical–dimensional model for personological taxa will be the specification of a distinctive defining feature for each clinical attribute of each personality disorder. If the clinical attribute of expressive mood is deemed of diagnostic value in assessing personality disorders, then a specific defining feature will be identified to represent the distinctive manner in which each personality disorder manifests its emotional feelings. To further enrich the qualitative categories (the several defining features that compose the clinical range of each attribute) with quantitative discriminations (numerical intensity ratings), clinicians will not only identify which features (e.g., distraught, hostile, labile) of a clinical attribute (e.g., expressive mood) best characterize a patient, but also record a number (e.g., 1–10) to represent the degree of prominence or pervasiveness of the chosen defining features. Clinicians will be encouraged in such a prototypal schema to record and quantify more than one defining feature per clinical attribute (e.g., if suitable, to note both distraught and labile moods, if their observations and inferences so incline them). Such a procedure as this illustrates that categorical (qualitative distinction) and dimensional (quantitative distinction) taxonic models need not be framed in opposition to, or be considered as exclusive of, each other.

Construction Methods

How have psychiatric taxonomies come into being? In the main, traditional classifications have been the product of a slowly evolving accretion of clinical experience (Menninger, 1963), which has been fostered and formalized periodically by the systematizing efforts of respected clinician-scholars such as Kraepelin (1899). It may be expected that empirical data or theoretical advances on matters of causality or structure will serve as a primary heuristic impetus, but such has not been the case. With but a few exceptions (e.g., the DSM-I and DSM-II [American Psychiatric Association, 1952, 1968] psychoanalytic explication of neurotic disorders, which was subsequently expunged as an organizing construct in DSM-III), theory-generated or research-grounded taxonomies have fared rather poorly. Another spur to developing classifications has originated in a series of quantitative methods known as "cluster analyses" (Sneath & Sokal, 1973). Time will tell whether these mathematical tools will generate taxa of sufficient consequence to gain acceptance in the clinical world. Describing taxonomic advances in the biological sciences, Sokal (1974) wrote:

> In classification, theory has frequently followed methodology and has been an attempt to formalize and justify the classificatory activity of workers in various sciences. In other instances, classificatory systems have been set up on a priori logical or philosophical grounds and the methodology tailored subsequently to fit the principles. Both approaches have their advantages and drawbacks; modern work tends to reflect an interactive phase in which first one and then the other approach is used, but in which neither principles nor methodology necessarily dominate. (p. 115)

Taxonomic methods in psychopathology are much less advanced than those in the biological sciences, but they are approaching the threshold at which some of the same controversies that occurred in their biological forerunners are likely to arise (Krueger & Tackett, 2006; Strack, 2006). Although each of the alternative construction methods to be discussed shortly may prove fruitful, psychiatric taxonomists are already engaged in debates as to which is best. It is important to recognize that there is no correct choice, and that no rules can be found in nature to confirm which are best or likely to be profitable. To make sense of and give order to the taxonomies they use, clinicians must know what approach to construction was followed and what attributes constituted their database. With the techniques and building blocks of these methods clearly in mind, clinicians may assess them intelligently and judge their relevance to the questions they pose.

A few words must be said about the similarities between methods of taxonomic formation and procedures for developing psychometric tools. Skinner (1981, 1986) has drawn on the logic outlined by Loevinger (1957) for sequentially validating diagnostic tests and applied it creatively to the composition of taxonomies. The mutually reinforcing strength achieved by a combination of Loevinger's three validation strategies may be kept in mind as I elaborate each of the three construction options. In Loevinger's seminal article, she recommended progress from the theoretical to the statistical (internal) to the clinical (external)—a sequence especially suitable to the validation of diagnostic tests, and one that both Jackson (1971) and I (Millon, 1977) followed in fashioning our psychometric inventories. I reverse this sequence to accord with the historical order in which taxonomies have and are likely to continue to be composed.

Clinically Derived Categories

Until recently, psychiatric taxonomies were formed solely on the basis of clinical observation—the witnessing of repetitive patterns of behavior and emotion among a small number of carefully studied mental patients. Hypotheses were generated to give meaning to these patterns of covariance (e.g., Hippocrates anchored differences in observed temperament to his humoral theory, and Kraepelin distinguished two major categories of severe pathology, dementia praecox and manic–depressive disease, in terms of their ostensibly divergent prognostic course). The elements of these theoretic notions were post hoc, however—imposed after the fact, rather than serving as a generative source for taxonomic categories. The most recent examples of a clinical taxonomy, tied explicitly to phenomenal observation and constructed to be both atheoretical and nonquantitative, are of course the DSMs. Spitzer, chairperson of the DSM-III Task Force, stated in DSM-III (American Psychiatric Association, 1980) that "clinicians can agree on the identification of mental disorders on the basis of their clinical manifestations without agreeing on how the disturbances came about" (p. 7).

Despite assertions to the contrary, the recent DSMs are products of implicit causal or etiological speculation. Nevertheless, the DSM-III Task Force sought to eschew theoretical or pathogenic notions, adhering to as strict an empiricist philosophy as possible. Only those attributes that could be readily observed or consensually validated were to be permitted as diagnostic criteria. Numerous derelictions from this epistemology were notable, however, especially among the personality disorders, whose trait ascriptions called for inferences beyond direct sensory inspection.

By no means do all who draw their philosophical inspiration from an empiricist mindset restrict themselves to the mere specification of surface similarities (Medin et al., 1982). Not only those who formulate theoretically generated nosologies succumb to the explanatory power and heuristic value of pathogenic, dynamic, and structural inferences. Feinstein (1977), a distinguished internist, provided an intriguing illustration of how one clinician's factual observations may be another's inferences:

> In choosing an anchor or focus for taxonomy, we can engage in two distinctly different types of nosologic reasoning. The first is to form names, designations, or denominations for the observed evidence and to confine ourselves exclusively to what has actually been observed. The second is to draw inferences from the observed evidence, arriving at inferential

> titles representing entities that have not actually been observed. For example, if a patient says, "I have substantial chest pain, provoked by exertion, and relieved by rest," I, as an internist, perform a denomination if I designate this observed entity as angina pectoris. If I call it coronary artery disease, however, I perform an inference, since I have not actually observed coronary artery disease. If a radiologist looking at a coronary arteriogram or a pathologist cutting open the coronary vasculature uses the diagnosis coronary artery disease, the decision is a denomination. If the radiologist or pathologist decides that the coronary disease was caused by cigarette smoking or by a high fat diet, the etiologic diagnosis is an inference unless simultaneous evidence exists that the patient did indeed smoke or use a high fat diet. (p. 192)

In great part, clinically based taxa gain their importance and prominence by virtue of consensus and authority. Cumulative experience and tradition are crystallized and subsequently confirmed by official bodies. Specified criteria are denoted and articulated, and they acquire definitional if not stipulative powers, at least among those who come to accept the attributes selected as infallible taxonic indicators.

Numerically Derived Clusters

Clinically based categories stem from the observations and inferences of diagnosticians; as such, they constitute, in circular fashion, the very qualities that clinicians are likely to see and deduce. Categories constructed in this manner will not only direct future clinicians to mirror these same taxa in their patients, but may lead future nosologists away from potentially more useful schemes with which to fathom less obvious patterns of attribute covariation. Toward the end of penetrating beneath the sensory domain to more latent commonalities, taxonomists have been led to turn either to numerical methods or to theoretical principles.

Andreasen and Grove (1982) enumerated the advantages of what they termed "empirical" or "numerical" methods for computing patient similarities:

> First, the empirical method gives an opportunity for the observed characteristics of the subjects to determine the classification and perhaps to lead to a classification that the clinician was unable to perceive using clinical judgment alone. Second, the empirical method allows a great deal of information on the subjects to enter into the genesis of the classifications; human beings can keep in mind only a relatively small number of details concerning a case at any given time, but the empirical approach can process very large sets of measurements. Third, empirical or numerical approaches can combine cases in more subtle ways than can clinicians; combinations of features too complex to grasp intuitively may yield better classifications than simple combinations. (p. 45)

There has been a rapid proliferation of powerful mathematical techniques for both analyzing and synthesizing vast bodies of clinical data. This expansion has been accelerated by the availability of inexpensive computer hardware and software programs. Unfortunately, this growth has progressed more rapidly than its fruits can be digested. As Kendell (1975) said, early in this development, "most clinicians . . . have tended to oscillate uneasily between two equally unsatisfactory postures of ignoring investigations based on these techniques, or accepting their confident conclusions at face value" (p. 106).

This growing and diverse body of quantitative methods can be put to many uses, of which only a small number are relevant to the goal of taxonomic construction. Some statistical techniques relate to the validation of existent nosologies (e.g., discriminant analyses) rather than to their creation. Among those used for taxonomic development, some focus on clinical attributes as their basic units, whereas patients themselves are the point of attention for others (Grove & Tellegen, 1991). For example, factor analysis condenses initially diverse sets of clinical attributes and organizes them into potential syndromic taxa. Cluster analysis, by contrast, is most suitable for sorting patient similarities into personological taxa. Reviews of these two numerical techniques, as well as other mathematical procedures for taxonomic construction and evaluation (e.g., latent-class analysis, log-linear analysis, discriminant analysis, and multivariate analysis of variance), may be examined in a number of useful publications (Blashfield, 1984; Grove & Andreasen, 1986; Hartigan, 1975; Kendell, 1975).

Although cluster algorithms have begun to mirror broad diagnostic classes, these slender advances do not answer the question of whether cluster analysis produces categories that resemble the natural structure of psychopathology any better than those of our more traditional or clinically based nosologies. Nor is there any evidence that they provide more accurate predictions of such nonstructural concerns as prognosis and treatment response.

Several authors have summarized the current state of affairs, and have addressed the problems that are likely to persist in the use of numerical construction procedures. For example, Skinner and Blashfield (1982) noted:

> Clinicians have at best given only a lukewarm reception to such classifications. They have been skeptical about the value of clustering methods to identify "naturally" occurring subgroups. Furthermore, the classifications generated by these methods have not seemed Particularly meaningful or relevant to everyday clinical practice. (p. 727)

Kendell's (1975) comment of more than three decades ago, on reviewing the preceding 20-year period, is unfortunately no less apt today than it was then:

> Looking back on the various studies published in the last twenty years it is clear that many investigators, clinicians and statisticians, have had a naive, almost Baconian, attitude to the statistical techniques they were employing, putting in all the data at their disposal on the assumption that the computer would sort out the relevant from the irrelevant and expose the underlying principles and regularities, and assuming all that was required of them was to collect the data assiduously beforehand. (p. 118)

Theoretically Deduced Constructs

In the early stages of knowledge, the categories of a classification rely invariably on observed similarities among phenomena (Tversky, 1977). As knowledge advances, overt similarities are discovered to be an insufficient, if not false, basis for cohering categories and imbuing them with scientific meaning (Smith & Medin, 1981). As Hempel (1965) and Quine (1977) have pointed out, theory provides the glue that holds a classification together and gives it both its scientific and its clinical relevance. In Hempel's (1965) discussion of classificatory concepts, he wrote that

> the development of a scientific discipline may often be said to proceed from an initial "natural history" stage . . . to subsequent more and more "theoretical" stages. . . . The vocabulary required in the early stages of this development will be largely observational. . . . The shift toward theoretical systematization is marked by the introduction of new, "theoretical" terms . . . more or less removed from the level of directly observable things and events.
>
> These terms have a distinct meaning and function only in the context of a corresponding theory. (pp. 139–140)

As Hempel (1965) stated, mature sciences progress from an observationally based stage to one that is characterized by abstract concepts and theoretical systemizations. Contemporary philosophers of science believe that classification alone does not make a scientific taxonomy, and that similarity among attributes does not necessarily constitute a scientific category (Smith & Medin, 1981). The characteristic that distinguishes a latent scientific classification is its success in grouping its elements according to theoretically consonant explanatory propositions. These propositions are formed when certain attributes that have been categorized have been shown or have been hypothesized to be logically or causally related to other attributes or categories. The latent taxa that undergird a scientific nosology are not therefore mere collections of overtly similar attributes or categories, but linked or unified patterns of known or presumed relations among them. These theoretically grounded patterns of relations provide the foundation of a scientific taxonomy.

Several benefits of systematizing clinical data in a theoretical fashion are not readily available from either clinical or numerical procedures (Wright & Murphy, 1984). Given the countless ways of observing and analyzing a set of data, a system of explanatory propositions becomes a useful guide to clinicians as they seek to comprehend the stream of amorphous signs and chaotic symptoms they normally encounter. Rather than shifting from one aspect of behavior, thought, or emotion to another, according

to momentary impressions of importance, theoretically guided clinicians may be led to pursue in a logical and perhaps more penetrating manner only those aspects that are likely to be related (Dougherty, 1978). In addition to furnishing this guidance, a theoretically anchored taxonomy may enable diagnosticians to generate insights into clinical relations they may not have grasped before. Furthermore, it ought to enlarge the sensitivity and scope of knowledge of observers by alerting them to previously unseen relations among attributes, and then guiding these new observations into a coherent body of knowledge.

Taxonomic theories need be neither fully comprehensive nor extensively supported to inspire and guide the early phases of taxonomic development. Meehl (1972) addressed these points with relevance to his concept of the schizophrenia taxon:

> I would not require that a genuinely integrated theory explain everything about schizophrenia, a preposterous demand, which we do not customarily make of any theory in the biological or social sciences. At this stage of our knowledge, it is probably bad strategy to spend time theorizing about small effects, low correlations, minor discrepancies between studies and the like.
>
> Being a neo-Popperian in the philosophy of science, I am myself quite comfortable engaging in speculative formulations completely unsubstantiated by data. To "justify" concocting a theory, all one needs is a problem, plus a notion (I use a weak word advisedly) of how one might test one's theory (subject it to the danger of refutation). (p. 11)

The reader may be taken aback by Meehl's seemingly tolerant views and conclude that theory can lead to scientific irresponsibility—a conclusion that would justify taking a rigorous atheoretical stance. As I have implied previously, however, the belief that one can take positions free of theoretical bias is naive, if not nonsensical (Heelan, 1977; Hempel, 1965; Kukla, 1989; Leahey, 1980; Weimer, 1979). Those who claim to have eschewed theory are likely to have (unknowingly) subscribed to a position that gives primacy to such experience-near data as overt behaviors and biological signs, as opposed to experience-distant data that require a greater measure of inference. The positivist (empiricist) position may once have held sway in philosophy, as it still does in some psychiatric and psychological quarters (Schwartz & Wiggins, 1986); however, it is difficult, as Meehl (1978) noted, "to name a single logician or a philosopher (or historian) of science who today defends strict operationism in the sense that some psychologists claim to believe in it" (p. 815).

What distinguishes a true theoretically based taxonomy from one that merely provides an explanatory summary of known observations and inferences? Essentially, the answer lies in its power to generate new attributes, relations, or taxa—that is, ones other than those used to construct it. This generative power is what Hempel (1961) termed the "systematic import" (p. 6) of a scientific classification. In contrasting what are familiarly known as "natural" (theoretically guided and based in deduction) and "artificial" (conceptually barren and based in similarity) classifications, Hempel (1965) wrote:

> Distinctions between "natural" and "artificial" classifications may well be explicated as referring to the difference between classifications that are scientifically fruitful and those that are not; in a classification of the former kind, those characteristics of the elements which serve as criteria of membership in a given class are associated, universally or with high probability, with more or less extensive clusters of other characteristics.
>
> Classification of this sort should be viewed as somehow having objective existence in nature, as "carving nature at the joints" in contradistinction of "artificial" classification, in which the defining characteristics have few explanatory or predictive connection with other traits.
>
> In the course of scientific development, classifications defined by reference to manifest, observable characteristics will tend to give way to systems based on theoretical concepts. (pp. 146–148)

Evaluative Standards

Feinstein (1977) commented that a classification system "can be a product of sheer speculation or arbitrary caprice" (p. 196). Hence once a taxonomy has been constructed, be it comprehensive or circumscribed, it behooves clinicians and scientists to examine it as a

unit—that is, to evaluate its constituent taxa, as well as the specific attributes and defining features that constitute each taxon. To ensure a minimum of "sheer speculation or . . . caprice," developers of taxonomies must keep in mind several principles or standards that may optimize both the validity and utility of their creations. Such guidelines may prove especially useful in the formulation and construction phases of a taxonomy and may serve to orient developers in ways that may enhance their system's ultimate efficacy

I now note a few of the principles or standards that may guide both the construction and evaluation of taxonomic taxa and attributes. More extensive discussions of these and other standards may be found elsewhere (Millon, 1987, 1991; Sneath & Sokal, 1973). For pedagogical purposes, I make a distinction between standards more applicable to the diagnostic attributes that constitute taxa and those more relevant to the structure of the taxonomy.

Optimal Attribute Standards

What are some of the properties of clinical attributes that enable them to serve as a secure base for diagnostic criteria? A few are worth noting.

• *Feature comparability.* One method of refining diagnostic comparisons is to spell out a series of defining features for every relevant clinical attribute associated with a set of parallel diagnostic taxa. For example, as I have noted earlier, if a clinical attribute—this time, let us say interpersonal conduct—is deemed of diagnostic value in identifying and differentiating personality disorder taxa, then a distinctive description must be written to represent the characteristic or singular manner in which persons with each personality disorder conduct their interpersonal lives. A format of this nature furnishes symmetry among the taxa that constitute a taxonomy and enables investigators to systematically compare each taxon's diagnostic validity (e.g., sensitivity and specificity), as well as the relative diagnostic efficiency (e.g., positive and negative predictive power) for each relevant attribute (e.g., interpersonal conduct).

• *Empirical reference.* As I have also mentioned previously, attributes that depend on higher-order inferences contribute to diagnostic unreliability. Whenever this is feasible, the features that constitute diagnostic criteria ought to be assigned properties in the observable world. Problems arise when one seeks to represent intrinsically unobservable processes (e.g., defense mechanisms), or when one attempts to balance the desire for generality or openness among attributes with the standard of empirical precision. Can attributes be empirically anchored (and thereby the ambiguity in language be minimized), while the attributes are simultaneously freed to encompass wide-ranging phenomena, including those that reflect interior processes?

• *Quantitative range.* Clinical features usually express themselves as matters of degree rather than of simple presence or absence (e.g., severity of depression and level of anxiety). It is useful, therefore, if the clinical attributes that constitute taxa permit the registration of a wide range of intensity or frequency differences. This psychometric property of quantitative gradation is one of the notable strengths of psychological tests, but it is not limited to them.

Optimal Structural Standards

Unless a taxonomy is easy to understand and use, it is quite unlikely to gain adherents in the clinical world, no matter how well formulated and scientifically sound it may otherwise be.

• *Clinical relevance.* Some historic taxonomies were shrouded in a cloak of words and dense concepts. The structure of others was so opaque that assumptions were concealed, principles difficult to extract, and consistent connections to the clinical world impossible to establish. In short, the structure and language of the taxonomy and its taxa were formulated more complexly and obscurely than necessary. Relevance and simplicity suggest that a taxonomy should depend on a minimum number of assumptions, concepts, and categories. Alone, these standards neither eliminate taxonomic opaqueness nor validate the clinical utility of a taxonomy's derivations. They merely suggest that excess and misguided baggage should be eliminated, so that the features of clinical relevance in the system can be seen more clearly.

- *Representative scope*. If a taxonomy is too narrow in its range of applicability, failing to encompass disorders for which clinicians have diagnostic responsibility, then its level of utility and acceptance will be markedly diminished. Ideally, the number of taxa and attributes that a taxonomy subsumes ought not to be limited. However, it is wise to recognize that a disparity will exist between the potential range of a taxonomy's applicability and its actual range of empirical support.
- *Concurrent robustness*. A question arises as to whether taxonic groupings will retain their membership composition under new conditions and with attributes other than those used to construct them initially. For example, monothetic (homogeneous) taxa based on a single source of data (e.g., test scores), on one type of attribute (e.g., interpersonal conduct), or on one class of patients (e.g., inpatients) may fail to cross-generalize—that is, to remain stable, distinct, and uniform when based on parallel yet unidentical sources of data (e.g., structured interviews), attributes (e.g., cognitive style), or populations (e.g., outpatients).

Closing Comments

Although this chapter is long, it is but a brief sketch of a burgeoning field. Perhaps this introduction will tempt the reader to examine the subject in greater detail. At present, there are no unequivocal answers to the many questions posed in psychopathological taxonomy—be they the matters of selecting attributes, choosing structures, or opting for one construction method or another.

In my critical remarks, I have not meant to imply that the philosophies and techniques of classification today are irrelevant, or that the theoretical or diagnostic underpinnings of contemporary practice are valueless. Rather, I wish to encourage current taxonomic conceptualizers to step back and reflect more deeply on established assumptions and formulations. On the one hand, taxonomies in psychopathology must not reduce the richness of the natural clinical world to a series of conflicts among competing and abstruse speculations; nor must this world be passively shaped by the dehumanized and arcane methods of mathematical analysis. Protected from convention, vogue, presumption, or cabalism, taxonomic psychopathology will become neither dogmatic, trivial, and formalistic nor devoid of a substantive life of its own. Prevailing frameworks must continue to be challenged, and imaginative alternatives encouraged.

Acknowledgment

Portions of this chapter have been adapted and updated from Millon (1991). Copyright 1991 by the American Psychological Association. Adapted by permission.

References

American Psychiatric Association. (1952). *Diagnostic and statistical manual of mental disorders*. Washington, DC: Author.

American Psychiatric Association. (1968). *Diagnostic and statistical manual of mental disorders* (2nd ed.). Washington, DC: Author.

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.

Andreasen, N., & Grove, W. (1982). The classification of depression: A comparison of traditional and mathematically derived approaches. *American Journal of Psychiatry*, *139*, 45–52.

Bandura, A. (1982). The psychology of chance encounters and life paths. *American Psychologist*, *37*, 747–755.

Benjamin, L. S. (1974). Structural analysis of social behavior. *Psychological Review*, *81*, 392–425.

Benjamin, L. S. (1986). Adding social and intrapsychic descriptors to Axis I of DSM-III. In T. Millon & G. L. Klerman (Eds.), *Contemporary directions in psychopathology: Toward the DSM-IV* (pp. 599–637). New York: Guilford Press.

Benjamin, L. S. (2006). *Interpersonal reconstructive therapy: An integrative, personality-based treatment for complex cases*. New York: Guilford Press.

Blashfield, R. K. (1984). *The classification of psychopathology*. New York: Plenum Press.

Block, J. (1989). Critique of the act frequency ap-

proach to personality. *Journal of Personality and Social Psychology*, *56*, 234–245.
Bridgman, P. W (1927). *The logic of modern physics*. New York: Macmillan.
Buss, D. M., & Craik, K. H. (1983). The act frequency approach to personality. *Psychological Review*, *90*, 105–126.
Buss, D. M., & Craik, K. H. (1987). Act criteria for the diagnosis of personality disorders. *Journal of Personality Disorders*, *1*, 73–81.
Cantor, N., & Genero, N. (1986). Psychiatric diagnosis and natural categorization: A close analogy. In T Millon & G. L. Klerman (Eds.), *Contemporary directions in psychopathology: Toward the DSM-IV* (pp. 233–256). New York: Guilford Press.
Cantor, N., & Mischel, W. (1979). Prototypes in person perception. In L. Berkowitz (Ed.), *Advances in experimental social psychology* (Vol. 12). New York: Academic Press.
Cattell, R. B. (1965). *The scientific analysis of personality*. Chicago: Aldine.
Dougherty, J. W. D. (1978). Salience and relatively in classification. *American Ethnologist*, *5*, 66–80.
Essen-Moller, E., & Wohlfahrt, S. (1947). Suggestions for the amendment of the official Swedish classification of mental disorders. *Acta Psychiatrica Scandinavica*, *47*(Suppl.), 551–555.
Eysenck, H. J. (1960). *The structure of human personality*. London: Routledge & Kegan Paul.
Feinstein, A. R. (1977). A critical overview of diagnosis in psychiatry. In V. M. Rakoff, H. C. Stancer, & H. B. Kedward (Eds.), *Psychiatric diagnosis* (pp. 189–206). New York: Brunner/Mazel.
Frances, A. (1982). Categorical and dimensional systems of personality diagnosis: A comparison. *Comprehensive Psychiatry*, *23*, 516–527.
Frances, A., & Widiger, T. A. (1986). Methodological issues in personality disorder diagnosis. In T. Millon & G. L. Klerman (Eds.), *Contemporary directions in psychopathology: Toward the DSM-IV* (pp. 381–400). New York: Guilford Press.
Grove, W. M., & Andreasen, N. C. (1986). Multivariate statistical analysis in psychopathology In T. Millon & G. L. Klerman (Eds.), *Contemporary directions in psychopathology: Toward the DSM-IV* (pp. 347–362). New York: Guilford Press.
Grove, W. M., & Tellegen, A. (1991). Problems in the classification of personality disorders. *Journal of Personality Disorders*, *5*, 31–41.
Gunderson, J. G., Links, P. S., & Reich, J. H. (1991). Competing models of personality disorders. *Journal of Personality Disorders*, *5*, 60–68.
Hartigan, J. A. (1975). *Clustering algorithms*. New York: Wiley.
Heelan, P. A. (1977). The nature of clinical science. *Journal of Medicine and Philosophy*, *2*, 20–32.
Hempel, C. G. (1961). Introduction to problems of taxonomy. In J. Zubin (Ed.), *Field studies in the mental disorders* (pp. 3–22). New York: Grune & Stratton.
Hempel, C. G. (1965). *Aspects of scientific explanation*. New York: Free Press.
Horowitz, L. M., Post, D. L., French, R. de S., Wallis, K. D., & Siegelman, E. Y. (1981). The prototype as a construct in abnormal psychology: 2. Clarifying disagreement in psychiatric judgments. *Journal of Abnormal Psychology*, *90*, 575–585.
Jackson, D. W. (1971). The dynamics of structured personality tests. *Psychological Review*, *78*, 229–248.
Kaplan, A. (1964). *The conduct of inquiry*. San Francisco: Chandler.
Kendell, R. E. (1968). *The classification of depressive illness*. London: Oxford University Press.
Kendell, R. E. (1975). *The role of diagnosis in psychiatry*. Oxford, UK: Blackwell.
Kiesler, D. J. (1983). The 1982 Interpersonal Circle: A taxonomy for complementarity in human transactions. *Psychological Review*, *90*, 185–214.
Kiesler, D. (1996). *Contemporary interpersonal theory and research: Personality, psychopathology, and psychotherapy*. New York: Wiley.
Kraepelin, E. (1899). *Psychiatrie: Ein Lehrbuch* (6th ed.). Leipzig: Barth.
Krueger, R. F., & Tackett J. L. (Eds.). (2006). *Personality and psychopathology*. New York: Guilford Press.
Kukla, A. (1989). Nonempirical issues in psychology. *American Psychologist*, *44*, 785–794.
Leahey, T. (1980). The myth of operationism. *Journal of Mind and Behavior*, *1*, 127–143.
Leary, T. (1957). *Interpersonal diagnosis and personality*. New York: Ronald Press.
Livesley, W. J. (1985). The classification of personality disorder: 1. The choice of category concept. *Canadian Journal of Psychiatry*, *30*, 353–358.
Livesley, W. J. (1991). Classifying personality dis-

orders: Ideal types, prototypes or dimensions. *Journal of Personality Disorders*, *5*, 55–59.

Loevinger, J. (1957). Objective tests as measurements of psychological theory. *Psychological Reports*, *3*, 635–694.

Lorr, M. (1966). *Explorations in typing psychotics*. New York: Pergamon Press.

Medin, D. L., Altom, M. W., Edelson, S. M., & Freko, D. (1982). Correlated symptoms and simulated medical classification. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *8*, 37–50.

Meehl, P. E. (1972). Specific genetic etiology, psychodynamics, and therapeutic nihilism. *International Journal of Mental Health*, *1*, 10–27.

Meehl, P. E. (1978). Theoretical risks and tabular asterisks: Sir Karl, Sir Ronald, and the slow progress of soft psychology. *Journal of Consulting and Clinical Psychology*, *46*, 806–834.

Menninger, K. (1963). *The vital balance*. New York: Viking.

Mezzich, J. E. (1979). Patterns and issues in the multiaxial psychiatric diagnosis. *Psychological Medicine*, *9*, 125–137.

Millon, T. (1969). *Modern psychopathology: A biosocial approach to maladaptive learning and functioning*. Philadelphia: Saunders.

Millon, T. (1977). *Millon Clinical Multiaxial Inventory manual*. Minneapolis, MN: National Computer Systems.

Millon, T. (1981). *Disorders of personality: DSM-III, Axis II*. New York: Wiley.

Millon, T. (1983). The DSM-III: An insider's perspective. *American Psychologist*, *38*, 804–814.

Millon, T. (1984). On the renaissance of personality assessment and personality theory. *Journal of Personality Assessment*, *48*, 450–466.

Millon, T. (1986). Personality prototypes and their diagnostic criteria. In T. Millon & G. L. Klerman (Eds.), *Contemporary directions in psychopathology: Toward the DSM-IV* (pp. 671–712). New York: Guilford Press.

Millon, T. (1987). On the nature of taxonomy in psychopathology. In C. G. Last & M. Hersen (Eds.), *Issues in diagnostic research* (pp. 3–85). New York: Plenum Press.

Millon, T. (1990). *Toward a new personology: An evolutionary model*. New York: Wiley-Interscience.

Millon, T. (1991). Classification in psychopathology: Rationale, alternatives, and standards. *Journal of Abnormal Psychology*, *100*, 245–261.

Osherson, D. N., & Smith, E. E. (1981). On the adequacy of prototype theory as a theory of concepts. *Cognition*, *9*, 35–58.

Panzetta, A. E. (1974). Towards a scientific psychiatric nosology: Conceptual and pragmatic issues. *Archives of General Psychiatry*, *30*, 154–161.

Pap, A. (1953). Reduction-sentences and open concepts. *Methodos*, *5*, 3–30.

Paykel, E. S. (1971). Classification of depressed patients: A cluster analysis derived grouping. *British Journal of Psychiatry*, *118*, 275–288.

Pincus, A. L., & Wiggins, J. S. (1990). Interpersonal problems and conceptions of personality disorders. *Journal of Personality Disorders*, *4*, 342–352.

Plutchik, R., & Conte, H. R. (1985). Quantitative assessment of personality disorders. In J. O. Cavenar (Ed.), *Psychiatry* (Vol. 1). Philadelphia: Lippincott.

Plutchik, R., & Platman, S. R. (1977). Personality connotations of psychiatric diagnoses: Implications for a similarity model. *Journal of Nervous and Mental Disease*, *165*, 418–422.

Quine, W. V. O. (1977). Natural kinds. In S. P. Schwartz (Ed.), *Naming, necessity and natural groups*. Ithaca, NY: Cornell University Press.

Rosch, E. (1978). Principles of categorization. In E. Rosch & D. B. Lloyds (Eds.), *Cognition and categorization*. Hillsdale, NJ: Erlbaum.

Sartorius, N. (1990). Sources and traditions of psychiatric classification: Introduction. In N. Sartorius, A. Jablensky, D. A. Regier, J. D. Burke, Jr., & R. M. A. Hirschfeld (Eds.), *Sources and traditions of classification in psychiatry* (pp. 1–6). Bern, Switzerland: Hogrefe & Huber.

Schwartz, M. A. (1991). The nature and classification of the personality disorders: A reexamination of basic premises. *Journal of personality Disorders*, *5*, 25–30.

Schwartz, M. A., & Wiggins, O. P. (1986). Logical empiricism and psychiatric classification. *Comprehensive Psychiatry*, *27*, 101–114.

Schwartz, M. A., Wiggins, O. P., & Norko, M. A. (1989). Prototypes, ideal types, and personality disorders: The return of classical psychiatry. *Journal of Personality Disorders*, *3*, 1–9.

Sim, J. P., & Romney, D. M. (1990). The relationship between a circumplex model of interpersonal behaviors and personality disorders. *Journal of Personality Disorders*, *4*, 329–341.

Simpson, G. G. (1961). *Principles of animal taxonomy*. New York: Columbia University Press.

Skinner, H., & Blashfield, R. (1982). Increasing the impact of cluster analysis research: The case of psychiatric classification. *Journal of Consulting and Clinical Psychology*, *50*, 727–735.

Skinner, H. A. (1981). Toward the integration of classification theory and methods. *Journal of Abnormal Psychology*, *90*, 68–87.

Skinner, H. A. (1986). Construct validation approach to psychiatric classification. In T. Millon & G. L. Klerman (Eds.), *Contemporary directions in psychopathology: Toward the DSM-IV* (pp. 307–329). New York: Guilford Press.

Smith, E. E., & Medin, D. L. (1981). *Categories and concepts*. Cambridge, MA: Harvard University Press.

Sneath, P. H. A., & Sokal, R. R. (1973). *Numerical taxonomy*. San Francisco: Freeman.

Sokal, R. R. (1974). Classification: Purposes, principles, progress, prospects. *Science*, *185*, 1115–1123.

Sokal, R. R., & Sneath, P. H. A. (1963). *Principles of numerical taxonomy*. San Francisco: Freeman.

Spitzer, M. (1990). Why philosophy? In M. Spitzer & B. A. Maher (Eds.), *Philosophy and psychopathology*. New York: Springer-Verlag.

Strack, S. (Ed.). (2005). *Handbook of personology and psychopathology*. Hoboken, NJ: Wiley

Strack, S. (Ed.). (2006). *Differentiating normal and abnormal personality*. New York: Springer.

Strack, S., Lorr, M., & Campbell, L. (1990). An evaluation of Millon's circular model of personality disorders. *Journal of Personality Disorders*, *4*, 353–362.

Strauss, J. S. (1986). Psychiatric diagnoses: A reconsideration based on longitudinal processes. In T. Millon & G. L. Klerman (Eds.), *Contemporary directions in psychopathology: Toward the DSM-IV* (pp. 257–264). New York: Guilford Press.

Tversky, A. (1977). Features of similarity. *Psychological Review*, *84*, 327–352.

Weimer, W. (1979). *Notes on the methodology of scientific research*. Hillsdale, NJ: Erlbaum.

Widiger, T. A., & Frances, A. (1985a). Axis II personality disorders: Diagnostic and treatment issues. *Hospital and Community Psychiatry*, *36*, 619–627.

Widiger, T. A., & Frances, A. (1985b). The DSM-III personality disorders: Perspectives from psychology. *Archives of General Psychiatry*, *42*, 615–623.

Williams, J. B. W. (1985a). The multiaxial system of DSM-III. Where did it come from and where should it go?: I. Its origins and critiques. *Archives of General Psychiatry*, *42*, 175–180.

Williams, J. B. W. (1985b). The multiaxial system of DSM-III. Where did it come from and where should it go?: II. Empirical studies, innovations, and recommendations. *Archives of General Psychiatry*, *42*, 181–186.

Wittgenstein, L. (1953). *Philosophical investigations*. Oxford, UK: Blackwell.

World Health Organization. (1992). *International statistical classification of diseases and related health problems* (10th rev.). Geneva: Author.

Wright, J. C., & Murphy, G. L. (1984). The utility of theories in intuitive statistics: The robustness of theory-based judgments. *Journal of Experimental Psychology: General*, *113*, 301–322.

Zubin, J. (1968). Biometric assessment of mental patients. In M. M. Katz, J. O. Cole, & W. E. Barton (Eds.), *Classification of psychiatry and psychopathology* (pp. 353–376). Washington, DC: U.S. Public Health Service.

CHAPTER 8

Diagnostic Taxa as Open Concepts

Metatheoretical and Statistical Questions about Reliability and Construct Validity in the Grand Strategy of Nosological Revision

PAUL E. MEEHL

Since I find it hard to conceive that a rational mind could think otherwise, I presuppose that, *ceteris paribus*, careful delineation of the signs, symptoms, and course of a disorder (I cannot interest myself much in the semantic hassle over whether to call it "disease") so as to increase the reliability of classifying clients or patients is desirable. While reliability and validity are not the same thing, it is a psychometric truism that the former bounds the latter, although it is worth mentioning that the bound is the square root of the reliability, so validity can theoretically be larger. Usually the operative validity (net attenuated construct validity) runs far below that upper bound set by the square root of the reliability coefficient. Hence alterations in the format of assessment or in the content sampled, which might under some circumstances reduce reliability, could nevertheless increase the net attenuated construct validity. Similarly, changes in content or format that increase reliability may theoretically decrease validity. For instance, an alteration in the open-ended, unstructured format of Rorschach administration (as was attempted during World War II to make it possible to test large numbers of individuals and score reliably without inquiry) seemed to eliminate whatever slight validity the instrument had as usually administered.

There is no mystery about this, although it is paradoxical at first look. We may be concerned about the reliability of behavior sampling by two different samplers ("interjudge agreement") or with the trustworthiness of a sample as drawn by an individual judge (how many marbles do we draw from the urn, and how do we draw them?). In either case, the point is this:

Whether an interview behavior or a psychological test item is viewed primarily as "sample" or as "sign" (Cronbach & Meehl, 1955), there are kinds of alterations in the examining situation and in the procedure of response classification that can alter qualitatively the intrinsic construct validity of the sample in such a way as to reduce its net validity—despite reliability, in either of the two senses mentioned, having been enhanced.

I am not arguing that such has occurred in the process of improving our old Mental Status Examination or in the construction of DSM-III, but merely that this methodological point should be kept in mind when discussing reliability/validity questions.

It requires neither psychometric nor philosophical expertise to see that the reliability/validity helps and tradeoffs can be somewhat complicated, and especially so when the aimed-at diagnostic construct itself (category or dimension) is an open concept, lacking a definitive "operational" criterion, specified implicitly ("contextual definition") by presumably fallible indicators. In that kind of knowledge situation, we subtly alter meanings as we discover facts; we amend theoretical definitions as we revise indicator weights. The basic point can be better brought out by considering the decision to include an unreliable indicator in a standard examination for any "disease." A general medical examination always includes blood pressure and not anthropometric determination of wrist width, despite the mediocre reliability of the former and $r = .98$ for the latter. We do not find this evidentiary preference puzzling; we simply say, "Blood pressure unreliably measured is a stronger indicator of more different and important conditions than wrist width reliably measured." Similarly, a psychotherapist who employs dream interpretation (with the manifest–latent content model) would not seriously consider substituting reliably scorable multiple-choice inquiry for free association under the Fundamental Rule, despite the grave reliability problems posed by the classical procedure. One might prefer to avoid Freud's technique altogether, and partly because of unreliability considerations (cf. Meehl, 1983); what one would almost surely not do is retain Freud's core idea and its entailed technique, while substituting a multiple-choice inquiry in the service of reliability.

The reasons for desiring diagnostic reliability are well known. The most important reason is generalizability of research findings by other investigators thinking about their research and by practitioners in applying research findings to clinical decision making. The easiest way to understand the former is in terms of the number of pairwise relationships of input and output variables involved in a decision-making process, whether of a theoretical or practical clinical nature (Meehl, 1959). If a set of behavior data (history and current status, interview, ward behavior, neurological, psychometric) permits us to classify patients in some "rational" (ultimately "causal"?) way, it is not necessary that each of the possible n output variables (e.g., treatment of choice, second choice, prognosis, employability, response to group therapy, suicide risk, genetic risk to offspring) has to be correlated singly pairwise with all the input variables—a process which would require studying mn relationships, where mn is in the thousands (Meehl & Golden, 1982, pp. 130–131). Instead, we can first relate the m input variables to the diagnostic dimension or rubric and then relate the diagnostic dimension or rubric to the several output variables of interest. Hence only $(m + n)$ correlations need to be studied. But that process cannot be carried out with any confidence if the relation of some of the input variables to dimension X or categorical rubric C as found at the University of Texas has only a little better than chance relationship between (only partially overlapping) relationships as reported by investigators in Milwaukee. The pulling together of research data to give a coherent interpretation of an alleged psychiatric entity, whether taxonomic or dimensional in nature, presupposes the possibility of scanning the research literature with at least some reasonable confidence that patients called "schizophrenic" by one investigator are like those called "schizophrenic" by another. Similarly, suppose a clinician reads a research report claiming that a certain drug is efficacious for [patients with] paranoid schizophreni[a], except when they have a history of an episode, when much younger, of acute catatonic excitement. That report is not helpful to the clinician who lacks rational belief that the investigator was looking at the same indicators of paranoid and catatonic schizophrenia that he or she can now look at in his or her clinical decision making.

It is an interesting question whether one can ever lose by improving reliability, except in the (rare? I don't know) sense discussed above. The main respect in which some workers, and I gingerly include myself here, seem to worry about it is that research aimed at improving, correcting, or—in the extreme case—refuting views implicit in the DSM-III conceptual system will somehow be cramped by an overly enthusiastic view of it, which sometimes takes the form of a dogmatic insistence upon its merits throughout. I have heard research-oriented clinicians express concern about this, but it is difficult to

track down persuasive examples where, for instance, an otherwise admirable research proposal was rejected by the peer reviewers on the grounds that it did not employ "official categories" approved by DSM-III. While people talk about this, and one sometimes hears it alleged that it has occurred, I do not myself know of any clear cases. Admittedly, it would be hard to ascertain whether a subtle kind of social process, of the kind that the Supreme Court likes to call a "chilling effect," is taking place. Some researchers might be otherwise disposed to advocate a mild Feyerabendian "proliferation of theories" (Feyerabend, 1970), which he advocates even for cases when the going theories are extremely powerful and well corroborated and, a fortiori, for theories in such primitive fields as psychopathology. Some of them might not be getting research grant money because they have timidly avoided challenging the establishment category system.

Here again, I have no affirmative evidence that such things happen. If they do, it would appear quite easy to find a way around it, and whether it failed would hinge upon whether some peer reviewers have become overidentified with the present product. For example, suppose I am interested in studying people with a cyclothymic personality makeup who have very mild ups and downs on an endogenous (genetic/biochemical) basis, but who at no time become diagnosably psychotic or even semipsychotic. The psychiatric tradition has connected endogenousness with severity, which there is no strong theoretical reason for insisting upon, although there is a correlation empirically. I don't see why a clinical investigator, behavior geneticist, or neurochemist should in any way be hampered by the received rubrics. He or she can be careful in adhering to criteria for diagnosing manic–depressive disorder as given by DSM-III. It may well be that the only available rubric for some of the other people he or she wants to study is "normal," or even perhaps some other piece of nonmanic–depressive terminology as specified in DSM-III. Nothing prevents the investigator from saying, in writing up a grant proposal, "It is my empirical conjecture that there are persons who don't manage quite to squeak through the conditions for diagnosing a manic–depressive attack (because of extreme damping in their cyclothymic cycle). But all of my classifications are indicated, and all of the correlations of them with all of the other things I studied, whether psychometric or genetic or familial or whatever, are clearly indicated, so that other investigators may rely on the fact that I stuck literally to the received criteria for making that diagnosis. I have also listed, however, the set of special criteria, together with their time sampling and interjudge reliabilities, that I used to demarcate my special subgroup of individuals that do not fit the official rubrics."

At no point does this investigator have to depart from the semantics of DSM-III; nor does he or she have to do any inordinate amount of work in order to include the DSM-III criteria as available for investigators who want to examine his or her data critically. It is, I suppose, imaginable that somebody might want to do something where the task of "double diagnosis" (i.e., according to his or her conjectured criteria for entities or dimensions not in the official list along with the received one) will be a considerable amount of excess work, but I am not aware of any clear [evidence] showing that that has happened. The diagnostic criteria for DSM-III simply do not involve that much additional work, and most of the overload will arise from his or her idiosyncratic system. Despite the fact that my own views on many categories are quite heterodox, when there is an adoption by an empowered body of clinicians and scientists as to a certain terminology, I think one is not unduly burdened or imposed upon by some extra scientific or clinical toil when the investigator chooses to deviate from it in his or her own research.

An interesting statistical question arises in the "context of discovery" (Reichenbach, 1938, pp. 6–7), where a plausible case—I do not urge that it is more than plausible—can be made for concern about increased difficulty of detecting subtle relationships. I mean by "detection" the development of a clinical hunch and, in a more formalized research context, the problem of the statistical power function failing to detect something that is there. Consider the following: By tightening up the diagnostic criteria, we have increased reliability and, hence (almost certainly), the net attenuated construct validity in identifying the whole class of patients called "schizophrenic." In the course of so doing, we have been forced to elimi-

nate some signs and symptoms that some clinicians have been relying on. Perhaps we ourselves had been doing so, but we are willing to pay this price. We are even willing to pay the price of dropping something that was considered fundamental by the master himself—as, for instance, DSM-III does not include Bleuler's ambivalence or his autism; or, to take an instance closer to my heart, Rado's anhedonia (Meehl, 1962, 1964, 1974–1975, 1975). Less counterconventional, one thinks of the pan-anxiety considered extremely important—perhaps the most important single symptom—in the "pseudoneurotic schizophrenia" syndrome described by Hoch and Polatin (1949). The latter two examples are of course controversial; but as to the former, it is hard to believe that we should omit two of Bleuler's cardinal signs unless this choice is dictated by difficulty objectifying them in the interest of reliability.

I repeat that I am not here disputing the claim that the net attenuated construct validity for identifying the whole class of [patients called] "schizophrenic" has been increased by the tightening process, and I am not at the moment concerned with the efficacy of clinical handling, but I am attending to the research context. It is surely possible, and to a statistically and philosophically sophisticated person not even paradoxical, that some subset of patients sharing underlying etiology and psychopathology (genetics, biochemistry, [central nervous system] fine structure, and psychodynamics or "personality structure") with the core group of [patients who have] schizophrenias but who, because of modifying genes and normal-range individual differences factors (Meehl, 1975) as well as life history experiences, do not develop the signs and symptoms that have remained in the selected list of DSM-III, or—equally possible despite average heightened reliability—do not have them in sufficient quantity to be clear instances. Such a state of affairs is not only consistent with, but is probabilistically inferable from either the medical model, classical psychometrics, genetics, learning theory, or ordinary trait theory. The point is that clinicians trained to classify patients with the reduced high-reliable list of criteria will not be psychologically disposed to consider the subset of peripheral or borderline cases as belonging to the schizophrenic group (as they should not in applying the objectified criteria). In the context of discovery, this could sometimes operate adversely, since the way you categorize your world, as we all know, will in considerable part determine what you are capable of noticing.

But suppose a perceptive clinician does notice something about these borderline cases and undertakes a systematic research study of something middling complicated and not easy to discern—say, for example, a second-order interaction between phenothiazines and a certain mode of psychotherapeutic intervention (e.g., [rational–emotive therapy]). Now if the polygenic modifiers or environmental factors that make the [patients with] atypical schizophrenias show a different kind of interaction effect from the core group, that will not be detected statistically, even having been noticed clinically by a gifted clinician, because such cases will only rarely (and mostly due to carelessness in applying the new criteria!) be included in the study. If one believes (as I do) that the psychiatric treatment of the future will involve complicated kinds of actuarial grounds for selecting and sequencing the treatment of choice (Meehl, 1972a, pp. 135–137), early research progress along such lines could be hampered in this way.

I want to emphasize that I'm not here invoking some kind of vague clinical intuitions about "patterns." I am making a simple point about research statistics—that is, that you can't detect a trend that makes a subset of subjects different from the other subjects in a certain group if there aren't any of the subset present in the study. Furthermore, as we move into higher-order interaction effects, such as Drug × Psychotherapy × Subdiagnosis patterns, the degrees of freedom shrink so that errors of Type II begin to preponderate due to marked reduction in statistical power.

It might be argued that while this may impose an irksome hurdle in the context of discovery at the intuitive stage for the clinician trained in the use of DSM-III, and thinking more or less automatically that way, it will not have any long-run bad effect because the cases not included in such studies will be detectable in studies focusing on some other diagnostic rubric. I think that is an optimistic view because it implies that

some sort of massive research network of all possible combinations of everything with everything is going to take place in psychiatry and clinical psychology, which it is not, both for economic and professional interest reasons. Furthermore, what kind of thing is detected will depend on what initial overall rubric is being studied. If these borderline cases were subsumed under "anxiety state" rather than "borderline schizophrenia," the interaction effect between an antipsychotic drug and cognitive therapy will not be a likely subject matter of investigation. Finally, what is perhaps the more serious statistical point, such people will not be found in any one rubric if misdiagnosed because of the tight criteria (by misdiagnosed, I of course mean subsumed to the wrong specific etiological group [Meehl, 1972b, 1977] in the eyes of Omniscient Jones), hut are likely to be dispersed. When one disperses a group of people who are heterogeneous in some respects, but homogeneous in some core feature of high causal relevance, into a number of heterogeneous diagnostic categories, the best bet is that they will simply get lost in the shuffle. While I do not claim to know that this is a serious problem, it is not a silly consideration that can be dismissed out of hand without thorough mathematical analysis.

Moving away from what one might call the "political–social–economic" impact of DSM-III, it is worthwhile to examine at a more philosophical level the ways in which a practitioner or researcher may view its categories and dimensions. I can see three (although not sharply demarcated), one of which is admirable, one of which is criticizable but fairly harmless, and only the third of which is scientifically malignant. The first is to view the delineation of a syndrome as an empirically observed (clinically or statistically!) cluster, a syndrome plus course, that suggests to us some kind of underlying causal homogeneity in the subjects who show it—although we may, depending on our theoretical predilections, sit quite loosely to this etiological promissory note. Its justification is mainly communicative and pragmatic, together with whatever degree of faith we have from the history of medicine (and genetics, and psychometrics) that future research will give us a more detailed understanding of whatever historical and "latent" (inner) current processes and structures are at work to produce the covariation of the signs, symptoms, aspects of course, prognosis, and response to treatment. Covariation is the essence of descriptive science and the touchstone of scientific thinking, whether we read such diverse writers as Freud, Skinner, Allport, Murray, Eysenck, Thurstone, or Cattell—strange bedfellows indeed, whose unanimity on this point should surely tell us something about how to study the mind! *Ceteris paribus* again, the more standardized the examination can be made, the more objectively described the classification of the responses, and as a result, the greater interjudge agreement by different examiners, and the more striking the empirical "tightness" of the cluster, the better we like the syndrome as an entity. As already stated, it is hard to understand why a rational mind would object to any approach that enhances these desirable properties.

Second, one may believe that DSM is the best that can be achieved, at least in the foreseeable future, and may be suspicious or even antagonistic to deviations from it, for either clinical or research purposes. This attitude troubles me, but I should think it can be adequately buffered by the practice I suggested above—that is, that investigators have a responsibility to employ it until it is officially revised by some "culturally empowered" group such as those who constructed it in the first place. But we do not pressure researchers or punish them financially or otherwise, once they have met these conditions in their semantics, for delineating some further conjectural entities or dimensions of their own, hoping to persuade the profession on the basis of clinical experience of better evidence that they are right.

It is the third attitude which I think is malignant, partly because of its potential chilling effect, but mainly because it is philosophically so terribly mistaken. It says not merely that "this is a good thing so far as it goes, and should not be lightly discarded or whimsically amended." This third view claims it is *the* truth, as a matter of some kind of rigorous definition process. The extreme (simplistic, "vulgar operationist") form of this view is that the very *meaning* of the concepts is contained, exhaustively and explicitly, in the "operational definitions" provided by DSM. It would be hard to find one single logician or historian of sci-

ence today (or for that matter, since around 1935!) who would countenance the conception of scientific method enshrined in this view. I find it puzzling that physicians, or for that matter, psychologists, unless they are of the most dogmatic behaviorist kind, should adopt this position when neither the history of organic medicine, nor of genetics (I don't mean here merely behavior genetics), nor of traditional trait theory in academic psychology, nor of classical psychometrics gives any support to it. It is simply not true that diseases in organic medicine are "defined by" the syndrome or by the syndrome and course together. Organic diseases are defined by a conjunction of their etiology and pathology when these are known, and otherwise—with much less scientific assurance—as syndromes remaining to be researched so as to be medically understood. A disease entity, as delineated in the early stages of clinical experience and scientific study, at the level of mere syndrome description when there is as yet no (or minimal and conjectural) knowledge of the etiology or pathology underlying it, is an open concept (Cronbach & Meehl, 1955; Meehl, 1972b, 1977; Meehl & Golden, 1982; Pap, 1953, 1958, Chap. 11). It is neither philosophically rigorous nor scientifically sophisticated to make a literal identification of a disease entity with its currently accepted signs and symptoms. Corresponding to organic medicine's pathology (in a more extended sense than that envisaged by Virchow) is personality structure (genotypic traits, psychodynamics). Corresponding to etiology are, except for an environmentalist fanatic, the genetic predispositions not only to specific mental disorders, but to "temperamental genotypic traits" generally, such as anxiety conditionability, rage readiness, hedonic capacity, general intelligence, and the like, and the learning history imposed on an organism whose varied behavior acquisition functions are characterized by such-and-such inherited parameters. Our problem in psychopathology of the so-called functional behavior disorders is obvious—to wit, that we do not possess an equivalent to the pathologist's and microbiologist's report telling us the "right answer" at the conclusion of a clinicopathological case conference (Meehl, 1973, pp. 284–289). If I make a psychodynamic inference, it is not possible for me to ask the psychopathologist whether his [or her] stained slides showed the patient's psyche had holes in the superego. To a thoughtful clinician with philosophical sophistication, it is perfectly obvious that disease syndromes are inherently open concepts, as mentioned above. Nothing but dogmatism on the one hand, or confusion on the other, is produced by pretending to give operational definitions in which the disease entity is literally identified with the list of signs and symptoms. Such an operational definition is a fake.

If somebody does not like the medical model (and if that's the case, one wouldn't be taking the DSM—concocted by a group of psychiatrists for medical purposes—seriously to begin with), he [or she] should be reminded that in classical psychometrics (such as factor analysis) or in more recent developments (such as multidimensional scaling), we cannot even write the basic equations, let alone the embedding interpretative text required to give empirical meaning to the variables in those equations, unless a clear distinction is already made between the manifest behavior indicators and the inferred (latent, causal) factors. The same is true of biophysical trait theory as classically elaborated by Allport (1937), Murray (1938), Cattell (1946), and others. Obviously, the great breakthrough in genetics with Mendel, and the rediscovery of Mendel's concepts at the turn of the [20th] century, hinged upon the distinction between the genotype and the phenotype. This distinction forced theoretical recognition that under many circumstances or available pedigrees, the weakly stochastic relationship between the two made an inference to genotype impossible.

One simple-minded mistake that I am surprised to find physicians making is to think that if, in a given concrete instance (single case, not class), we do not have a touchstone for testing whether a certain inferred construct property such as a latent disease is present or absent, that lack means that it is scientifically meaningless to ask the question—a view that the logician Carnap, a strongly positivist and tough-minded philosopher of science, refuted definitively almost a half century ago!

The same is true of most variants of learning theory—the old-fashioned kind (Tolman, Hull, or Guthrie) as well as the souped-up developments in mathematical learning theory, information processing, and

cognitive processes generally that took place subsequently. The only plausible exception to the genotypic–phenotypic, inner–outer, inferred–observed distinction in learning theory is strict Skinnerian learning theory, which is almost entirely dispositional, although not as "pure" in this respect as some of its adherents like to think when they talk metatheory about it.

I am fond of referring clinical psychology students to a little known two-page article published many years ago by the late T. A. Peppard (1949), a reputedly brilliant diagnostician who practiced internal medicine in Minneapolis for many years. He made a statistical study of the source of his diagnostic mistakes, using very strict criteria against postmortem findings. Errors of omission (well known to be commoner than errors of commission in medical diagnosis) sometimes occurred because he failed to look for something, other times because he looked for it but didn't give it the proper weight, other times because he made an "error" on a judgment call, and so on. But the interesting thing is that 29% of the errors of omission were attributable, even by very tight standards imposed on himself, to the factor he called "symptoms and signs not found." Of course, all physicians know the concept of "silent disease" such as an undiagnosed staghorn kidney or an early Pick's frontal lobe atrophy, not to mention subjects with an epileptic brain wave who never have a fit and would not be discovered except for being the monozygotic twin of somebody who has a clinically recognizable convulsive disorder. I repeat that I find it strange that one must remind physicians about the distinction between the construct "disease" and its presently accessible symptom picture, although it is not so surprising that some psychologists confuse them.

Finally, of course the most obvious example, which would still be persuasive to some of my generation, is psychodynamics, whose essence consists in the distinction between the easily observed manifest behavior or self-awareness and the "hidden, latent, underlying source" of some aspect of observable covariation.

Since neither psychodynamics, classical psychometrics, taxometrics, organic medicine, genetics, learning theory, nor trait theory has proceeded by explicit identification between theoretical entities and their indicators, it would be strange to hold that rational use of DSM-III requires us to consider its syndromes as literally definitive and totally noninferential.

It might be argued that if the builders of DSM had achieved consensus on constructing a purely descriptive (atheoretical, noninferential) "phenomenological" taxonomy, they should have proceeded by applying an appropriate formal cluster algorithm to a huge batch of carefully gathered clinical data, "letting the statistics do the whole job for them," which would have saved a lot of conference time as well as generating a more objective scientific product. This sounds plausible to a psychologist, and maybe to some statisticians, but the main trouble with it is that there is no "accepted" cluster algorithm which is known to be sufficiently powerful to be used in this way (cf. Meehl, 1979). Even if there were such an agreed-upon cluster analysis algorithm, one doubts that the committee could have proceeded in that way. The fact is that different clinicians do not share an equally "operational" view, partly for the reasons I have given and partly because of certain clinical (perhaps one could even say ideological) identifications—for example, between organicists and psychoanalysts, biotropes and sociotropes, scientists interested in genes and psychotherapists interested in battle-ax mothers.

I am inclined to think that the next DSM development round ought to at least settle on some way of deciding when the orientation should be taxonomic versus dimensional. But that would hinge upon having a sufficiently well-trusted algorithm for determining whether the latent order of a syndrome or dimension should be thought of as taxonic or nontaxonically factorial. Another possibility, which again seems simplistic and arbitrary until you ask what are the reasons for doing it another way, would be to collect all of the information or input kinds of variables, including life history data and the like, that go into diagnosis, and all of the output dispositions that are clinical reasons for making a diagnosis, such as differential response to psychotropic drugs, response to individual and group therapy, danger of acting out, suicide risk, and long-term employability. Absent cogent reasons for giving higher weight to some of these output ones

than others, it is arguable that the proper statistical model should be canonical correlation, in which we simultaneously optimize the predictability of the most predictable composite on the output side by optimal weights on the input variables. If the various output consequences of clinical importance are not prima facie very different in "importance," if they are, so to speak, qualitatively of equal significance to us in decision making, then the difference in the weights they get might best be to weight them so as to make them collectively most predictable. The justification for defining a syndrome (or a nontaxonic factor) by some subset of input and output considered jointly would be that the canonical correlation between the two sets reaches a certain minimum size. It would be interesting, by the way, to ascertain whether such a distribution of candidate canonical correlations would show, if not an actual break, at least some tendency to bimodality, suggesting that some syndromes are "real" and others are more or less arbitrary carvings out by the clinician of regions of slightly greater densification in the multivariate descriptor space (but see Murphy, 1964). My own research interests are such that I consider that the initial distinction between whether one should proceed taxometrically or factorially should be given very great priority in the next revision.

The question as to the desirability of adopting a fixed-rule approach to diagnostic criteria involves a complicated mix of statistical, philosophical, and clinical issues that are beyond the space limitations of this chapter and about which I myself have formed no definite opinion. This question has been aired recently in papers by Finn (1982, 1983) and Widiger (1983) (see also Meehl & Rosen [1955], comment by Cureton [1957], and Rorer, Hoffman, LaForge, & Hsieh [1966]). In thinking about this difficult question, it is necessary first to distinguish between issues regarding base rate fluctuations in different clinical or research populations and the separate but intimately related issues of clinical utility in treatment and prognosis. In saying these are distinct but intimately related, I mean to emphasize that from the standpoint of scientific realism (surely the implicit assumption of organic medicine, whether medical researchers or practitioners use the philosopher's terminology for it or not!), one does not wish to conflate the probability or corroboration of a diagnostic statement as a factual claim with the seriousness of a mistake. As Widiger worried about in his exchange with Finn, we do not want to adopt a decision rule based on a policy of systematically misdiagnosing patients on the grounds that correctly diagnosing a subset of them would, in certain pragmatic contexts, be too costly or risky or have too many side effects or make them more uncomfortable than the disease makes them or whatever. Crudely put, the first business of a diagnostic assertion is to be right! We cannot make use of differential utilities and disutilities of clinical errors without at least some crude assessments of diagnostic confidence, whereas we can investigate the optimality of a diagnostic procedure with regard to truth value without referring to any utilities other than the "cognitive utility" of being correct in our assertions. It would seem best, if it can be done and is psychologically acceptable to practitioners, to optimize the diagnoses by some suitable adjustment for known or guesstimated base rates in a given clinical population, and subsequently to raise the question of the various utilities involved in adopting a certain treatment plan or making predictions to the patient, court, employer, insurer, family, or whatever. In that mode of reasoning, the best inferable diagnostic statement is made first and the utilities are plugged in afterward.

But this of course doesn't take care of the base rate problem. Theoretically we know that both the cutting score on a variate which is an indicator of the disease entity and any formal or informal weighting of the scores or way of combining them into a pattern, as in Bayes's formula, should not be done independently of the base rates. In ordinary clinical medicine, practitioners who never heard of the Reverend Thomas Bayes or the subsequent controversy about his ideas (this use of the formula itself is, of course, hardly controversial) make implicit use of it. They know that if you diagnose syphilis in Puerto Rico on the grounds of a positive Wassermann, you are likely to fall into errors that you would not make in Minnesota because of the geographic epidemiology of lues versus yaws. Every general practitioner at times says to the patient, "Well, I think you've got the winter crud; there's a lot of

that going ground these days," an informal Bayesian inference. It is an unsettled question how much the explicit and formalized inverse probability machinery of the statistician should become part of the decision making by a busy doctor. Of course, even given a certain diagnosis, perhaps tentatively arrived at with the intention to be flexible about revising it should the predicted results of a therapeutic intervention fail to materialize in the usual fashion, it is common practice, within the category of patients who meet the diagnostic criteria, to pay attention to the pattern of symptoms that is relevant to treatment choice and to include in this those "extraneous" characteristics (e.g., age, family, income, unrelated concurrent illness) that themselves did not enter into the diagnostic decision proper.

There is nothing either wrong or particularly complicated about any of this. The only question is the extent to which formalization improves or impairs certain of these generally accepted clinical practices. Unfortunately, the behavior of a Bayes-theorem-computed inverse probability depends in somewhat complicated ways upon the distribution of sign validities, the relationship between valid and false-positive rates, the extent to which the independence assumption of the signs pairwise is not satisfied, how robust the inferred diagnostic *p* value is with respect to departures from those assumptions, differential responsiveness of error rate at different regions of the base rate continuum, and the like. It would seem that some rather large-scale but also intensive research by statisticians and clinicians would be in order.

I do not think it is safe to assume that because such actuarial refinements are not part of the everyday mental habits of practitioners in organic medicine, then we don't have to worry about it in psychopathology. There are probably important differences in the latter area. Furthermore, we still do not know the extent to which ordinary clinical practice of organic medicine commits more diagnostic errors than need be because of the extent to which the mathematics of clinical reference is not explicitly employed by the practitioner (Blois, 1980; Dawes, 1979; Dawes & Corrigan, 1974; Engelhardt, Spicker, & Towers, 1979; Goldberg, 1970, 1976; Gough, 1962; Holt, 1970, 1978; Kahneman, Slovic, & Tversky, 1982; Kleinmuntz, 1982; Meehl, 1954, 1956a, 1956b, 1956c, 1957, 1960, 1967; Sawyer, 1966; Sines, 1970). Finally, how one thinks about this and what kinds of research are conducted depend on how confident we are that the underlying psychopathology is intrinsically taxonic (categorical, "typal") versus nontaxonically multidimensional, where class concepts and qualitative predicates are only handy rubrics for roughly designating regions in an ontologically continuous descriptor hyperspace.

While the very title of this volume orients us toward revision, one hopes that the intellectual fretfulness of primates and the availability of taxpayer dollars will not induce us to attempt substantial revisions until a large mass of evidence, including experimental research, clinical trials, quantitative analysis of clinical file data, and exchange of experience by seasoned practitioners of various persuasions, puts us in a position to do something more than speculate or nitpick. A tremendous amount of work by able people and a lot of taxpayer money went into generating the DSM product, and it is foolish to tinker with it very much, let alone undertake a complete overhaul, because it isn't perfect, or because the results of an unavoidable compromise are not located precisely where one might prefer [them], given [one's] own theory and practice. Sometimes the best advice is that of the Baptist preacher, "Leave it lay where Jesus flang it." I bethink myself of how difficult it is for me, after 40 years on the Minnesota faculty, to interest myself in interminable discussions about how we should revise the written preliminary examination for the PhD so as to get a better assessment, reduce student anxiety, or whatever. A half century of observation (if I include my student days) reveals mostly primate meddlery, irrational optimism, a disinclination to consult the past, and the Hegelian swing of even short-term history!

It goes without saying that the most important developments one can anticipate that would make it rational to revise are substantive advances in our understanding of mental disorder. But there are also, I think, several metaquestions that it would be desirable to have "settled" (if not exactly solved) before the next round of major revision. The reader will discern that the "answers" to these metaquestions involve a mix of mathematical development of statistical

methods especially suitable for taxonomic problems, the usual impact of substantive developments upon methodology (no contemporary philosopher of science conceives of methodology as entirely prior to theory), and considerations of clinical utility. I repeat that it is a grave mistake to conflate this last class of questions with questions regarding the intrinsic science—that is, factual validity—of any proposed concept. There are four metaquestions that should meanwhile be addressed by high-competence investigators so that we will be in good methodological shape when the time for major revision arrives. Without dogmatism, I might go so far as to say that in my judgment until these four are answered, at least in the sense of a fairly high consensus among qualified individuals (there is no point in absolute democracy in a field like this!), we are probably not in a cognitive position that warrants a major revision being attempted.

First, what role should a conjectural etiology, when moderately to strongly corroborated, play in the taxonomic strategy? Here one must avoid a simplistic division into "known" and "unverified" etiology, assuming a sharp dividing line where none exists even in organic medicine, genetics, or other fields of knowledge. It is obvious on mere inspection of the present list of rubrics that etiological factors partially understood, and in which varying degrees of "strong influence" as causal factors (Meehl, 1972b, 1977) must have been taken into account at least behind the scenes, have been unavoidable. It will be necessary to have a uniform standard of proof rather than a double standard of methodological morals such as prevails in some quarters today. For example, there are clinicians in the medical and psychological professions who resist recognizing the genetic influences in major mental disorders or, while reluctantly recognizing them, would not want to split the nosology of affective disorders into unipolar and bipolar, despite the strong evidence available presently as to the reality of that distinction genetically and its correlates with certain aspects of the syndrome, course, and so on. Yet some of these same clinicians, while justly pointing out that an absolute hammer-blow unavoidable demonstration (there is no such thing as this in empirical science, of course) has not been given for the unipolar–bipolar distinction, will in their own diagnostic thinking rely upon highly speculative psychodynamics, or family factors, or other alleged causal influences, whose degree of evidentiary support at the present time is nowhere in the running with that for the biological distinctions made. This parallels some clinical psychologists who, because of hostility to medicine (or simply poor training at a second-rate school?), continue to decry all psychiatric diagnosis as "mere labeling" or "completely unreliable," refusing to read the quantitative evidence of diagnostic reliability developed in recent years, and then by some obscure mental process (which I confess myself quite unable to understand) proceed to substitute for such "unreliable" psychiatric nosology a batch of unproved, politicized social determiners, or flimsy psychodynamics inferred from an instrument with as low reliability and validity as the Rorschach! That is the sort of thing I mean by a double standard of epistemological morals.

Second, the strategic distinction between thinking in terms of dimensions and categories (types, species, taxa, disease entities) remains with us. While one can get by with a kind of compromise between these, the basic theoretical claim of a classification system should be methodologically clear, even if a sizable proportion of patients are not clearly sortable into one or the other (a different question). Sooner or later we should get clear about which of our nosological rubrics are intended to be rough designations of persons' location in a multidimensional descriptor space (whether phenotypic or genotypic, psychodynamic or genetic, that's not the point) and which rubrics have a genuine typological (taxonomic) theoretical intent. Thus, for instance, the very meaning of some standard terms in epidemiology and psychometrics, such as "false positive" and "base rate," which can be made tolerably clear on a taxonomic model, becomes fuzzy and—if the point is pressed—hardly interpretable on a nontaxonomic model. An adequate understanding of the philosophical and statistical aspects of this in relation to substantive theories of causation might properly lead us to abandon the idea of rubrics entirely for some subsets of conditions. For example, when I used to teach clinical psychology, in order to make this point I sometimes pushed the following (doubtless exaggerated) doc-

trine: There are several major mental disorders (e.g., schizophrenia, manic–depression, unipolar depression, delirium tremens, Alzheimer's disease) that are truly taxonomic in nature, and for which category rubrics are semantically strictly appropriate, not merely as rough ways of delineating regions in a continuous descriptor space. There is also, in my opinion, a true entity of the solid-gold essential psychopath[y] (sociopathic personality, asocial [type], amoral type). But when we get to the so-called neuroses and psychophysiological disorders of the neurotic kind, there is only one rubric (with the possible exception of the textbook obsessional neurosis)—namely, "psychoneurosis, mixed," a term no longer found in the official nomenclature. The distinctions within that mixed category are quantitative only; they are merely differing degrees of anxiety, depression, somatization, and defense mechanisms in the neurotic mixture. In the long run, it may be worth the trouble to teach clinicians to think more dimensionally than categorically and mold their verbal and inferential habits in those directions.

Third, we should get clearer than we presently are about the matter of sliding cuts on various indicators of an entity in relationship to base rates and various clinical populations in geographic, social classes, and the like, and the relevance of Bayes's theorem. [See the MCMI (Millon, 1977) for a promising beginning in the rise of base rates in identifying personality disorders of different prevalences.] In matters where extremely asymmetrical likelihoods exist for the combination of a small number of high-valid signs, the importance of the base rate, except for the most extreme values, is considerably reduced, and it is probably statistical pedantry to push some kind of Bayes's theorem algorithm onto working clinicians under such circumstances. I think that more mathematical analysis in relationship to the diagnostic habits of practitioners is in order here before altering the character of a psychiatrist's or clinical psychologist's education in this regard. Nobody acquainted with my writings would suspect me of being even faintly "antistatistical" in my biases; but I believe we should think like behavioral engineers in considering ourselves and others as clinical practitioners, taking into account what kinds of psychological disruptions in diagnostic cognitive activity could take place that might reduce net efficiency, even though the underlying mathematical model makes it look like an improvement.

Finally, at the risk of projecting my own current research interests, I would say that a desideratum for the next major revision is agreement upon the general taxometric problem as such, which I see as having two elements: (a) Is a taxometric procedure in psychopathology aimed at anything more than identifying phenotypic clusters; and, if it is, (b) which of the available formal taxometric methods (if any!) have shown themselves capable of detecting an underlying causal structure (whatever its biological or social nature), being meanwhile free of any appreciable tendency to detect taxonic structures that aren't there (Meehl, 1979)? I think it not unduly optimistic to opine that we will have a pretty clear answer to the second question before the end of this decade (Grove & Andreasen, 1986; Meehl & Golden, 1982; Sneath & Sokal, 1973).

Acknowledgment

This chapter is reprinted from Meehl (1986, pp. 215–231). Copyright 1986 by The Guilford Press. Reprinted by permission.

References

Allport, G. W. (1937). *Personality: A psychological interpretation*. New York: Henry Holt.

Blois, M. S. (1980). Clinical judgment and computers. *New England Journal of Medicine*, *303*, 192–197.

Cattell, R. B. (1946). *Description and measurement of personality*. New York: World Book Company.

Cronbach, L. J., & Meehl, P. E. (1955). Construct validity in psychological tests. *Psychological Bulletin*, *52*, 281–302.

Cureton, E. E. (1957). Recipe for a cookbook. *Psychological Bulletin*, *54*, 494–497.

Dawes, R. M. (1979). The robust beauty of improper linear models in decision making. *American Psychologist*, *34*, 571–582.

Dawes, R. M., & Corrigan, B. (1974). Linear models in decision making. *Psychological Bulletin*, *81*, 95–106.

Engelhardt, T. H., Spicker, S. F., & Towers, B. (Eds.). (1979). *Clinical judgment: A critical appraisal*. Boston: Reidel.

Feyerabend, P. K. (1970). Against method: Outline of an anarchistic theory of knowledge. In M. Radner & S. Winokur (Eds.), *Minnesota studies in the philosophy of science: Vol. 4. Analysis of theories and methods of physics and psychology*. Minneapolis: University of Minnesota Press.

Finn, S. E. (1982). Base rates, utilities, and DSM-III: Shortcomings of fixed-rule systems of psychodiagnosis. *Journal of Abnormal Psychology*, *91*, 294–302.

Finn, S. E. (1983). Utility-balanced and utility-imbalanced rules: Reply to Widiger. *Journal of Abnormal Psychology*, *92*, 499–501.

Goldberg, L. R. (1970). Man versus model of man: A rationale plus some evidence for a method of improving on clinical inferences. *Psychological Bulletin*, *73*, 422–432.

Goldberg, L. R. (1976). Man versus model of man: Just how conflicting is that evidence? *Organizational Behavior and Human Performance*, *16*, 13–22.

Gough, H. G. (1962). Clinical versus statistical prediction in psychology. In L. Postman (Ed.), *Psychology in the making*. New York: Knopf.

Grove, W. M., & Andreasen, N. C. (1986). Multivariate statistical analysis in psychopathology. In T. Millon & G. L. Klerman (Eds.), *Contemporary directions in psychopathology* (pp. 153–168). New York: Guilford Press.

Hoch, P., & Polatin, P. (1949). Pseudoneurotic forms of schizophrenia. *Psychiatric Quarterly*, *3*, 248–276.

Holt, R. R. (1970). Yet another look at clinical and statistical prediction. *American Psychologist*, *25*, 337–339.

Holt, R. R. (1978). *Methods in clinical psychology: Vol. 2. Prediction and research*. New York: Plenum Press.

Kahneman, D., Slovic, P., & Tversky, A. (1982). *Judgment under uncertainty: Heuristics and biases*. London: Cambridge University Press.

Kleinmuntz, B. (1982). Computational and noncomputational clinical information processing by computer. *Behavioral Science*, *27*, 164–175.

Meehl, P. E. (1954). *Clinical versus statistical prediction: A theoretical analysis and a review of the evidence*. Minneapolis: University of Minnesota Press.

Meehl, P. E. (1956a). Clinical versus actuarial prediction. In *Proceedings of the 1955 Invitational Conference on Testing Problems* (pp. 136–141). Princeton, NJ: Educational Testing Service.

Meehl, P. E. (1956b). The tie that binds. *Journal of Counseling Psychology*, *3*, 163–164.

Meehl, P. E. (1956c). Wanted—a good cookbook. *American Psychologist*, *11*, 263–272.

Meehl, P. E. (1957). When shall we use our heads instead of the formula? *Journal of Counseling Psychology*, *4*, 268–273.

Meehl, P. E. (1959). Some ruminations on the validation of clinical procedures. *Canadian Journal of Psychology*, *13*, 102–128.

Meehl, P. E. (1960). The cognitive activity of the clinician. *American Psychologist*, *15*, 19–27.

Meehl, P. E. (1962). Schizotaxia, schizotypy, schizophrenia. *American Psychologist*, *17*, 827–838.

Meehl, P. E. (1964). *Manual for use with checklist of schizotypic signs*. Minneapolis: Psychiatry Research Unit, University of Minnesota Medical School.

Meehl, P. E. (1967). What can the clinician do well? In D. N. Jackson & S. Messick (Eds.), *Problems in human assessment* (pp. 594–599). New York: McGraw-Hill.

Meehl, P. E. (1972a). Reactions, reflections, projections. In J. N. Butcher (Ed.), *Objective personality assessment: Changing perspectives* (pp. 131–189). New York: Academic Press.

Meehl, P. E. (1972b). Specific genetic etiology, psychodynamics and therapeutic nihilism. *International Journal of Mental Health*, *1*, 10–27.

Meehl, P. E. (1973). Why I don't go to case conferences. In P. E. Meehl, *Psychodiagnosis: Selected papers*. Minneapolis: University of Minnesota Press.

Meehl, P. E (1974–1975). Genes and the unchangeable core. *Voices: The Art and Science of Psychotherapy*, *38*, 25–35.

Meehl, P. E. (1975). Hedonic capacity: Some conjectures. *Bulletin of the Menninger Clinic*, *39*, 295–307.

Meehl, P. E. (1977). Specific etiology and other forms of strong influence: Some quantitative meanings. *Journal of Medicine and Philosophy*, *2*, 33–53.

Meehl, P. E. (1979). A funny thing happened to us on the way to the latent entities. *Journal of Personality Assessment*, *43*, 563–581.

Meehl, P. E. (1983). Subjectivity in psychoanalyt-

ic inference: The nagging persistence of Wilhelm Fliess's Achensee question. In J. Earman (Ed.), *Minnesota studies in the philosophy of science: Vol. 10. Testing scientific theories*. Minneapolis: University of Minnesota Press.

Meehl, P. E. (1986). Diagnostic taxa as open concepts: Metatheoretical and statistical questions about reliability and construct validity in the grand strategy of nosological revision. In T. Millon & G. L. Klerman (Eds.), *Contemporary directions in psychopathology* (pp. 215–231). New York: Guilford Press.

Meehl, P. E., & Golden, R. (1982). Taxometric methods. In P. Kendall & J. Butcher (Eds.), *Handbook of research methods in clinical psychology*. New York: Wiley.

Meehl, P. E., & Rosen, A. (1955). Antecedent probability and the efficiency of psychometric signs, patterns, or cutting scores. *Psychological Bulletin*, *52*, 194–216.

Millon, T. (1977). *The Millon Clinical Multiaxial Inventory manual*. Minneapolis, MN: National Computer Systems.

Murphy, E. A. (1964). One cause? Many causes?: The argument from a bimodal distribution. *Journal of Chronic Diseases*, *17*, 301–324.

Murray, H. A. (1938). *Explorations in personality*. London: Oxford University Press.

Pap, A. (1953). Reduction sentences and open concepts. *Methodos*, *5*, 3–30.

Pap, A. (1958). *Semantics and necessary truth*. New Haven, CT: Yale University Press.

Peppard, T. A. (1949). Mistakes in diagnosis. *Minnesota Medicine*, *32*, 510–511.

Reichenbach, H. (1938). *Experience and prediction*. Chicago: University of Chicago Press.

Rorer, L. G., Hoffman, F. J., LaForge, G. E., & Hsieh, K. E. (1966). Optimum cutting scores to discriminate groups of unequal size and variance. *Journal of Applied Psychology*, *50*, 153–164.

Sawyer, J. (1966). Measurement and prediction, clinical and statistical. *Psychological Bulletin*, *66*, 178–200.

Sines, J. O. (1970). Actuarial versus clinical prediction in psychopathology. *British Journal of Psychiatry*, *116*, 129–144.

Sneath, P. H. A., & Sokal, R. R. (1973). *Numerical taxonomy*. San Francisco: Freeman.

Widiger, T. A. (1983). Utilities and fixed diagnostic rules: Comments on Finn (1982). *Journal of Abnormal Psychology*, *92*, 495–498.

CHAPTER 9

Contemplations on Meehl (1986)

The Territory, Paul's Map, and Our Progress in Psychopathology Classification (or, the Challenge of Keeping Up with a Beacon 30 Years Ahead of the Field)

MARK F. LENZENWEGER

Paul E. Meehl (1920–2003; see Figure 9.1) was recognized by many as perhaps the leading intellectual light in psychological science of the past century. The breadth and depth of his interests, as well as his contributions to the scientific psychological corpus, remain peerless. The numerous tributes written in his honor attest to his impact, creativity, and generativity for psychology writ large, as well as for psychiatry, philosophy of science, and law (see the special tribute issues of the *Journal of Clinical Psychology* [2005, 61(10)], the *Journal of Abnormal Psychology* [2006, 115(2)], *Applied and Preventive Psychology* [2004, 11(1)], and *Developmental Psychopathology* [2003, 15(3)]). Simply stated, a Mozart comes along rarely. We were fortunate that Paul devoted his considerable intellectual energies to psychological science (particularly psychopathology) as he helped to chart the course for the discipline in a way that remains timeless. Thus, when Theodore Millon called to ask me to reflect on Paul's contribution to the 1986 *Contemporary Directions in Psychopathology* volume, I hesitated and wondered, "What can be said that Paul Meehl did not already mention in the verdant original work?" Ted engaged in some gentle arm twisting and suggested I comment freely on the chapter. I reread and pondered the 1986 piece; I thought a bit more about Ted's invitation; and here we are.

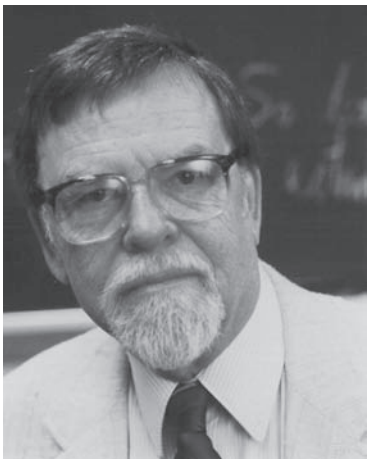


FIGURE 9.1. Paul E. Meehl, PhD (1920–2003). Photograph provided by and reproduced with permission of Leslie J. Yonce, PhD.

One of the hallmarks of Meehl's thinking was that it was typically well ahead of its time (and the field) by a good 30 years

or so. Consider just one example: his seminal model of schizotaxia, schizotypy, and schizophrenia, published in 1962. At a time when the field was still consumed with ideas regarding the psychosocial origins of schizophrenia (e.g., the schizophrenogenic mother; marital schism and skew) and was caught in its own double bind of sorts with competing psychoanalytic models of schizophrenia, Meehl proposed a genetically based, neurodevelopmental model of the origins and pathogenesis of schizophrenia. What is remarkable is that it took the field 30 years to realize that most of the major components in his model of the illness were quite on target, though debate still rages over the precise nature/structure of the genetic liability for the illness (mixed vs. oligogenic vs. latent-trait vs. multifactorial polygenic-threshold models vs. genetic structural variants). For example, the neural basis of what Meehl (1962) called "synaptic slippage" is the focus of modern conceptualizations of the neural dysfunction in the illness, though it has been given different names by different theoreticians (e.g., "cognitive dysmetria," "reduced developmental synaptic connectivity," "intermittent degradation").

Paul liked to work in territory that was poorly mapped—or, if a map existed, he decidedly recharted it (e.g., clinical vs. statistical prediction, Meehl, 1954; construct validity, Cronbach & Meehl, 1955; see also Maher & Gottesman, 2005). His contribution to the 1986 volume had the same quality as these prior substantive efforts. The chapter was titled "Diagnostic Taxa as Open Concepts: Metatheoretical and Statistical Questions about Reliability and Construct Validity in the Grand Strategy of Nosological Revision" (it is reprinted as Chapter 8 of the present volume),[1] and it was (and remains) a treasure trove of intellectual nuggets. In my view, Meehl's (1986) chapter should be read and pondered by current (and aspiring) DSM architects if the classification business in psychopathology is ever to move forward appreciably in terms of validity and predictive power.[2] What I have done, therefore, in this chapter is to take Ted Millon at his word: I contemplate the issues raised by Meehl (1986) in light of where, in my view, we currently stand in the field—explicating some issues in greater detail to underscore their meaning and importance, as well as pointing to areas where progress is still needed. In addition, I address the four "metaquestions" that Meehl left for us at the end of his 1986 chapter; these, he argued, were "big questions" that needed to be resolved (or at least "settled") before the next big revision of the nomenclature. As many readers will realize, and as I discuss below, these metaquestions were certainly not settled prior to the publication of DSM-IV (American Psychiatric Association, 1994). But what of DSM-V and the four metaquestions?

I would now like to reflect on a number of the points raised by Meehl (1986) in his chapter. I must confess that my survey consists of what grabbed my eye in the original piece; therefore, my commentary is necessarily selective and nonexhaustive in content and scope. I have also sought to highlight items that I believe may benefit contemporary psychiatry and clinical psychological science, particularly as nearly 25 years' worth of PhDs and MDs have earned their degrees since the original publication appeared. One of the wonderful things about Meehl's writing is that it needs to be read and reread because new insights and ideas emerge from each reading. This view is not idiosyncratic to yours truly, but has been the collective impression of long-time readers of Meehl's works (see Waller, Yonce, Grove, Faust, & Lenzenweger, 2006).

Let us begin with the reliability and validity issue that Meehl raised early in his chapter. He suggested that in general, the effort after reliability was a productive venture, in that it probably helped in most instances to increase the net construct validity of the disorders listed in the diagnostic manual (hereafter DSM). He spent some time explaining the complex relationships that can exist between reliability and validity, which bear restatement and examination here.

High Reliability Is Not Always Your Friend

The reliability and validity issues, as well as the "attenuation paradox" (see Loevinger, 1954), were addressed by Meehl (1986); however, this part of the text was highly concentrated, and the profound implications of his remarks may have eluded many on a first reading. It is generally assumed that reliabil-

ity is a good thing to seek in the area of diagnostic assessment. Although long known in the field of measurement and psychometrics, the value of reliability made itself known in the psychopathology diagnostic process after a series of studies in the early to mid-1960s highlighted difficulties with the execution of psychiatric diagnosis. Clearly things improved in terms of diagnostic reliability after the advent of DSM-III (American Psychiatric Association, 1980), with its explicit criteria and diagnostic guidelines. However, appreciable gains in the reliability of diagnosis since DSM-III-R (American Psychiatric Association, 1987) have been modest and limited to just a few areas (e.g., personality disorders). Meehl alerted readers to the situation whereby the validity of assessments may actually *decrease* if efforts at maximizing reliability in assessment or measurement are pushed too hard. Contemporary workers should continue to keep this admonition in mind, as it does happen in the real world, so to speak. For example, as the definition of schizophrenia was narrowed excessively to positive symptomatology in the DSM-III criteria set, one saw an important criterion of validity (i.e., familial schizophrenia) squeezed right out of the construct, as evidenced by two studies (Abrams & Taylor, 1983; Pope, Jonas, Cohen, & Lipinski, 1982). Moreover, we have seen that training raters of psychopathological phenomena to achieve high levels of reliability does not in any way guarantee validity. This was demonstrated by the clever study of Hooley and Richters (1991), which showed that the ratings of expressed-emotion indicators by intelligent undergraduate raters (Harvard students) possessed high reliability, but little validity. The moral here is straightforward: Questing after high reliability may not always result in increases in validity.[3]

The Notion of "Open Concepts"

The notion of "open concepts" (Pap, 1953) ran through and through Meehl's chapter; it was mentioned specifically over a dozen times. In short, if one got nothing else out of Meehl (1986) other than an appreciation for open concepts in relation to psychiatric diagnosis, then the chapter could be viewed as a pedagogical success by any measure. (See Meehl, 1978, as well as his rich 1977 paper on specific etiology.)[4]

In his classic "two knights" paper of 1978 (the paper's title refers to Sir Karl Popper and Sir Ronald Fisher), Meehl distinguished among three kinds of openness in constructs:

> (a) openness arising from indefinite extensibility of our provisional list of operational indicators of the construct; (b) openness associated with each indicator singly, because of the empirical fact that indicators are only probabilistically, rather than nomologically, linked to the inferred theoretical construct; and (c) openness due to the fact that most of our theoretical entities are introduced by an implicit or contextual definition, that is, by their role in the accepted nomological network, rather than their inner nature. (p. 815)

With respect to psychiatric diagnosis, both points (a) and (b) are rather central and critical. The joint meaning of the two kinds of openness with respect to diagnosis implies that (a) the list of indicators for a given disorder is necessarily incomplete and could be extended/expanded (thus a given criteria set is not definitive); and (b) the indicators of a disorder are linked to the underlying disease or disorder construct via stochastic or probabilistic relations (i.e., the signs and symptoms of a disorder are not directly linked to the underlying construct, but are merely [fallibly] associated with the construct). The third kind of openness as defined in (c) above speaks to our field's lack of understanding of the inner nature of the constructs that we specify, but our need to embed them in our theories, models, and analytic approaches. Whether the topic of a theoretical discussion or an open circle in a path diagram depicting latent variables (which prompted Meehl to call this type of openness "Little Orphan Annie" eyes as suggested by the comic strip character of yesteryear). It is important to admit that we still have precious little understanding of the pathogenesis and pathology involved in most forms of psychopathology. This state of affairs is reflected, therefore, in our lack of a definitive understanding of the mode of action of most psychiatric medications.[5] Our primary constructs in psychopathology are defined in an external sense or by their implicit roles in our models; stated differently, the conceptual context in which

they are embedded defines them. Ultimately, our full understanding of constructs (gained through empirical research) will emerge, and the inner nature of such open concepts will be illuminated—in much the same way as the inner nature of the gene emerged with the discovery of DNA, although prior to this discovery the notion of a gene existing was highly plausible and helpful in explanatory networks.[6]

Don't Let the DSM Rubrics Get You Down

Meehl always encouraged psychopathologists to think outside the proverbial box when it came to identifying meaningful parsings in the realm of mental disorder. Remember that this was the fellow who taught the field to conceive of latent constructs (e.g., love, anxiety, schizotypy, and perfectionism are all latent constructs) and construct validity at a time when most psychological scientists were only willing to think in terms of observable behaviors.[7] Thus it should come as no surprise that he advised workers not to let themselves be excessively hampered by received rubrics in the search for order in psychopathological phenomenology. Experimental psychopathology and clinical psychological science, it seems, have been more comfortable exploring the manifestations of severe psychopathology that fall outside what appears in print on the pages of DSM. Such concepts as schizotypy (the liability for schizophrenia), psychopathy (as distinct from the sociodemographically defined antisocial personality disorder), and borderline personality organization (e.g., Kernberg's [1984] phenomenological organizing framework for personality pathology) serve as only a few examples. Each of these theoretical concepts has spawned rich research literatures that have illuminated important aspects of more traditionally defined psychiatric conditions. For example, the laboratory study of schizotypy has generated a remarkable corpus of findings that support schizotypic psychopathology as an alternative manifestation of schizophrenia liability. To work outside the realm of DSM disorders in psychopathology research requires persistence and tenacity, however, as many reviewers and funding agencies only feel comfortable dealing with papers and proposals seeking to investigate "established" (if you will) entities. This is not completely unexpected, as review processes (including National Institute of Mental Health [NIMH] study sections) are characterized by an inherently conservative spirit and, according to some long-time observers, "groupthink" processes, which limit the adventurousness of all concerned.

Along the lines of advocating that we not let established rubrics get us down, I would like to suggest to the psychopathology research community (which includes, of course, psychiatry and the DSM-V microcommunity) that we consider moving away from the 100-year-old "signs and symptoms" phenomenological approach to defining psychopathology, and move toward organizing our understanding of psychopathology in terms of neural circuits, neurobehavioral systems, and validated environmental stressors.[8] In short, should we abandon the phenotypic-indicator-based approach to diagnosis and talk about assessing neural systems? For example, would it make sense to move away from discussing the many varied anxiety disorders—which may or may not correspond to the way that nature is really organized—and speak of the neural circuits known to be related to anxiety and negative affect systems? Such progress would necessarily depend on advances in our understanding of the neural circuitry of the brain, as well as of the neurobiology of complex human behaviors, but this strikes me as an achievable goal. There are, of course, those who object to the "hobbyhorse" nature of much neuroimaging research, as well as those who argue cogently that present-day neuroimaging techniques are simply too coarse (resolution is limited; important neural events happen at the level of cells) and too slow (discrete mental events of greatest interest at the level of greatest interest are long past by the time the hemodynamic response catches up in the form of a blood-oxygen-level-dependent [BOLD] signal). Nonetheless, I think it would behoove the field to attempt a classification system based on the pathology of neural circuits and neurobehavioral processes, with relevant environmental inputs (e.g., exposure to tetrahydrocannabinol in those carrying a liability for schizophrenia). This is some-

thing the DSM-V committee should actively consider encouraging as a "research system in need of further study."

The "Context of Discovery" versus Anxiety in Bethesda

Meehl strongly supported exploratory thinking, as well as empirical research carried out within the "context of discovery." He advocated generating ideas from the armchair and probing one's data well beyond a priori hypotheses, and he had enormous respect for the creative process in scientific research. So, one might ask, just what exactly is the "context of discovery"? Hans Reichenbach (1938) proposed that this context, as distinct from the "context of justification," represented the part of the scientific creative process that was to some extent outside the purview of the logician and could be carried on in a highly nonlinear and emergent manner, offering few insights into its inner nature. Stated differently, a scientist can engage in all sorts of mental activities—hypothesis generation; theory formulation; juxtaposition of constructs, processes, and temporal parameters in a divergent manner—in the search for knowledge in the context of discovery. The context of discovery does often, of course, result in testable and falsifiable conjectures (the work of the context of justification), but in Reichenbach's view, the discovery context is the playground of intellectual ideas and creativity. He felt that this intellectual "free-for-all" should not be criticized, but rather should be viewed as an important part of the scientific enterprise.

Meehl's enthusiasm for the context of discovery reflects a certain spirit—a certain zeal for the exploratory and creative process in psychological science. In my view, contemporary psychopathology research, including efforts to improve the classification system, could use an infusion of this zeal for inquiry. Clearly, creative insights and proposals can be developed a priori, based entirely on theory or conceptual models, and/or they can be gleaned from post hoc analyses of existing data sets. Notions such as "expanded phenotypes" for certain forms of psychopathology (e.g., schizophrenia), "endophenotypes" (Gottesman & Gould, 2003), and deficits in smooth-pursuit eye movements in schizophrenia (Holzman, Proctor, & Hughes, 1973) have grown out of the rich matrix of the context of discovery, to name just three interesting advances consistent with Meehl's enthusiasm for the context of discovery.

At this juncture in considering the "context of discovery," the issue of post hoc analysis and its inherent value requires explicit comment. Whereas many psychologists and psychiatrists have been taught that in empirical research one needs to formulate a hypothesis, gather data, run a statistical test, reject the null hypothesis, and write up the results, those of us who are experienced researchers know that this is not really how the process works. We do begin with ideas (we can even "conjecture from the armchair," as Meehl was fond of saying); we do collect data; but then we must deeply probe the data that took considerable time, effort, and resources to collect. That considerable gold, in the form of findings, can be mined in post hoc analyses is well known; that manuscript and grant proposal reviewers are made anxious by post hoc analyses of data is also well known. The moral here should be self-evident, and I believe Meehl would have endorsed this gentle admonition: Allow yourself the freedom to dive into the context of discovery in your work. Do not be hampered by the desire to hew to the line of NIMH interests in your applications,[9] or by the publication preferences (biases) of journal editors when reporting on the results of post hoc analyses in the "Results" or "Discussion" sections of manuscripts. It is well known that most research grants are for the support of what Kuhn (1970) called "normal science," the work of filling in the blanks and connecting dots of existing models. But thinking that potentially reflects paradigm shifts—the "big" leaps forward—is not likely to be received well (at least initially) by the scientific masses, or likely to be funded by conservative funding agencies. How can the spirit of Meehl's enthusiasm for the "context of discovery" be built into the funding process—to fix, in part, what has become thought of as the "broken pipeline" (National Institutes of Health, 2008)? In the spirit of innovation, I would suggest that the NIMH should consider creating "context of discovery" grant awards, whereby worthy recipients would be identified and unrestricted awards would be made to them—something

like a Career Development (K) Award without the requirement of concurrent R0-1 (or other mechanism) funding, or a Method to Extend Research in Time (MERIT)-Award-like mechanism without the prerequisite of numerous years of prior NIMH involvement. Perhaps a blue-ribbon panel of high-impact researchers could serve to advise the NIMH about potential recipients of such "context of discovery" awards; perhaps the top 10 publications of a potential recipient would serve as the basis for evaluation (not total publications, not total number of prior awards). In regard to post hoc analysis, I think reviewers and journal editors should routinely ask investigators, "What interesting things did you learn in the post hoc analysis of your data? Would you consider including some description of these findings, with appropriate caveats (i.e., pending replication and so on), in your 'Discussion' section as food for thought for others?"

The Study of Subsyndromal (Fringe) Cases

Just as we should not let the established DSM rubrics limit our research interests or sense of creativity, we should be sure to recruit subsyndromal cases of a disorder (construct) of interest for our studies, as well as to identify them in a data set in order to use the leverage provided by their group membership to advance knowledge. Meehl (1986) was clear to note the advantages associated with the enhanced attention to detail that came with DSM-III, and he suggested that there would probably be a net increase in construct validity associated with this increase in orderliness and explicit description (e.g., tightening up the diagnostic definition of schizophrenia would mean that the construct of schizophrenia would benefit in terms of increased construct validity, as noted above). That said, Meehl advocated the study of subsyndromal or "fringe" cases that inhabit the borders of established constructs. For example, consider the large number of individuals who inhabit the realm of schizotypy, but are not clinically psychotic or manifesting fulminant schizophrenic illness. Study of such individuals has genuinely advanced our understanding of schizophrenia as well as supplied an empirical basis for methodological decisions, such as the inclusion of this group in genetic analyses focused on schizophrenia. The important points here are that (1) subsyndromal cases should be studied, and (2) they need to be identified in data sets.

These points seem rather mundane, but they are nontrivial. If we do not study (i.e., collect data on such folks) *and* identify the subsyndromal cases in databases and, by implication, statistical analyses, then nothing can be learned about them. If a cell for them does not exist in an analysis of variance (or if it contains no subjects), it is not going to tell us much about phenomena that might be of great potential interest to us. Thus, if we are only working on DSM-defined constructs in our research, and subsyndromal variants are not assessed and included in analyses, we are likely to miss a valuable opportunity to advance our knowledge in psychopathology. An important corollary here is that the boundaries of most (if not all) psychopathology entities have some degree of fuzziness (some are fuzzier than others), and thus the issue of subsyndromal cases really concerns the boundaries of the constructs at issue in DSM or other nomenclatures.[10]

Just What Do You Believe about the Constructs in DSM?

One of the more engaging sections of Meehl's (1986) chapter focused on what he described as possible intellectual views of the DSM constructs. He regarded one as "admirable," one as "criticizable but fairly harmless," and only the third as "scientifically malignant" (p. 220). Although I suspect that most research-oriented psychiatrists and psychologists as well as other psychopathology researchers would not subscribe to Meehl's third point of view on the DSM constructs, it is nonetheless possible to encounter some (perhaps more likely among practitioners) who do.

DSM is a powerful professional–social–political document. This claim does not speak to the validity of the document; rather, it notes how the document affects the conduct of research, research proposal development, the treatment of patients, and public health planning. Meehl (1986) was careful to note that the manner in which one regards the document can have profound effects on

research and clinical progress, as well as the emergence of new ideas and themes for revision. My experience, based on many discussions with researchers, practitioners, and students over the years, suggests that Meehl's analysis is worth repeating here. Thus Meehl argued that there are those who view the diagnostic constructs explicated in DSM as representing symptomatically defined syndromes that (1) hang together (descriptively or statistically), (2) have some unknown or unspecified etiology (which is likely to be shared by those diagnosed with the conditions), and (3) have some communicative value. This reveals a reasonable intellectual approach to the thorny problem represented by the classification and treatment challenges represented by psychopathology.

There are two other viewpoints, speaking coarsely (there is probably some gradient across these viewpoints), that may be seen among those working with the DSM system. The more common one is reasonably harmless, although intellectually impoverished. The other is, as Meehl (1986) noted, "scientifically malignant" (p. 220)"; it is the view that somehow DSM articulates the *truth*, as it were. The view that is relatively harmless is that the constructs defined in DSM are somehow correct and the best we can do for now, with an implied openness to revision down the line. This view is OK, so to speak, just as long as those who think that DSM is the best we can do for now do not get in the way of those seeking to explore other entities or models of psychopathology. In other words, with respect to new roads and avenues of exploration in alternative approaches to psychopathology, adherents of this view cannot use this view as a basis for blocking progress. As Bob Dylan sings, "Your old road is rapidly agin',/Please get out of the new one, if you can't lend your hand" in "The Times They Are A-Changin'."

The mistaken and intellectually indefensible view that the DSM constructs represent the truth is indeed bad news. Not only can such a misguided view get in the way of progress; it is also grounded in a view of entities and operational definitions that has long been abandoned by informed philosophers of science and psychological science researchers. What is meant here? If someone believes that the *meaning* of the conditions in DSM is defined by the list of signs and symptoms (or DSM-defined diagnostic rules), then one is implicitly subscribing to an "operationism" (so-called operational definitions) that has long been discounted in philosophy and psychology. Conditions (or diseases) in traditional organic medicine are defined not merely by signs and symptoms; rather, they represent (implicitly) information regarding etiology, pathophysiology, and so on. If information about etiology and pathophysiology is absent from such constructs in organic medicine, then, at a minimum, the construct in question is viewed as in need of further research. In short, the constructs defined explicitly in DSM[11] do not represent the truth, so to speak, and they do not represent the intellectual end of the line. For readers who might think that this is self-evident stuff, I might only remind them of the frequent precursor to the modal question that follows many conference talks or colloquia—namely, "According to DSM-IV, yadda, yadda, yadda." I think that a routine rejoinder to such a preface to commentary after talks should be "Just what do you believe about the constructs in DSM?"

Densifications in the Multivariate Descriptor Hyperspace

If one conceives of phenotypic (or endophenotypic) indicators as defining a multivariate hyperspace, this space may be characterized by a topography that resembles a uniform surface (imagine a blanket) with little to no variation or perhaps a steady gradient of variation, on the one hand; or by a variety of quasi-independent clumpings (imagine satellites floating in space); or by all manner of possibilities in between. The clumpings may represent what Meehl (1986) referred to as "densifications." Our work has yet to determine the precise nature of these densifications: Do they represent quantitative variation along continua, or dimensions, or genuine qualitative discontinuities? Meehl suggested that before the next big revision of DSM, the system's architects should decide "when the orientation should be taxonomic versus dimensional." This recommendation remains essentially as valid today as it was in 1986. Despite the flourishing taxometric literature and what to some seems to be the "bandwagon science" quality of taxometric

exploration, DSM remains fundamentally committed to a categorical model of psychopathology. Well-designed taxonomic studies using taxometric methodology appropriately, as well as other approaches (e.g., finite-mixture modeling, latent-growth-mixture modeling), remain sorely needed to generate the empirical data needed to satisfy the demand of Meehl's recommendation (see Lenzenweger, 2004).

There Are No "Operational" Definitions in DSM

Although I have alluded to this in note 11, it is worth stressing explicitly here that there are no "operational definitions" in DSM. There were none in DSM-III, DSM-III-R, DSM-IV, or DSM-IV-TR, and there will not be any in DSM-V. The manual does not specify the operations that one uses to make a diagnosis in any strict sense. Though we do not seek a set of Bridgman-approved (Bridgman, 1927) operational definitions in DSM (as this would reflect adherence to an abandoned philosophical/scientific position), more guidance from the architects of DSM as to the actual diagnostic process would be useful. By this I do not mean simply narrative in the text that *n* of *N* symptoms are required for a given disorder. Rather, can more specification of the diagnostic operations to be followed be provided in future diagnostic systems?

In this context, it is essential to note that the signs and symptoms of the disorders in DSM do not constitute the entities themselves. Meehl (1986) emphasized that such an intellectual error would only serve to propagate further confusion and/or dogmatism. As noted above, Meehl devoted considerable time to developing the notion of a "latent construct" (MacCorquodale & Meehl, 1948)—not surprisingly, since he "wrote the book," as it were, on construct validity and the nomological net (Cronbach & Meehl, 1955; see also Hempel, 1952)—and contemporary psychopathologists may need to be reminded of the necessity of the latent-construct formulation. To hypothesize the existence of a latent construct in psychopathology is not just some sort of high-flown intellectual endeavor or escapade; rather, it is an essential theoretical refinement for understanding psychopathological entities. I realize that I am probably "preaching to the choir" for many readers, but the notion of a latent construct is still not well understood by many practicing psychologists and psychiatrists. Simply stated, this distinction (latent construct vs. observable indicators) is needed, and it promotes further substantive and statistical analysis: For example, factor analysis, taxometric analysis, and other multivariate techniques require the assumption of a latent entity or construct. Just as genetics and genomics have found the phenotype–genotype distinction to be not only useful but a scientific necessity, so should psychopathology research more fully embrace the notion of latent constructs. In short, defining a disorder solely in terms of signs and symptoms is just really poor thinking. As Meehl said, "Such an operational definition is a fake" (p. 222). The moral here is straightforward: Everything in DSM is a hypothetical entity and thereby represented by a latent construct, and one should not conflate the signs/symptoms of a disorder with the underlying entity. One way to improve the validity of the DSM approach is to continue to encourage discovery of what will eventually come to be regarded as criteria of validity for a disorder, and such criteria of validity (*à la* Cronbach & Meehl, 1955; see also Maher & Gottesman, 2005) should come in the form of endophenotypes (Gottesman & Gould, 2003) and neural processes underpinning behavioral and/or psychological processes that are discovered through laboratory studies (cf., Lenzenweger & Hooley, 2003).

There Is Always a Role to Be Played by Clinical Observation

Meehl's (1986) chapter clearly had an air of quantitative sophistication, and the value of quantitative thinking was clearly implied throughout (see Waller et al., 2006). However, many people mistakenly think that Meehl only advocated a statistical approach to organizing information regarding psychopathology. Nothing could be further from the truth: He strongly advocated the value of clinical observation. His manual of schizotypic signs grew out of years of observations drawn from clinical practice; he maintained a psychotherapeutic prac-

tice well into the later years of his career; and, believe it or not, he had a psychoanalytic couch in his office at the University of Minnesota. Clearly, the impressive observational skills of the clinician were not lost on Meehl. In this context, I should merely like to note that clinical observation remains a crucial vantage point for potential additions to or revisions of the DSM system. A caveat is needed here to avoid a potential misreading of the foregoing text. In short, we should not confuse how we *collect* data (e.g., clinical observation) with how we *combine* those collected data for the purposes of prediction or whatever. The value of clinical observation is not to be underestimated, especially in the age of advanced technological methods (e.g., neuroimaging) and complex and sophisticated statistical methods. No data collection method or statistical procedure or test is self-interpreting; it cannot tell us where to look; it cannot resolve problems. We need only consider the classic blood pressure controversy (quantitative vs. qualitative variation) to gain an appreciation for the value of clinical observation in conjunction with complex statistical analyses of data (see Swales, 1985; cf. Lenzenweger, McLachlan, & Rubin, 2007).

Where Does Neuroimaging Fit In?

At the time Meehl (1986) wrote his original chapter, few would have envisioned the manner in which neuroimaging would have taken off as an approach to the study of human brain functioning. Not only were the early technologies of neuroimaging (computed tomography and positron emission tomography) only just beginning to be applied to psychopathology; there was little in the way of a guiding strategic approach for neuroimaging itself in the study of human brain function (e.g., "region of interest" approaches were essentially nonexistent). Thus Meehl did not really address this emerging technology. However, with the benefit of nearly 25 years' progress, and with the clear-cut relevance of neuroimaging now well established in psychopathology research, we might wonder how the data derived from this powerful family of methodologies should be used in the explication of disorders in DSM. Notwithstanding the concerns of some who view neuroimaging techniques as inherently too slow and too coarse to really get at the discrete neural events of greatest interest to psychopathologists, we might consider a bold (no pun intended) proposal: With the help of knowledge gained from neuroimaging studies, might it be possible to abandon the phenotypic (signs and symptoms) approach to diagnosis altogether (as implied by the proposal above regarding neural circuits as a basis for classification/description)? A shorter-term goal for neuroimagers might be to understand the basis of specific signs or symptoms (eschewing the need to organize these by categorical phenotypes in the DSM); by way of a longer-term goal, disturbances in established and well-understood neural circuits (which cut across any number of DSM-defined constructs) would become the bases for classification. Eschewing the established disorder phenotypes in such work would be desirable, as the phenotypes are noisy and heterogeneous, particularly when there is minimal organization of the phenotypic indicators. Thus, as suggested previously, we might actually come to define pathologies in terms of disturbances in the reward circuitry, or the fear circuitry, or the nonaffective constraint system (see Depue & Lenzenweger, 2005; Epstein, Isenberg, Stern, & Silbersweig, 2002). I am fully aware that this would be a long-term strategic shift, especially given that to this day, DSM contains not a single biological criterion for use as an inclusion or exclusion indicator in the landscape of psychopathology.

The Weightless Environment of DSM Space

The world of DSM remains a "weightless" environment, and this deep-space quality only serves to limit the utility of the methodological approach embodied in it. The diagnostic criteria for the various disorders remain simply listed in a numbered format, and they are not weighted in terms of their diagnostic importance in any compelling manner. By retaining a fundamentally unweighted approach (in terms of either predictive power or what Dana and Dawes, 2004, would call simple "improper" linear modeling) to the organization of diagnostic

criteria, DSM only serves to propagate the definitions of disorders that will maintain or enhance heterogeneity across cases that presumably have the same form of psychopathology. For example, if one only needs any five of nine criteria for the diagnosis of borderline personality disorder (PD), then there are many, many (126, actually—order does not matter) ways for a patient to present as having borderline PD. We must ask: Is this the best way to define borderline PD? What does such heterogeneity do to the communication value of the diagnosis or the unit of analysis for research studies? Is this the best way to define and diagnose psychopathology in general? Does a one-methodological-"size" approach fit all?

Related to the problem of an unweighted system is the manner in which the definitions of disorder contained within DSM may serve to structure relations among symptoms in a manner that limits (even precludes) studies seeking to illuminate a latent organization among the signs and symptoms that could advance classification efforts. For example, consider the diagnosis of schizophrenia. As it stands, even though the diagnostic criteria for the disorder are unweighted in any formal sense, it is essentially the case that a patient needs to display either hallucinations or delusions from the start to receive the diagnosis of schizophrenia. Why does this matter? It matters because the relations observed among the various phenotypic signs and symptoms of schizophrenia when derived from, say, the DSM-IV definition of schizophrenia will be *conditioned* on the presence of these positive symptoms. In terms of multivariate statistical procedures, the covariance matrix for the symptoms under investigation will bear the stamp of this conditioning. Thus all statistical studies of the latent organization of schizophrenia symptoms/signs will be affected by this implicit organization imposed by DSM. If one, therefore, wants to study the latent organization of phenomenology in schizophrenia, then one will need to be comfortable in departing from the narrative rules provided in DSM for the diagnosis of schizophrenia (see Lenzenweger & Dworkin, 1996).

Not unrelated to the issue of the weightless nature of the DSM nomenclature is the problem that the criteria are still largely consistent only with, at best, what could be termed "one-way pathognomonicity"—that is, the notion that the presence of a sign/symptom suggests the presence of an illness. However, it should be stressed that the signs and symptoms listed in DSM often fall short of true one-way pathognomonicity; for example, the presence of "unstable interpersonal relations" does not unequivocally predict the presence of borderline PD. As far as I can tell, there is no symptom or sign in the DSM that has true "two-way pathognomonicity," in that its presence indicates the presence of the disorder and (importantly) its *absence* indicates the *absence* of the disorder. If the diagnostic nomenclature is to move forward, it must begin to resolve these issues related to one-way and two-way pathognomonicity. It is important to note that even if one aspires to a "dimensional" basis for the definition of psychopathology, where levels and cutoff scores will be used to characterize pathology, one still needs to know whether putative cutoff scores or levels are associated with one-way or two-way pathognomonicity. For example, let us consider nonaffective constraint measured dimensionally, and ask ourselves whether or not specific levels on such a dimension suggest the presence or absence of pathology in either a one-way or two-way manner. The issue of one-way *versus* two-way pathognomonicity received considerable attention in Meehl's thinking, and the interested reader is referred to his papers on the topic (see Waller et al., 2006).

The Pitfall of Heterogeneity

Finally, there remains a crucial problem to be dealt with not only in the revision of definitions of disorders in the current diagnostic system, but also in the analysis of data gleaned from nearly any studies of DSM constructs in psychopathology research. This problem, simply stated, concerns *heterogeneity*—heterogeneity among patients owing to the unweighted, polythetic diagnostic system embodied in DSM, but also heterogeneity in performance on laboratory and other measures used in the study of psychopathology. Not all patients present the same symptom profile, owing to the nature of the rules of diagnosis in the DSM system, as well as the reality of polythetically

defined constructs. How does heterogeneity affect the research enterprise?

First, heterogeneity in the composition of patient samples, despite a common diagnostic label, serves to add noise to the unit of analysis. For example, patients with borderline PD drawn from one setting may be quite different from patients with borderline PD drawn from another setting, despite both samples being described as suffering from borderline PD (Korfine & Hooley, 2009). Such variation across patient samples is clearly going to be a source of heterogeneity in performance on measures in laboratory and other studies. However, the heterogeneity issue is not merely a function of variations in symptom profiles across groups of patients sharing the same diagnosis. Even within relatively well-characterized subject samples, where the inclusion criteria have been set up so that all subjects under study are relatively homogeneous, one also sees considerable heterogeneity of performance on laboratory measures. For example, in my own laboratory, where we have defined schizotypy in a consistent manner across all subjects, we find that (1) not all schizotypic individuals are deviant on all laboratory tasks and, moreover, (2) different subjects show different degrees of deviance on different measures (Lenzenweger, 1998). For example, one schizotypic subject may show deviance on eye tracking, working memory, and negative priming tasks, but not on other measures in a protocol; another subject may display thought disorder and sustained attentional deficits, but may not reveal deficits on other measures in the same protocol. The issue of heterogeneity looms large for psychopathology research, and resolution of this issue represents a research challenge that must be embraced if progress is to be made. The interested reader is referred to Maher (2003) and Lenzenweger, Jensen, and Rubin (2003); an older reference, but a wonderful one, with reference to the resolution of heterogeneity is Blashfield (1984).

Let's Consider Meehl's Four Metaquestions

Meehl ended the 1986 chapter with what he termed "four metaquestions," which he suggested represented crucial issues in need of resolution before any large-scale revision of the diagnostic nomenclature was undertaken. Needless to say, these four metaquestions were not resolved prior to the publication of the DSM-III-R, DSM-IV, and DSM-IV-TR. Despite the best-seller status of all these versions of DSM, the nomenclature embodied in them remains in need of improvement along the lines discussed above, as well as in terms of Meehl's metaquestions. Although the committee wheels for DSM-V are turning already, let us consider where we are with respect to the metaquestions from 1986.

Conjectural Etiology

"First, what role should a conjectural etiology, when moderately to strongly corroborated, play in the taxonomic strategy?" (Meehl, 1986, p. 227). At this point, it appears that conjectural etiology still plays essentially no role in the taxonomic enterprise as embodied in the DSM approach. By this I mean that corroborated causal factors have not found their way into the diagnostic system in any genuine manner. Thus the information garnered from years of genetic research in schizophrenia, trauma research in borderline PD, and so on does not play a role in the definition of these disorders or in the diagnostic criteria. The "endophenotype" concept (Gottesman & Gould, 2003), long known in experimental psychopathology (Gottesman & Shields, 1972; Lenzenweger & Loranger, 1989), has begun to be picked up by many researchers and may eventually serve to organize information that can be included in a future diagnostic nomenclature. However, endophenotype information has not been included in past versions of the nomenclature—and, to the best of my knowledge, will not be included as a basis for diagnostic decisions in the forthcoming DSM-V.

We have been through various "decades of this and that" research foci; yet it seems we have miles to go before genetic, neural, neurobehavioral, or other brain-based indices are included in a taxonomic scheme. As a senior observer of the field once said to me, "The brain isn't just a place holder in the skull. It is high time we understand it and take that information into the diagnostic system." Some of the slowness in the incorporation of etiological factors into the

diagnostic scheme may be due to the fact that psychiatry was slow to advance to a research basis, after decades of fascination with psychoanalysis. Moreover, despite initial lightning strikes of luck in the psychopharmacological arena from the early 1950s to the early 1960s (e.g., imipramine, a depression treatment, worked well for panic; chlorpromazine, a medication for operative and anesthetic shock, worked well for psychosis; lithium, a medication for gout, worked well for mania), psychiatry was very slow to advance to the laboratory. As noted by Epstein and colleagues (2002), psychiatry lacked the tools, both conceptual and methodological (especially statistical), for this advance. I would like to make it clear that with respect to the linkage of the brain with the grand traditions of psychological testing and assessment, clinical psychology has been similarly slow to move into the laboratory as well. The subdiscipline of experimental psychopathology (see Lenzenweger & Hooley, 2003) has been a force in moving both psychiatry and clinical psychology into the laboratory. Let us hope that information gathered from the laboratory regarding conjectural etiology and pathology will eventually find its way into future taxonomic systems.

Traits versus Types

"Second, the strategic distinction between thinking in terms of dimensions and categories (types, species, taxa, disease entities) remains with us" (Meehl, 1986, p. 228). Those who are familiar with Meehl's thinking, especially as it relates to the development of coherent cut kinetics (taxometric analysis), will quickly recognize that this was a major interest of his—namely, distinguishing taxa from quantitative dimensions. In short, the answer to the question "Is it quantitatively or qualitatively structured at the latent level?" remains unanswered for the most part, although progress has been made with respect to methods (see Lenzenweger, 2004; Lenzenweger et al., 2007; Waller & Meehl, 1998). For example, it is now generally recognized that schizotypy, or the underlying liability for schizophrenia, is probably structured in a qualitative fashion (see Lenzenweger & Korfine, 1992; Lenzenweger et al., 2007). Whether the qualitative discontinuity reflects a complete discontinuity in schizophrenia liability or a severe step function remains an area of active study (see Lenzenweger et al., 2007). Other areas of inquiry about latent structure are active and generating new findings rapidly, such as latent-structure studies of depression, posttraumatic stress disorder, psychopathy, and so on. Thus, although this second metaquestion is not resolved, methods exist for its resolution and we are making progress. In this context, it is essential to note that this issue, as properly conceived, has addressed itself to the underlying structure of nature and not to the 1980s hassle about how to assess psychopathology, especially PDs. Rather, Meehl's metaquestion concerned which form of psychopathology had a quantitative latent structure versus a taxonic (categorical) latent structure. Meehl (1995) made it clear that this profound issue could not be settled by methods and viewpoints reflective of guild preferences (i.e., psychologists are trained to think dimensionally, and psychiatrists are trained to think categorically). Rather, it would require considerable time and effort; careful theoretical analysis; and dedicated taxonomic research using proper methods, such as taxometric analysis (Waller & Meehl, 1998),[12] latent-class analysis, and finite-mixture modeling (McLachlan & Peel, 2000; see also Lenzenweger et al., 2007; Lenzenweger, Clarkin, Yeomans, Kernberg, & Levy, 2008), among others.

Along these lines it is important to note that psychiatrists (particularly the architects of DSM-V) have begun to discover the utility of a dimensional approach to measurement, as well as the methodological tools needed to explore such an approach (largely factor analysis). Thus, after years of being psychoanalyzed, psychiatry is now likely to go through an extended factor analysis! Indeed, the research agenda for DSM-V has been defined in part as a dimensional enterprise (Helzer et al., 2008). The enthusiasm for factor analysis as a panacea for the classification problems we face, however, must be tempered, and the early experience of psychology with the technique provides a bracing whiff of reality (i.e., despite initial enthusiasm for the power of factor analysis, many in the field now regard the methods as simply data reduction techniques). I would merely urge caution in the adoption of a

wholesale factor-analysis-based, dimensional approach to the measurement and classification of psychopathology. I cannot help having a sense of *déjà vu* when pondering the current fascination with dimensional models for psychopathology (especially the internalizing vs. externalizing scheme that has been with us since the 1960s), bearing in mind the extensive corpus of such prior efforts reviewed by Blashfield (1984). I would recommend a balanced approach as we move forward, incorporating both dimensional and taxometric approaches to the resolution of the dimensions versus types issue in relation to psychopathology. I say this, although I am mindful that the volume *A Research Agenda for DSM-V* (Kupfer, First, & Regier, 2002) does not even include the word "taxometric," "construct validity" appears once, and "endophenotype" appears only three times.

In this context, this issue of traits versus taxa and their relative frequency in psychopathology deserves comment. I have frequently been asked, as one who has done taxometric research and reviewed dozens of taxometric manuscripts over the past 20 years, "How many taxonic entities will be found in psychopathology?" My answer has typically been "Who knows?" My response is not guided by cynicism or the desire to be a wise guy. I simply do not know how many taxa are out there in psychopathology. I should note that I normally want to have a good substantive model in play—one that hypothesizes the existence of a latent taxon in a particular form of psychopathology. Frankly, not that many models or theories argue for the existence of latent, qualitative classes a priori in psychopathology. Is this merely my view? An informal polling of and discussions with well-informed taxometricians suggest that many do not believe that numerous taxa will be discovered.[13] In this context, it is just as important to point out that very few truly (genuine) dimensional entities will be found. Few data sets in the real world conform faithfully to a factor model; thus the notion of a dimensional model of psychopathology is something of a Platonic ideal (just as some think the notion of taxa represents an ideal). The reality is that most data sets in psychopathology, when analyzed carefully, will produce results regarding latent structure that will fall between dimensional and taxonic ideals. Moreover, within the dimensional framework, it is important to realize that even though data can be treated as if they are dimensional and may be factor-analyzable, this provides no assurance that there still might be interesting things going on out in the tails of putative continua that would be of interest to classificationists and taxometricians. Finally, it is important for all who want to push the "factor analysis" (or, more typically, "principal components") button on their statistical software program to realize that factors (continua) will always emerge from factor analysis. But this statistical reality does not mean that nature is truly dimensional at the latent level.

The Reverend Bayes, Base Rates, and Cutoff Scores

"Third, we should get clearer than we presently are about the matter of sliding cuts on various indicators of an entity in relation to base rates and various clinical populations in geographic, social classes, and the like, and the relevance of Bayes's theorem" (Meehl, 1986, p. 229). Upon rereading Meehl's chapter nearly 25 years later, I cannot help saying, upon reflecting on the state of psychiatric diagnosis and classification, "Yes, Paul, you are right; you were right in 1986, and you are right today." It is clearly the case that the underlying mathematical basis for taking base rates and sliding cuts into account in the classification problem is well grounded, is based on a firm statistical foundation, and has a true elegance that should be considered carefully in the context of nosological revision (as well as everyday practice). One of Meehl's concerns, however, was not whether or not taking base rates and the like into account was relevant, but whether it was worth the time and effort. Would it make sense to push Bayes's theorem into the hearts and minds of diagnosticians of all sorts, in research as well as clinical settings (the latter was a true concern of Meehl's)? In short, he wanted the adjustment of the nomenclature and diagnostic practice, based on a consideration of base rates and sliding cuts, to be an effective and validity-enhancing enterprise. In 1986, Meehl felt that considerable work needed to be done on this issue before we went ahead and began to overhaul medical and clinical

science training to reflect such an approach in the diagnostic process. In short, that work is still needed, and this metaquestion is far from resolved (early 1980s discussion of this issue notwithstanding).

The Taxonic Question and Taxometric Problem

> Finally, at the risk of projecting my own current research interests, I would say that a desideratum for the next major revision is agreement upon the general taxometric problem as such, which I see as having two elements: (a) Is a taxometric procedure in psychopathology aimed at anything more than identifying phenotypic clusters; and, if it is, (b) which of the available formal taxometric methods (if any!) have shown themselves capable of detecting an underlying causal structure (whatever its biological or social nature), being meanwhile free of any appreciable tendency to detect taxonic structures that aren't there? (Meehl, 1986, p. 229)

This is an area where activity continues apace. The taxometric and related latent-structure work in schizotypy (Lenzenweger & Korfine, 1992; Lenzenweger et al., 2007), for example, has shown that endophenotypes for schizotypy (i.e., schizophrenia liability) have a qualitative latent structure—transcending the realm of phenotypic indicators reflected in signs and symptoms. Importantly, this latent structure has been found to be associated with important criteria of validity, such as increased rates of familial schizophrenia among the members of an identified schizotypy-positive class (without, importantly, an abundance of other forms of psychopathology in the same family members) (Lenzenweger et al., 2007). As regards the concern expressed in the second part of Meehl's taxometric metaquestion—the identification of methods that do not falsely identify latent classes—this also remains an area of active inquiry. The methodological morals guiding the field of taxometric research have received scrutiny (Lenzenweger, 2004), and alterations of the basic taxometric research strategy claiming to address this issue have been appropriately scrutinized and greeted with caution (Beach, Amir, & Bau, 2005). What must be stressed in this discussion of taxometric analysis is that if it is to serve a useful purpose, it should be conducted in a manner that conforms faithfully to Meehl's description, especially in the ample use of consistency tests. It is remarkable how many published taxometric reports have not conducted consistency tests in a manner consistent with Meehl's writings and intentions. It is worth revisiting Meehl's (1995) recommendations on the necessity of consistency tests: "It is a mistake to think of consistency tests in bootstrap taxometrics as a sort of luxury, pleasantly reassuring if one is so lucky as to get it. Consistency tests are an absolute necessity in bootstrap taxometrics, and the more available the better" (p. 272).[14] Consistent with a central theme of this chapter, it would be particularly useful if future taxometric work in psychopathology focused more on laboratory-assessed, ratio-scale data, particularly within the domain of endophenotypes (i.e., if it moved away from the taxometric analysis of phenotype-level values) (see Lenzenweger, 2004).

Coda

Paul E. Meehl was a thinker who walked many trails in the intellectual high country of the philosophy of science, psychopathology, assessment and prediction, schizophrenia, taxometrics, and (yes) psychoanalysis and beyond (see Waller et al., 2006). The breadth of his thinking, as well as its rigor, continues to be a source of inspiration for many and still has much to offer current and would-be architects of the official diagnostic nomenclature (i.e., DSM-V and beyond). Unfortunately, one still encounters relatively advanced researchers in psychiatry (and, to a lesser extent, clinical psychology) who are fundamentally unfamiliar with many of Meehl's central contributions that have shaped the field of psychopathology and, if understood and applied, will continue to help the field advance (Maher & Gottesman, 2005). This suggests to me that there are still intellectual and professional (guild) walls in psychopathology that need to come down for the field to advance in the nosological enterprise—and, importantly, to fill in the open constructs. The song "Pancho and Lefty by the late Townes Van Zandt includes the lines "He wore his gun outside his pants,/for all the honest world to feel." Paul Meehl wore his scientific values and propos-

als for an improved science of psychopathology very openly; he left no doubt where he stood on most important substantive and methodological issues. In doing so, he left us an excellent map in the 1986 chapter and elsewhere, as well as an important scientific attitude. We need to use these contributions as a basis for continuing to probe the territory of psychopathology—to better organize our classification system, discern the latent structure of psychopathology, and move in the direction of illuminating specific etiology.

Acknowledgments

I thank Irving I. Gottesman, Brendan A. Maher, Lauren Korfine, David A. Silbersweig, Niels G. Waller, and Theodore P. Beauchaine for useful discussions during the preparation of this chapter. I also thank Niels G. Waller for useful comments on an earlier version of the chapter. Preparation of this chapter was supported in part by sabbatical leave funds provided by the State University of New York at Binghamton.

Note

1. Meehl (1986) is also available as a PDF download (see *www.tc.umn.edu/~pemeehl/pubs.htm*).
2. A colleague, who is by any measure a world-class psychopathologist, suggested that all members of the DSM-V revision groups should receive a copy of Meehl's (1986) chapter, should be told to read it, and subsequently should be quizzed on its contents before making any changes in the current nomenclature.
3. Given that some of the constructs in the DSM system are defined in a very narrow fashion, one must also be alert to the possibility that measures associated with these constructs are at risk for being "bloated specific" measures (Cattell, 1966). Such measures often lack theoretical utility, are rarely linked to broader and more established measures of substantive interest, and have only limited predictive power.
4. Both the 1977 and 1978 Meehl papers can be found in the recently published *A Paul Meehl Reader* (Waller et al., 2006). The Meehl (1978) paper can also be downloaded from the Meehl website, which is maintained by Leslie J. Yonce (*www.tc.umn.edu/~pemeehl/pubs.htm*).
5. One need only read the boxed warning medication inserts for many psychiatric medications to come across the ubiquitous statement "The mechanism of action of [drug name], as with other drugs having efficacy in [name of disorder], is unknown." Statements such as these represent the third type of openness referred to by Meehl (1978).
6. A concept that Meehl frequently discussed in connection with open concepts in psychopathology was "specific etiology" (Meehl, 1977). Although this concept was not developed in any great detail in the 1986 chapter, the reader is referred to Meehl (1977) for a rich discussion of this important concept, as well as to those papers in Waller and colleagues (2006) that address specific etiology.
7. The notion of latent constructs and their utility remains a conceptual issue that has been difficult for psychiatry to grasp fully and embrace (it is less so in psychology).
8. I do not wish to suggest that a "signs and symptoms" approach has no value whatsoever. In fact, I think that a considerable amount of work still needs to be done on parsing the psychopathology domain to improve phenotypic-indicator-based classification systems. Rigorous and well-conducted multivariate work still, in my view, has much to offer the "signs and symptoms" approach.
9. A well-known psychiatrist/researcher in schizophrenia noted in a keynote address at a meeting of the American Psychopathological Association a few years ago that the NIMH tends to be about 10 years behind the curve when it comes to supporting innovative thinking. Although I have no way of knowing whether this is a precise estimate (and one can imagine that such a suggestion would surely not sit well in Bethesda), the spirit of the comment, made in the presence of numerous top-notch researchers, is well worth considering.
10. One could easily extend this line of thinking to the issue of quantitative variation and the perennial question of "where to drop the cut" on a dimension of interest. Moving the cutoff score around on a dimension of interest is one straightforward way of studying variation at a boundary or border. This issue could be the focus of an article in its own right and is not developed further here.

11. It is essential to note that there are no "operational definitions" in DSM. That is, the operations by which the disorders are related to the signs and symptoms defining the disorder are not specified; nor are the operations needed to make the diagnosis specified in any strict sense of the term "operational" (see the following text section).
12. Given that many students are likely to read this volume, I think it important to stress that Waller and Meehl (1998) should serve as the foundational reference for taxometric method in addition to the original papers written specifically on the topic. Many "how-to" taxometric books are beginning to appear on the scene, and Waller and Meehl remains the classic and error-free exposition on taxometrics.
13. I have heard some editors and editorial board members at reputable journals say that those who conduct taxometric analyses are "committed to a taxonic model" of psychopathology. This is rather crude thinking, and simply quite far from the truth in my experience. In my observations, particularly in the schizophrenia and PD research domains (where I have my primary research and clinical experience), I believe that the energetic pursuit of dimensional models in psychopathology has far outstripped what appears to be the case for taxonic models. Consider that over 500 citations for the keywords "dimensional and personality disorders" emerge from a PsycInfo search, whereas 19 citations for the keywords "taxonic or taxa and personality disorders" emerge for the same time period.
14. My colleague Niels G. Waller, at Minnesota, tells me that there are at least 35 identifiable consistency tests for use in taxometric analysis. However, the vast majority of taxometric reports do not really follow Meehl's (1995) stern guidance on the use of consistency tests in their taxometric research efforts.

References

Abrams, R., & Taylor, M. A. (1983). The genetics of schizophrenia: A reassessment using modern criteria. *American Journal of Psychiatry*, *140*, 171–175.

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., rev.). Washington, DC: Author.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Beach, S. R. H., Amir, N., & Bau, J. (2005). Can sample specific simulations help detect low base rate taxonicity? *Psychological Assessment*, *17*, 446–461.

Blashfield, R. K. (1984). *Classification of psychopathology: Neo-Kraepelinian and quantitative approaches*. New York: Plenum Press.

Bridgman, P. W. (1927). *The logic of modern physics*. New York: Macmillan.

Cattell, R. B. (1966). Psychological theory and scientific method. In R. B. Cattell (Ed.), *Handbook of multivariate experimental psychology* (pp. 1–18). Chicago: Rand McNally.

Cronbach, L. J., & Meehl, P. E. (1955). Construct validity in psychological tests. *Psychological Bulletin*, *52*, 281–302.

Dana, J., & Dawes, R. M. (2004). The superiority of simple alternatives to regression for social science predictions. *Journal of Educational and Behavioral Statistics*, *29*, 317–331.

Depue, R. A., & Lenzenweger, M. F. (2005). A neurobehavioral model of personality disturbance. In M. F. Lenzenweger & J. F. Clarkin (Eds.), *Major theories of personality disorder* (2nd ed., pp. 391–453). New York: Guilford Press.

Epstein, J., Isenberg, N., Stern, E., & Silbersweig, D. (2002). Toward a neuroanatomical understanding of psychiatric illness: The role of functional imaging. In J. E. Helzer & J. J. Hudziak (Eds.), *Defining psychopathology in the 21st century: DSM-V and beyond* (pp. 57–70). Washington, DC: American Psychiatric Press.

Gottesman, I. I., & Gould, T. D. (2003). The endophenotype concept in psychiatry: Etymology and strategic intentions. *American Journal of Psychiatry*, *160*, 636–645.

Gottesman, I. I., & Shields, J. (1972). *Schizophrenia and genetics: A twin study vantage point*. New York: Academic Press.

Helzer, J. E., Kraemer, H. C., Krueger, R. F., Wittchen, H.-W., Sirovatka, M. S., & Regier, D. A. (Eds.). (2008). *Dimensional approaches in diagnostic classification: Refining the research agenda for DSM-V*. Arlington, VA: American Psychiatric Association.

Hempel, C. G. (1952). *Fundamentals of concept formation in empirical science*. Chicago: University of Chicago Press.

Holzman, P. S., Proctor, L. R., & Hughes, D. W. (1973). Eye-tracking patterns in schizophrenia. *Science*, *181*, 179–181.

Hooley, J. M., & Richters, J. E. (1991). Alternative measures of expressed emotion: A methodological and cautionary note. *Journal of Abnormal Psychology*, *100*, 94–97.

Kernberg, O. F. (1984). *Severe personality disorders*. New Haven, CT: Yale University Press.

Korfine, L., & Hooley, J. M. (2009). Detecting individuals with borderline personality disorder in the community: An ascertainment strategy and comparison with a hospital sample. *Journal of Personality Disorders*, *23*, 62–75.

Kuhn, T. S. (1970). *The structure of scientific revolutions* (2nd ed.). Chicago: University of Chicago Press.

Kupfer, D. J., First, M. B., & Regier, D. A. (Eds.). (2002). *A research agenda for DSM-V*. Washington, DC: American Psychiatric Association.

Lenzenweger, M. F. (1998). Schizotypy and schizotypic psychopathology: Mapping an alternative expression of schizophrenia liability. In M. F. Lenzenweger & R. H. Dworkin (Eds.), *Origins and development of schizophrenia: Advances in experimental psychopathology* (pp. 93–121). Washington, DC: American Psychological Association.

Lenzenweger, M. F. (2004). Consideration of the challenges, complications, and pitfalls of taxometric analysis. *Journal of Abnormal Psychology*, *113*, 10–23.

Lenzenweger, M. F., Clarkin, J. F., Yeomans, F. E., Kernberg, O. F., & Levy, K. N. (2008). Refining the borderline personality disorder phenotype through finite mixture modeling: Implications for classification. *Journal of Personality Disorders*, *22*, 313–331.

Lenzenweger, M. F., & Dworkin, R. H. (1996). The dimensions of schizophrenia phenomenology?: Not one or not two, at least three, perhaps four. *British Journal of Psychiatry*, *168*, 432–440.

Lenzenweger, M. F., & Hooley, J. M. (Eds.). (2003). *Principles of experimental psychopathology: Essays in honor of Brendan A. Maher*. Washington, DC: American Psychological Association.

Lenzenweger, M. F., Jensen, S., & Rubin, D. B. (2003). Finding the "genuine" schizotype: A model and method for resolving heterogeneity in performance on laboratory measures in experimental psychopathology research. *Journal of Abnormal Psychology*, *112*, 457–468.

Lenzenweger, M. F., & Korfine, L. (1992). Confirming the latent structure and base rate of schizotypy: A taxometric analysis. *Journal of Abnormal Psychology*, *101*, 567–571.

Lenzenweger, M. F., & Loranger, A. W. (1989). Detection of familial schizophrenia using a psychometric measure of schizotypy. *Archives of General Psychiatry*, *46*, 902–907.

Lenzenweger, M. F., McLachlan, G., & Rubin, D. B. (2007). Resolving the latent structure of schizophrenia endophenotypes using expectation-maximization-based finite mixture modeling. *Journal of Abnormal Psychology*, *116*, 16–29.

Loevinger, J. (1954). The attenuation paradox in test theory. *Psychological Bulletin*, *51*, 493–504.

MacCorquodale, K., & Meehl, P. E. (1948). On a distinction between hypothetical constructs and intervening variables. *Psychological Review*, *55*, 95–107.

Maher, B. A. (2003). Psychopathology and delusions: Reflections on methods and models. In M. F. Lenzenweger & J. M. Hooley (Eds.), *Principles of experimental psychopathology: Essays in honor of Brendan A. Maher* (pp. 9–28). Washington, DC: American Psychological.

Maher, B. A., & Gottesman, I. I. (2005). Deconstructing, reconstructing, and preserving Paul E. Meehl's legacy of construct validity. *Psychological Assessment*, *17*, 415–422.

McLachlan, G., & Peel, D. (2000). *Finite mixture models*. New York: Wiley.

Meehl, P. E. (1954). *Clinical versus statistical prediction: A theoretical analysis and a review of the evidence*. Minneapolis: University of Minnesota Press.

Meehl, P. E. (1962). Schizotaxia, schizotypy, schizophrenia. *American Psychologist*, *17*, 827–838.

Meehl, P. E. (1977). Specific etiology and other forms of strong influence: Some quantitative meanings. *Journal of Medicine and Philosophy*, *2*, 33–53.

Meehl, P. E. (1978). Theoretical risks and tabular asterisks: Sir Karl, Sir Ronald, and the slow progress of soft psychology. *Journal of Consulting and Clinical Psychology*, *46*, 806–834.

Meehl, P. E. (1986). Diagnostic taxa as open concepts: Metatheoretical and statistical questions about reliability and construct validity in the grand strategy of nosological revision. In T. Millon & G. L. Klerman (Eds.), *Contempo-*

rary directions in psychopathology (pp. 215–231). New York: Guilford Press.

Meehl, P. E. (1995). Bootstraps taxometrics: Solving the classification problem in psychopathology. *American Psychologist*, *50*, 266–275.

National Institutes of Health. (2008). *A broken pipeline?: Flat funding of the NIH puts a generation of science at risk*. Washington, DC: Author.

Pap, A. (1953). Reduction sentences and open concepts. *Methodos*, *5*, 3–30.

Pope, H. G., Jonas, J. M., Cohen, B. M., & Lipinski, J. F. (1982). Failure to find evidence of schizophrenia in first-degree relative of schizophrenic probands. *American Journal of Psychiatry*, *139*, 826–828.

Reichenbach, H. (1938). *Experience and prediction: An analysis of the foundations and the structure of knowledge*. Chicago: University of Chicago Press.

Swales, J. D. (1985). *Platt versus Pickering: An episode in recent medical history*. London: Keynes Press.

Waller, N. G., & Meehl, P. E. (1998). *Multivariate taxometric procedures: Distinguishing types from continua*. Newbury Park, CA: Sage.

Waller, N. G., Yonce, L. J., Grove, W. M., Faust, D. A., & Lenzenweger, M. F. (2006). *A Paul Meehl reader: Essays on the practice of scientific psychology*. Mahwah, NJ: Erlbaum.

CHAPTER 10

Issues of Construct Validity in Psychological Diagnoses

Gregory T. Smith
Jessica Combs

Over the last century, there has been enormous progress in "validation theory," which (as its name indicates) describes how to validate psychological theories and the measures of constructs described in those theories (Kane, 2001; Smith, 2005; Smith & Zapolski, 2009). There has also been remarkable progress in researchers' understanding of psychopathology and in their ability to describe it cogently, reliably, and validly, as reflected in the contents of this volume.

Progress in these two areas has been closely linked. As psychologists learned how to validate measures of unobservable constructs, and theories relating constructs to one another, it became possible to define and test progressively more sophisticated theories of psychopathology. At the same time, advances in understanding psychopathology probably spurred the advances in validation theory: Cronbach and Meehl's (1955) classic introduction of construct validity followed considerable pressure to develop sound means to test theories, rather than just to predict criteria. The same process of reciprocal influence between the two fields is operative today.

We believe that this process has produced recent advances in both psychopathology theory and validation theory that are profoundly important. Specifically, validation theorists have come to recognize that single scores that combine multiple dimensions of functioning necessarily lack clear theoretical meaning (Edwards, 2001; McGrath, 2005; Smith, Fischer, & Fister, 2003; Smith, McCarthy, & Zapolski, 2009). They have concluded that unidimensional traits and unidimensional symptoms are the proper objects of theory and measure validation. Virtually simultaneously, psychopathology researchers have been applying advanced statistical techniques to disaggregate psychological disorders that have long been recognized as heterogeneous (American Psychiatric Association, 1994), with the goal of identifying homogeneous dimensions of dysfunction (see Brinkley, Newman, Widiger, & Lynam, 2004; Jang, Livesley, Taylor, Stein, & Moon, 2004; Lynam & Widiger, 2007; Watson & Wu, 2005). In the bulk of this chapter, we focus on those advances and their implications.

The advances are likely to lead to a fundamentally different, and more valid, means

of describing psychological dysfunction. Perhaps they have been made in time to influence the organizational structure of diagnostic systems, such as the DSM and ICD approaches. To make this argument, we first describe aspects of the last century's worth of advances in psychopathology description and validation theory, which paved the way for the recent progress to which we refer. We then describe this recent progress, doing so in terms of the conclusions drawn by validation theorists and in terms of psychopathology research programs that have fruitfully disaggregated heterogeneous disorders. We touch briefly on a new generation of reaggregation approaches as well, and we consider evidence for the validity, parsimony, and utility of these new approaches.

Once we have made this argument, we turn to a second recent concern of validation theorists: how to conduct sound validation tests in the absence of well-developed, lawful networks of relationships among psychopathology constructs. When such lawful networks are absent, the meaning of construct validation has not always been clear. As a result, validation efforts sometimes appear to have an ad hoc, opportunistic quality, in which any correlation is considered evidence of construct validity (Cronbach, 1988; Kane, 2001). By focusing on how *informative* a validation test is, given the current state of knowledge, researchers can conduct sound, rigorous tests even in the absence of well-developed lawful networks of relationships among constructs. We turn to this issue later in this chapter.

Theoretical Advances over the Last Century: A Very Brief History

Early Efforts

One of the early attempts to codify what was known about psychopathology was provided by the Woodworth Personal Data Sheet (WPDS). The WPDS was developed in 1919 to assist in screening out U.S. Army recruits who might be psychologically vulnerable to the stress of war; it was thought to measure emotional stability (Garrett & Schneck, 1928; Morey, 2003). To construct the test, Woodworth created 116 dichotomous items on both rational grounds (he drew on case histories of identified patients for item content) and empirical grounds (he deleted items endorsed by 50% of more of a normal comparison group) (Garrett & Schneck, 1928).

Although the items appear to have been developed by plausible methods, the scale did not perform very well. The total WPDS score did not differentiate between college freshmen and so-called "avowed psychoneurotics" (Garrett & Schneck, 1928); nor did it correlate with teacher ratings of students' emotional stability (Flemming & Flemming, 1929). To understand this failure, researchers focused on the item content and concluded that a total score appeared to combine multiple, different forms of dysfunction (Garrett & Schneck, 1928; Laird, 1925). Indeed, sample WPDS items included "Have you ever lost your memory for a time?", "Can you sit still without fidgeting?", "Does it make you uneasy to have to cross a wide street or an open square?", and "Does some particular useless thought keep coming into your mind to bother you?" In noting the diversity of mental complaints that were summed to yield an overall score, Garrett and Schneck (1928) concluded:

> It is this fact, among others, which is causing the present-day trend away from the concept of mental disease as an entity. Instead of saying that a patient has this or that disease, the modern psychiatrist prefers to say that the patient exhibits such and such symptoms. (p. 465)

These authors thus emphasized the identification of specific symptoms, rather than aggregations of those symptoms. Even in this early study, Garrett and Schneck recognized the need to avoid combining individuals with different symptom pictures. They appear to have anticipated the current construct validity focus on homogeneous constructs and on the inadequacy of single scores to reflect multidimensional entities. In fact, the subsequent history of psychopathology theory has reflected movement in precisely the direction advocated by Garrett and Schneck. The first step in this direction was to describe dysfunction in terms of syndromes, or constellations of symptoms thought to stem from a common cause (Kraepelin, 1883/1981). This approach has been predominant for most of the 20th century and into the 21st. The dif-

ferentiation of syndromes from one another represented an important advance in specificity, as we describe below.

More recently, researchers have begun to describe psychological dysfunction in a new, different way. They have come to describe dysfunction in terms of basic dimensions; this approach appears to provide even greater precision and clarity than did the traditional syndrome approach. We next consider the syndrome approach, its obvious generativity, and its relationship to advances in validation theory.

Syndromes of Dysfunction

In surveying the broad category of "mentally disturbed" individuals, Kraepelin (1883/1981) famously perceived that certain groups of symptoms tended to occur together. Such groups of symptoms were called "syndromes," and he understood them to have a common biological cause, just as medical diseases often involve a cluster of symptoms stemming from a single cause. The use of the medical perspective to identify separate syndromes of psychological dysfunction has proven enormously generative; it has helped generations of astute clinicians and theoreticians define different classes of dysfunction that probably had different etiologies and different consequences for individuals. Each newly developed version of the DSM and the ICD has reflected the productive differentiation of different forms of dysfunction.

From a theoretical standpoint, with each new differentiation, it becomes possible to investigate ever more precise, better-delineated theories of dysfunction. For example, the recognition that disorders of personality (i.e., chronic dysfunction in characteristic modes of perceiving the world and behaving in it) are distinct from other forms of dysfunction has been enormously important (Millon, 1996). Together with the awareness of the stability of human temperament and personality across the lifespan (Caspi & Roberts, 2001; Roberts & DelVecchio, 2000), it has led to both theories and treatments specific to personality dysfunction (Linehan, 1993; Millon, 1996; Widiger & Boyd, 2009; Widiger, Simonsen, Krueger, Livesley, & Verheul, 2005).

The progressive differentiation among different psychological disorders has also proven highly beneficial to persons suffering from these disorders. Increasingly, psychologists have identified different treatments for different forms of dysfunction. For example, the basic description of the tripartite model of emotional distress (Clark & Watson, 1991) has led to different interventions for those high only in negative affect and for those who are both high in negative affect and low in positive affect. The former pattern is understood to characterize anxiety, and treatments involve exposure to the specific stimuli that elicit the distress. The latter pattern is understood to characterize depression, and an important focus of treatment is to increase positive affect through behavioral activation: Individuals return to active engagement in activities that were previously reinforcing, and the reinforcement and positive mood follow (Barlow, Allen, & Choate, 2004; Dimidjian et al., 2006). A second example is the emergence of dialectical behavior therapy as an effective treatment for borderline personality disorder. Before the development of such a treatment was possible, researchers had to identify the disorder as distinct from other forms of dysfunction.

Syndromes of Dysfunction and Test Validation Procedures

In the first half of the 20th century—perhaps in part due to the failures of rationally based test construction, and perhaps in part due to philosophical objections to the invocation of unobservable entities (Blumberg & Feigl, 1931)—test validity came to be understood as a test's ability to predict a criterion (Kane, 2001). In fact, many validation theorists explicitly rejected the idea that scores on a test mean anything beyond their ability to predict an outcome. As Anastasi (1950) put it, "It is only as a measure of a specifically defined criterion that a test can be objectively validated at all. . . . To claim that a test measures anything over and above its criterion is pure speculation" (p. 67).

In a great deal of psychopathology research at the time, the criterion to be predicted was psychiatric diagnosis, or presence of a syndrome. The criterion validity approach led to the criterion-keying test construction approach, in which one selects items entirely on the basis of whether the items predict the criterion. This method was used in the

construction of two of the most prominent measures of personality and psychopathology, the Minnesota Multiphasic Inventory (MMPI) (Cox, Weed, & Butcher, 2009) and the California Psychological Inventory (CPI) (Megargee, 2009). Typically, items were selected for scales on the basis of whether they differentiated between individuals thought to have a certain syndrome and individuals thought not to have the syndrome.

Substantively, psychopathology scales constructed with criterion-keying methods have afforded important benefits to clients. For example, the MMPI-2 has provided valid clinical assessments and has proven useful for treatment planning (Butcher, 1990; Greene, 2006; Nichols & Crowhurst, 2006; Perry, Miller, & Klump, 2006). The CPI has also validly predicted a wide range of criteria (Gough, 1996). The substantive success of criterion-keyed tests meant that clinical psychologists, and others, could make important diagnostic decisions that affected people's lives with improved validity.

From today's perspective, there are two obvious problems with this approach. The first is that it presumes the validity of the criteria. As Bechtoldt (1951) put it, reliance on criterion-related validity "involves the *acceptance* of a set of operations as an adequate definition of whatever is to be . . . [predicted]" (p. 1245; original emphasis). Thus the criterion validity approach, though useful for identifying predictors of psychopathological syndromes, generally did not involve direct evaluations of the validity of the syndrome descriptions themselves. In short, the criterion validity approach tended to presume, rather than test, the validity of the syndrome characterization of psychological dysfunction.

The second problem with exclusive reliance on the criterion validity approach is this: When tests are developed only to predict a circumscribed criterion, and when they are only validated with respect to that predictive task, the process adds little to basic theory. Criterion-validated tests are not well suited for use in testing theories describing relationships among psychological processes. To test such theories, one often needs tests representing psychological constructs that cannot be captured by a single criterion (Cronbach & Meehl, 1955). Today it seems that an exclusive focus on predicting narrow, syndromal criteria retarded theory development; thus comprehensive theories of personality functioning (Costa & McCrae, 1992), or models identifying core dimensions of psychopathology (Krueger & Markon, 2006) or personality dysfunction (Widiger et al., 2005; Widiger & Trull, 2007), were simply not available as objects of inquiry.

However, this limitation needs to be understood within its historical context. In the first half of the 20th century, the state of knowledge in psychopathology simply did not permit the development of sound measures based on well-developed theory. The predictive failure of the WPDS and other instruments reflects that reality. Perhaps the reliance on the criterion-related validity approach was necessary at the time. In fact, despite its limitations for theory testing, it did in fact lead to a vast growth in knowledge that made possible many of the theoretical advances that have taken place since then. As researchers came to understand which criterion-keyed test items or subscales identified which disorders, it became possible to extrapolate from successful prediction to the development of theories of dysfunction.

Perhaps ironically, the success of the criterion-based validation method led to its ultimate replacement with construct validity theory. Increasingly sophisticated, integrative theories of psychopathology that relied on the operation of unobservable psychological entities could not be validated by using the criterion approach. Thus there was a need for advances in validation theory to make it possible to test theories that included unobservable entities. Construct validity theorists developed an impressive theoretical and methodological perspective to meet this need (Campbell & Fiske, 1959; Cronbach & Meehl, 1955; Loevinger, 1957). We discuss construct validity theory later in this chapter.

Limitations of the Syndrome Approach

The syndrome approach has two fundamental limitations. First, historically, decisions concerning which sets of symptoms go together have not been made on fully empirical grounds; in fact, the statistical and computational tools necessary for readily investigating symptom covariation were developed

after symptoms were initially assigned to syndromes. It has only recently become possible to test empirically whether certain sets of symptoms do in fact covary in ways that justify their combination into a syndrome. As we describe below, there is now good reason to doubt that many syndromes actually reflect a uniquely high degree of covariation among their element symptoms.

Second, many symptoms are common across numerous putative syndromes (Barlow et al., 2004; Brown, Chorpita, & Barlow, 1998; Clark & Watson, 1991; Watson, 2005; Widiger & Costa, 2002; Widiger et al., 2005; Widiger & Trull, 2007; Whiteside & Lynam, 2001). If symptoms are frequently common across syndromes, one has reason to question the value of the syndrome organization. Perhaps, instead, hierarchical models can be organized along dimensions of dysfunction, thus better representing the empirical covariation of symptoms among humans.

The movement from describing psychological dysfunction in terms of syndromes to describing it in terms of homogeneous dimensions of dysfunction is the core advance we discuss in this chapter. To provide a full appreciation for the methodological and substantive basis for this movement, we proceed in two steps. First, we review the basis for the conclusion by validation theorists that the study of unidimensional entities provides the foundation for sound theory testing. We then review a sample of recent programs of psychopathology research that have identified basic dimensions of dysfunction.

Construct Validation and Theory Testing: The Importance of Unidimensionality

Psychopathology theory involves, in part, specifying the nature of relationships among different psychological attributes (causes, correlates, mediators, moderators, etc.). One of the fascinating challenges of psychological science is that the psychological entities we study cannot be directly observed (Cronbach & Meehl, 1955); researchers must infer their existence. Researchers do so in order to best approximate their understanding of real psychological phenomena (Borsboom, Mellenbergh, & van Heerden, 2003). Their doing so has clear utility for helping us understand human behavior, differences among individuals, and dysfunction (Smith, 2005; Smith et al., 2007).

Psychopathology researchers test a theory by developing measures of the inferred entities and testing whether the measures relate to measures of other inferred entities as specified by the theory. Repeated findings consistent with the theory produce increasing confidence in both the theory and the measures used to represent the constructs of interest. Findings inconsistent with the theory raise questions about both the theory and the measures used to test it. The indeterminate nature of this process is clear and has been described many times before (Smith, 2005).

It follows that when we refer to "construct validity investigations" of psychopathology, we are necessarily referring to simultaneous tests of theories and of measures (Cronbach & Meehl, 1955; Smith, 2005). The process of construct validation requires sound definitions of target constructs and a clear statement of anticipated relationships among these constructs. Tests of the validity of construct measures must inevitably be tests of theories specifying relationships among the constructs.

Working from this perspective, validation theorists have drawn an important conclusion concerning test construction, test validation, and theory validation. To the degree that one uses a single score from a target measure that includes multiple dimensions (such as a measure of posttraumatic stress disorder [PTSD] thought to include four factors or a measure of neuroticism thought to have six facets), one's construct validation/theory test has theoretical uncertainty built in. Such a test is likely to have reduced scientific value (Edwards, 2001; Hough & Schneider, 1996; McGrath, 2005; Paunonen, 1998; Paunonen & Ashton, 2001; Schneider, Hough, & Dunnette, 1996; Smith et al., 2003; Smith & McCarthy, 1995; Smith et al., 2009; Smith & Zapolski, 2009).

If one correlates a total score of a multidimensional measure with a criterion, one builds two sources of uncertainty into one's test. The first source of uncertainty is that with a single score, one cannot know the nature of the different dimensions' contribution to that score. Conceivably, an over-

all correlation could reflect the same magnitude of relation between each dimension and the criterion, but it need not. In fact, it is more likely that such a correlation reflects a kind of average of strong and weak relationships between different dimensions and the criterion (Smith et al., 2003; Smith & McCarthy, 1995). Mathematically, one cannot know the meaning of a single score representing a multidimensional measure (Borsboom et al., 2003; McGrath, 2005; Smith et al., 2009).

Psychometricians have been making this point in various ways for the last 10–15 years. Edwards (2001) noted that researchers have long appreciated the need to avoid heterogeneous items; if such an item predicts a criterion, one will not know which aspect of the item accounts for the covariance. The same reasoning extends to tests: If a test includes multiple dimensions, one cannot know which dimensions account for the test's covariance with measures of other constructs. If one uses single scores from multidimensional tests, one has simply moved the heterogeneity problem from the item level to the scale level (Smith et al., 2003). Hough and Schneider (1995), McGrath (2005), Paunonen and Ashton (2001), and Schneider and colleagues (1996), among others, have all noted that using scores of broad measures often obscures predictive relationships. Indeed, studies that have compared prediction using specific facets of broad personality dimensions with prediction using scores on the dimensions themselves show that prediction is improved when one represents each facet individually (Paunonen, 1998; Paunonen & Ashton, 2001). Essentially, one gives oneself the chance to study the separate and incremental roles of each dimension involved in one's measures, rather than averaging across the different dimensions before predicting.

Concerning the second source of uncertainty, it is not just that a composite score averages the functioning of separate constructs in its association with measures of other constructs. The problem is more severe than that. The same composite score will tend to reflect different combinations of construct scores for different individuals in a sample. For example, imagine two individuals with the same overall neuroticism score on the NEO Personality Inventory—Revised (NEO PI-R) measure of the five-factor model (FFM) of personality (Costa & McCrae, 1992). Two of the six facets of neuroticism in that measure are angry hostility and anxiety. One person could be high in angry hostility but low in anxiety, and the other could be low in angry hostility but high in anxiety. This possibility is not just hypothetical; individuals exhibiting psychopathy appear to be high in angry hostility but low in anxiety (Lynam & Widiger, 2007). The same overall neuroticism score could easily be obtained by a person with psychopathy and a highly internalizing individual suffering from anxiety. Indeed, the two traits correlated $r = .47$ in the standardization sample, meaning that they shared only 22% of their variance (Costa & McCrae, 1992). It follows that covariation of an overall neuroticism score with another variable lacks clear meaning. It is not just that when one correlates neuroticism with another variable, one cannot know whether the correlation was "carried" by, in this case, angry hostility or anxiety. It is that the same score could reflect angry hostility elevations for some individuals and anxiety elevations for others.

For these reasons, the central construct validation process should be to test hypothesized relationships among what are thought to be homogeneous, precisely defined constructs. In the present instance, one should study either angry hostility or anxiety, but not a composite that obscures their different roles. Of course, the determination of unidimensionality is itself a validation/theory-testing enterprise. Unidimensionality is established through analyses both internal to a measure, such as factor analysis, and external to a measure, such as tests of convergent and discriminant validity.

Psychopathology research has already proceeded in the pursuit of homogeneous dimensions of dysfunction; we next review programs of research that have sought to identify such dimensions within multidimensional syndromes. As we will show, these efforts indicate that many putative syndromes are in fact characterized by multiple dimensions that are only modestly interrelated, that sometimes appear to have different etiologies, and that are sometimes differentially responsive to a given treatment. When this is true, the use of the syndrome descriptions appears not to be indicated, and may even be counterproductive. We use the current Axis I–Axis II distinction to present this research.

The Disaggregation of Mental Disorders: Axis I

Schizophrenia

Schizophrenia is now well understood to be a "heterogeneous disorder with diverse history, course, and symptoms" (Bell, Lysaker, Beam-Goulet, Milstein, & Lindenmayer, 1994, p. 295). One approach to understanding the nature and implications of the diversity of schizophrenia symptoms has involved the development of the Positive and Negative Syndrome Scale (PANSS; Kay, Fiszbein, & Opler, 1987). Although there is some debate about the precise number of factors on the PANSS, a five-factor structure is commonly found (Bell et al., 1994; Kay et al., 1987; Levine & Rabinowitz, 2007; Nakaya, Suwa, & Ohmori, 1999). A typical description of the five dimensions includes negative symptoms, positive symptoms, disorganization, anxiety/depression, and symptoms of excitement (Levine & Rabinowitz, 2007). The five dimensions are not highly correlated: Levine and Rabinowitz found intercorrelations ranging from –.14 to .41, and the intercorrelations reported by Nakaya and colleagues (1999) ranged from –.09 to .52. Clearly, individuals can have elevations in one dimension without having elevations in other dimensions. An overall schizophrenia symptom count, whether treated as an interval scale variable or as a basis for DSM classification, would combine endorsements of clearly separable constructs.

Attempts to clarify the implications of the heterogeneous processes included in the "schizophrenia" label have included attempts to "deconstruct" schizophrenia by studying presumably unidimensional endophenotypes. The rationale for this approach is that there appear to be multiple, moderately related underlying processes involved in "schizophrenia," and each such process may be influenced by different etiological factors (Braff, Freedman, Schork, & Gottesman, 2007). By studying specific, unidimensional endophenotypes, researchers hope to be able to describe the different neurobiological and genetic architectures of patients with schizophrenia who exhibit different symptom pictures (Braff et al., 2007). It seems increasingly clear that the dimensions of schizophrenia, not the overall diagnosis, are the proper objects of theoretical inquiry.

Often the positive symptoms are understood to be the indicators of psychosis (Buchanan & Carpenter, 1994; Serretti & Olgiati, 2004). However, the experience of psychosis itself has been shown to be multidimensional by numerous authors (see Mizrahi et al., 2006, for a review). Typical dimensions identified include conviction in the psychotic experience, cognitive preoccupation with the psychotic experience, behavioral impact of the experience, emotional involvement with the experience, and external perspective about the experience. A comparative factor analysis indicated that a five-factor structure of a measure of positive symptoms fit far better than did a single-factor solution (Mizrahi et al., 2006). Perhaps most interestingly, antipsychotic medications appear to influence different dimensions of psychosis differently: In one recent study, medication produced a 32% improvement on the behavioral impact dimension, but only a 6% improvement in the degree of conviction about the psychotic experience and no improvement in external perspective about the experience (Mizrahi et al., 2006). It is not the case that antipsychotic medications influence the disorder as a whole; rather, they influence specific dimensions of functioning.

In addition, there is increasing recognition that different psychotic syndromes actually share common dimensions of functioning (Serretti & Olgiati, 2004). These authors, studying a mixed group of patients with psychotic disorders, identified five dimensions of "psychosis" (broadly construed): mania, positive symptoms, disorganization, depression, and negative symptoms. They observed that several of these dimensions are shared across syndromes; perhaps the dimensions, not the constructed syndromes, should be the basis for understanding psychopathology (Serretti & Olgiati, 2004).

Depression

Jang and colleagues (2004) studied the factor structure of depression. Using several symptom lists, they identified 14 subfactors. Examples of these subfactors included feeling blue/lonely, insomnia, positive affect, loss of appetite, and psychomotor retardation. Strikingly, intercorrelations among the factors ranged from .00 to .34, and the factors were differentially heritable, with heritability coefficients ranging from .00

to .35. It therefore seems that (1) some of the dimensions of depression do not covary substantially, and (2) some have a heritable basis and others do not (i.e., their etiologies appear to differ). McGrath (2005) provides interesting examples of the heterogeneity of depression symptom items. If the factors of depression share between 0% and 12% of their variance, then two individuals with the same depression score can easily be experiencing very different symptoms. And if the factors have different etiologies, then the sources of the difficulties the two individuals face may also be very different.

It thus appears to be the case that depression is not a homogeneous psychological construct; it may not even represent a sound hierarchical aggregation of homogeneous constructs. Use of overall depression scores as a variable in psychopathology research is likely to be problematic. For example, testing whether stressful events are a risk factor for depression is imprecise. Are they a risk factor for each construct subsumed within the overall label? Are they a risk factor for only one construct, or for some subset of constructs? For example, do they tend to reduce positive affect, but not influence negative affect? Or do they increase negative affect but not relate to positive affect? Do they influence both? The imprecise test yields imprecise results.

Obsessive–Compulsive Disorder

Many authors have separated obsessive–compulsive disorder (OCD) into several dimensions. Watson and Wu (2005) identified obsessive checking, obsessive cleanliness, and compulsive rituals as separate and only moderately related constructs (sharing between 25% and 31% of their variance), and concluded that OCD may be both phenotypically and genotypically heterogeneous. Mathews, Jang, Hami, and Stein (2004) identified four very similar factors: contamination, repeating/doubts, checking/detail, and worries/just right. They found that the different dimensions had different external correlates. For example, trait anxiety correlated .44 with checking/detail and .15 with worries/just right, whereas depression correlated .30 with worries/just right, but only .04 with checking/detail. Leckman and colleagues (1997) reported similar findings.

The putative syndrome appears to have multiple dimensions, which share only a moderate amount of variance with each other. Clearly, individuals can be high on one dimension without being high on another dimension (e.g., elevation in obsessive checking does not necessitate elevation in hoarding). The dimensions have different external correlates, and they may have different etiologies. It thus appears that OCD scores combine different psychological entities; therefore, the use of overall OCD scores or OCD diagnoses is problematic in psychopathology research.

Posttraumatic Stress Disorder

The symptoms of PTSD have been shown to fall on four factors (intrusions, avoidance, dysphoria, and hyperarousal) by Simms, Watson, and Doebbeling (2002). The four-factor model fit far better than did a model describing a single PTSD dimension, and the four factors had different external correlates. King, Leskin, King, and Weathers (1998) also found that a four-factor model (reexperiencing, effortful avoidance, emotional numbing, and hyperarousal) fit their 17-symptom clinical interview better than did any other model, including a single-factor model or a hierarchical model, in which an overall PTSD factor was thought to underlie the four factors. Most recently, Palmieri, Weathers, Difede, and King (2007) also found four factors with different external correlates. Clearly, identical PTSD symptom counts can refer to very different symptom pictures. There is little evidence for the existence of an overall construct of PTSD. Thus there is good reason to question the use of the PTSD syndrome; use of the four dimensions instead appears to involve a more valid approximation of individual differences in dysfunction within this domain.

The Disaggregation of Mental Disorders: Axis II

Psychopathy

The disaggregation of the many components of psychopathy has received considerable research attention (Brinkley et al., 2004; Cooke & Michie, 2001; Harpur, Hakstian,

& Hare, 1988; Harpur, Hare, & Hakstian, 1989; Lynam & Widiger, 2007). In one classic modern description of psychopathy, Hare's (2003) Psychopathy Checklist—Revised (PCL-R) identified two separate factors—one representing the callous and remorseless use of others, and the other representing a deviant and antisocial lifestyle.

In the PCL-R, the two factors share only 25% of their variance (Hare, 2003; Harpur et al., 1988), and they have numerous different correlates (Harpur et al., 1989). In addition, the factors may have different etiologies. Based on a series of findings, Patrick and colleagues suggested that high scores on the dimension of callous, emotional detachment may reflect a deficit in normal responsivity to aversive stimuli, whereas high scores on the antisocial behavior dimension may reflect a deficit in the higher-order processes governing goal setting and delay of gratification (Patrick, Bradley, & Lang, 1993; Patrick, Cuthbert, & Lang, 1994). If the two dimensions share only a moderate amount of variance, have different external correlates, and have different etiologies, then a single score combining the two obscures important differences in psychological processes. More importantly, these findings question the value of combining the two dimensions and referring to a single syndrome of psychopathy.

Others have disaggregated the syndrome differently. Cooke and Michie (2001) identified three factors, described as (1) arrogant and deceitful interpersonal style, (2) deficient affective experience, and (3) impulsive and irresponsible behavioral style. Furthermore, it appears to be the case that the PCL-R does not include all of the dimensions of the classic description of psychopathy provided by Cleckley (1941); for example, low anxiousness is not represented (Lynam & Widiger, 2007; Rogers, 1995). It may be that the putative syndrome of psychopathy includes several dimensions of dysfunction.

Most recently, Lynam and Widiger (2007) took advantage of the hierarchical FFM of personality to describe the psychopathy construct across all 30 basic personality traits identified in the NEO PI-R five-factor measure. For each trait, they identified whether the trait was related to psychopathy and whether high or low trait scores reflected the psychopathy construct. The result was a placement of psychopathy along each of the 30 homogeneous dimensions of personality; in this view, psychopathy is understood to represent a multidimensional combination of constructs, rather than a coherent theoretical entity in and of itself. Interestingly, Lynam and Widiger's view of psychopathy made extensive use of distinctions between personality facets on the same broad personality domain. For example, persons exhibiting psychopathy were understood to be high on impulsiveness and angry hostility, and low on anxiety and self-consciousness; all four of those traits are placed on the neuroticism domain of the FFM.

Schizotypal Personality Disorder

Fossati and colleagues (2005) compared several different factor structures for the schizotypal personality disorder criteria, and found that a three-factor model (cognitive–perceptual, interpersonal, and disorganization) fit best. Intercorrelations among the three factors ranged from .14 to .63, again indicating substantial unshared variance in each factor. Here, too, individuals were often high on one factor but not on another. Again in this case, the same quantitative symptom count could reflect very different dysfunctional experiences.

Narcissism

Emmons (1987) found four scales in the Narcissistic Personality Inventory (NPI; Raskin & Hall, 1979): leadership/authority, self-absorption/self-admiration, superiority/arrogance, and exploitiveness/entitlement. Intercorrelations among these four factors ranged from .16 to .57. Use of the total NPI score obscures differences among the four factors and can lead to lack of empirical clarity. For example, Emmons (1987) found the correlation between the total NPI score and the Narcissistic Personality Disorder Scale (NPDS) to be nonsignificant and small (r = .12), perhaps suggesting a lack of convergent validity between the two measures. However, this value essentially reflected an average of different correlations between individual NPI subscales and the NPDS: Superiority/arrogance correlated –.04 with the NPDS, but exploitiveness/entitlement correlated .32 with the measure. The four scales also had

different correlations with external criteria. Perhaps, then, narcissism is not a coherent psychological construct, but rather an aggregate of constructs. If so, then theory testing should be conducted separately on the dimensions.

We have sampled from research programs seeking to identify the homogeneous dimensions of putative psychiatric syndromes. Disaggregation research is currently underway with respect to other disorders as well; some research suggests that certain syndromes are valid ways to describe dysfunction, but other research does not. For example, Sanislow and colleagues (2002) identified three dimensions to borderline personality disorder (affective dysregulation, behavioral impulses, and disturbed relatedness), but intercorrelations among the three were all greater than .90, suggesting homogeneity to the disorder. Fossati and colleagues (1999) also found evidence for homogeneity. On the other hand, intercorrelations among the DSM-IV criteria for borderline personality disorder range from .01 to .34 (Sanislow, Grilo, & McGlashan, 2000), and individuals with certain clusters of symptoms are not very likely to have other symptom clusters (Rusch, Guastello, & Mason, 1992). It does seem that individuals diagnosed with borderline personality disorder can have very different symptom pictures, and hence very different psychological experiences.

Implications of the Advances in Validation Theory and of Disaggregation Research

The advances in validation theory and in the disaggregation of putative syndromes offer three important advantages to psychopathology research: theoretical clarity, parsimony, and utility. We consider each in turn.

Theoretical Clarity

Because it is now clear that single scores representing multiple dimensions average the different effects of different psychological processes, and because it is increasingly clear that many putative syndromes combine different dimensions of dysfunction with different etiologies, theoretical clarity requires a focus on homogeneous dimensions of dysfunction. For multidimensional syndromes, the use of symptom counts or overall scores in theory or validation studies cannot be defended. For many disorders, then, the use of diagnostic status or a disorder score as either a predictor or a criterion will tend to produce unclear results. A score on depression, or a depression diagnosis, reflects scores on several different constructs. To test a theory that experience *x* is a risk factor for depression is therefore to be imprecise. It may be the case that experience *x* is a risk factor for one factor within depression, but not for other factors. A proper test of that possibility requires assessment of the separate components of depression and examination of the association between experience *x* and the target factor. If one were to use overall depression scores instead, and if none of the other factors were related to experience *x*, one would risk missing the association altogether.

Perhaps more problematically, in such a situation one has to assume that the symptom count score reflects the same psychological experience for each person, but this does not appear to be true. Individuals may have very similar symptom counts, but very different patterns of scores on individual constructs (McGrath, 2005; Widiger & Trull, 2007). When that is the case, the symptom count does not refer to a coherent theoretical entity, and its correlation with measures of other constructs has unclear meaning. In short, to continue using single scores to represent multidimensional entities is to risk retarding the rate of advance in understanding psychopathology.

Certainly the hetereogeneity of disorders, such as depression, is no surprise to practicing clinicians. Clinicians know full well that they do not fully understand a client's symptom picture simply because they understand the client to be depressed. Clinicians proceed from that diagnosis to try to understand whether the client is experiencing sad affect, anhedonia, loss of appetite, sleep disruption, or other forms of dysfunction. They tailor their interventions to the symptoms that are present.

Parsimony

The approach we advocate does not mean endless fractionation. In fact, the opposite

appears to be true: By focusing on dimensions of dysfunction, psychopathology researchers are likely to find fewer such dimensions of importance than there are constellations of symptoms called syndromes. Perhaps the most well-developed body of relevant research concerns dimensional descriptions in the domain of personality disorders. A recent international conference indicated that researchers appear close to a consensus on describing the domains of dysfunction relevant to personality disorders in terms of four basic dimensions of personality (Widiger & Simonsen, 2005; Widiger et al., 2005). That is, patterns of elevations across four personality dimensions and their underlying facets can be used to describe the dysfunction currently described by the full set of personality disorders. There appears to be an advance in parsimony from targeting basic dimensions of personality dysfunction, rather than trying to delineate multiple syndromes.

In fact, integrative work holds the promise of identifying dimensions of dysfunction shared by personality disorders and Axis I clinical disorders (Krueger, 2002, 2005; Widiger & Simonsen, 2005). Krueger (2005) has suggested that the two groups of disorders share basic dimensions of normal and abnormal personality functioning. This suggestion is supported by the findings of Markon, Krueger, and Watson (2005), which constituted a compelling empirical integration of measures thought to reflect normal and abnormal personality functioning. It appears that the same five factors summarize both domains, and that what is understood to be abnormal personality functioning simply represents extremes of normal dimensions (Clark, 2007; Widiger & Trull, 2007). Again, the demands of parsimony indicate the value of focusing on basic dimensions of dysfunction, rather than multiple putative syndromes that sometimes reflect slightly different combinations of those dimensions.

Serretti and Olgiati's (2004) identification of basic dimensions of psychosis that apply across current diagnostic distinctions suggests parsimony in the dimensional description of psychosis. Recognition of the two broad dimensions of positive affect and negative affect (Clark & Watson, 1991) has shed light on dimensions of dysfunction common to multiple disorders and contributed to the development of a unified model for the treatment of emotional disorders (Barlow et al., 2004); using this approach, one addresses the relevant dimension of functioning rather than specific diagnostic syndromes.

Utility

For several reasons, descriptions of psychopathology in terms of dimensions of dysfunction are likely to be far more clinically useful than descriptions using categories of syndromes have been. First, validity is a necessary condition for utility. Investigations of the causes, correlates, and consequences of unidimensional psychological constructs produce interpretable results: The associations observed refer to a definable dimension of functioning. But investigations of the causes, correlates, and consequences of multidimensional constructs represented by a symptom count or diagnosis produce unclear results: One cannot know which dimensions of a construct influence the associations observed and which do not. Thus the matrix of validity evidence for such constructs rests on weaker grounds and so can provide clinicians with ambiguous or misleading results.

Consider the finding that behavioral activation therapy reduces depression (Dimidjian et al., 2006). This finding is very important, but it may also lack important precision. Suppose it were the case that behavioral activation increases positive affect but has no influence on negative affect. If so, then after behavioral activation treatment, high levels of negative affect may remain to be treated. The conclusion that the treatment alters depression levels does not provide the clinical precision necessary; it would be more useful to clinicians to know that a given treatment influences one symptom but not others.

Verheul (2005), reviewing evidence for clinical utility between syndromal and dimensional models of psychopathology, concluded that "overall, the categorical system has the least evidence for clinical utility, especially with respect to coverage, reliability, subtlety, and clinical decision-making" (p. 295). The syndrome model involves heterogeneity among individuals with a common diagnosis, a lack of precision in description, diagnostic co-occurrence, and arbitrary diagnostic boundaries. Each of these characteristics complicates the diag-

nostic and treatment tasks facing clinicians (Widiger & Lowe, 2008).

There is also good reason to believe that a descriptive system based on homogeneous dimensions would prove more feasible for clinical use than the syndrome-based system has. Widiger and Lowe (2008) have recently offered one practical approach to personality disorder assessment, in which individuals are assessed across 26 facets of personality that are thought to be clinically relevant. They note that assessment along these dimensions takes about half the time that a DSM-IV assessment takes.

The existing diagnostic syndromal categories generally do not lead to identification of treatments specific to disorders (Kupfer, First, & Regier, 2002). In the domain of personality disorders especially, an approach based on dimensions of functioning appears to lead more directly to treatment implications. To earn a DSM-IV personality disorder diagnosis, one must be experiencing "clinically significant distress or impairment in social, occupational, or other important areas of functioning" (American Psychiatric Association, 1994, p. 633). The FFM of personality appears quite useful in identifying dimensions of functioning that are directly related to these concerns (e.g., extraversion–introversion and agreeableness–antagonism pertain specifically to social functioning). Widiger and Lowe (2008) offer specific suggestions tying personality profiles to appropriate interventions.

Empirical demonstrations of improved utility for dimensional descriptions are underway. Samuel and Widiger (2006) showed that descriptions of clinical cases using the 30 NEO PI-R facets of the FFM were significantly more useful to clinicians than were DSM-IV diagnoses. The detailed descriptions of patients along each of 30 unidimensional components of personality were rated as more useful in providing global personality descriptions of clients, in communicating information to clients, in describing clients' important personality difficulties, and in formulating effective treatment interventions. As those authors noted, the detailed information provided by the use of all 30 facets, rather than just the five broad personality scales, probably improved the model's clinical utility significantly. Clinical psychologists can now use numerous detailed and coherent constructs to describe clients' characteristic functioning, and doing so has proven quite useful. Widiger and Trull (2007) address this issue in depth.

Having noted these advantages in regard to utility, we should also recognize the practical difficulties associated with any shift of this magnitude. First (2005) observed that this change would complicate record keeping; cause significant administrative problems; require retraining; disrupt research; and disrupt clinicians' ability to (1) integrate prior research into clinical care and (2) communicate effectively with other mental health practitioners. Of course, as First recognized, none of these issues concern the validity of the science involved. As he noted, if the change provided improved validity and clinical utility, researchers might conclude that it would be worth the disruption. We agree. Surely disruptions of these kinds can be tolerated as by-products of an improved descriptive system.

Dimensions and Taxons

Before we conclude this chapter, we need to clarify our use of terms. We have repeatedly referred to "homogeneous dimensions of functioning"; we chose the term in order to emphasize the concept of "homogeneity." Our intent has been to argue that different constructs should not be combined to produce a single score or to represent what is thought of as a single entity. A different question concerns whether a given psychological entity should be understood to exist along a continuum or as a category/taxon. The arguments we have made here, concerning the need to avoid combining multiple entities, apply whether the entities are taxa or continua. We have used the language of "dimensions," rather than "taxa," for ease of communication. Although we do feel confident that most aspects of psychopathology exist along continua (Widiger & Trull, 2007), this is a separate matter. The core new contribution of validation theory concerns homogeneity and so applies in either case.

To finish our discussion of the implications of current construct validity theory for psychopathology description, we turn to a different issue altogether. We identify a

current difficulty with the implementation of construct validation procedures, and we offer a practical solution to that difficulty.

Informative Validation Tests

In their treatment of construct validity, Cronbach and Meehl (1955) relied on the concept of a "nomological network," which refers to a set of lawful relations among entities. As they described it, the process of construct validation involved specifying the lawful relations between an inferred construct and other constructs, and then testing whether one's measure of the inferred construct produced the results specified in the nomological network. Unfortunately, the idea that one can define constructs by their place in a lawful network of relationships assumes a theoretical precision that is generally not present in psychopathology research specifically, and in the social sciences generally. Psychopathology researchers are often faced with the task of validating their measures and theories despite the absence of a set of expected lawful relations among constructs. When this is true, the meaning of "construct validity," and of what counts as validation evidence, is ambiguous.

Cronbach (1988) addressed this issue by contrasting strong and weak programs of construct validity. Strong programs depend on precise theory that leads to specific predictions. They represent what appears to have been meant by construct validation using a nomological network of relationships. Given the typical lack of precision in psychological science, strong programs should perhaps be understood to represent an ideal to which researchers aspire. Weak programs, on the other hand, stem from less fully articulated theories and construct definitions. With weak validation programs, there is less guidance as to what counts as validity evidence (Kane, 2001). One result can be approaches in which almost any statistically significant correlation between a target measure and another measure, of any magnitude, can be described as validation evidence (Cronbach, 1988). In the absence of a commitment to precise construct definitions and specific theories, validation research can have an ad hoc, opportunistic quality (Kane, 2001), in which sets of correlations with readily available measures are cobbled together as evidence of construct validity. The results of this kind of approach do not tend to provide strong bases for confidence in the validity of measures.

If strong programs of construct validity tend to be aspirational rather than real, and if weak programs tend to provide little information, how should psychopathology researchers approach the validation of their measures? Fortunately, researchers are not stuck between an unattainable ideal and weak, ad hoc theory testing. Rather, there is an iterative process in which tests of partially developed theories provide information that leads to theory refinement and elaboration, which in turn provide a sounder basis for subsequent theory validation research and hence more precise validation tests. Cronbach and Meehl (1955) referred to this "bootstrapping" process and to the inductive quality of construct definition and theory articulation. This process has proven effective; it is evident throughout this book that striking advances in clinical research have provided clear benefits to the consumers of clinical services.

We suggest a standard for evaluating validation research that acknowledges the iterative nature of theories and empirical tests of them. Psychopathology researchers should consider whether their theoretical statements and validation tests are *informative*, given the current state of knowledge (Smith, 2005). The informative nature of these tests depends on their satisfactory answers to the following questions: To what degree does a hypothesis involve direct criticism of a theory, or direct comparison between two alternative theoretical explanations? To what degree does a hypothesis involve a direct response to a criticism of a theory? To what degree does a hypothesis involve a claim that, if supported, would undermine criticism of one's theory? To what degree does a hypothesis involve a claim that, if not supported, would cast real doubt on one's theory? Given the state of development of any one theory, tests of this kind may or may not constitute strong programs of construct validation, but they do not represent weak validation programs. They address questions that will clarify the validity of theories and the measures used to test them. Because of the iterative nature of theory and mea-

sure development and validation, theoretical tests of this kind are likely to provide useful information.

One characteristic of informative theory tests is that they evaluate, as directly as possible, specific claims made for a theory. In psychopathology risk factor research, if one holds that trait *A* is a risk factor for problem *B*, one must generate tests of the claim that, if not supported, would undermine the most important components of the claim. Demonstration of a positive cross-sectional correlation between *A* and *B* does provide information (the absence of a correlation would pose serious problems for a risk theory), but its information value is limited because it is not a direct test of the risk factor hypothesis. In contrast, a longitudinal study in which trait *A* predicts the onset of problem *B*, and other possible explanations for the onset of *B* have been controlled, are more informative. A positive result from such a test is greater reason for confidence in both the theory and the measures of the constructs specified by the theory. Tests of mediation in which the putative cause predicts subsequent changes in the putative mediator, and the mediator then predicts still later changes in the putative consequence (e.g., Fried, Cyders, & Smith, 2008; Stice, 2001), are informative because they are more direct tests of a mediational risk process than are cross-sectional correlations; they have ruled out several possible explanations that compete with the mediational theory. Tests comparing alternative theoretical explanations of the same data (e.g., Bartusch, Lynam, Moffitt, & Silva, 1997) can also be informative because they hold multiple theories up to critical examination, both individually and in comparison to each other. Doing so is an effective way to provide information to researchers and clinicians.

Research that advances understanding of psychopathology is research that involves direct tests of claims made by psychopathology researchers. To relate this concern to the main focus of this chapter, let us consider the challenge of determining whether a diagnosis represents single or multiple dimensions of functioning. To make that determination, it is necessary to compare alternative factor structures of the diagnosis directly. Confirmatory factor-analytic techniques permit direct comparisons of the degree to which different hypothesized factor structures accurately represent the covariances in the data. If a single factor represents the data as well as multifactor solutions do, then parsimony requires adoption of the single factor and rejection of the claim of multidimensionality. If, on the other hand, a multifactor solution best represents the data, the research enterprise is not yet complete. It is then necessary to demonstrate that the different factors relate differently to variables external to the measure; establishing that two factors represent different constructs involves demonstrating that they play different roles in the theoretical structure of psychopathology. An informative demonstration of multidimensionality requires analyses both internal and external to the diagnosis in question.

Summary

In this chapter, we have noted that advances in construct validation theory and advances in psychopathology theory have been closely linked; advances in one field spur advances in the other. One dimension along which advances in both fields have taken place concerns the need to differentiate accurately among different forms of psychopathology. The current system, which relies on syndrome descriptions, has been in place for a century or more. Advances in both fields now indicate that the syndrome approach may compromise the validity of psychopathology descriptions by combining different entities and describing them with a single name and a single score. We have reviewed the validation theory argument against doing so, and we have described research programs that have identified separate dimensions of dysfunction within many currently used syndromal diagnoses. We have argued that a shift to describing psychopathology in terms of dimensions of functioning will increase validity, parsimony, and utility.

We have also briefly considered another advance important for psychopathology researchers. To avoid conducting weak programs of construct validation (Cronbach, 1988; Kane, 2001), researchers should develop studies designed to be informative; that is, researchers should develop construct validation and theory validation tests that

resolve important outstanding questions. Doing so will facilitate subsequent advances in theory and understanding.

The recent history of psychopathology research is one of considerable success. The last century has seen dramatic increases in knowledge, with accompanying increases in clinicians' ability to intervene and ameliorate psychological distress. As researchers apply the recent advances in validation theory discussed in this chapter, their understanding of psychopathology will only continue to increase.

References

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Anastasi, A. (1950). The concept of validity in the interpretation of test scores. *Educational and Psychological Measurement*, *10*, 67–78.

Barlow, D. H., Allen, L. B., & Choate, M. L. (2004). Toward a unified treatment for emotional disorders. *Behavior Therapy*, *35*, 205–230.

Bartusch, D. R. J., Lynam, D. R., Moffitt, T. E., & Silva, P. A. (1997). Is age important?: Testing a general versus a developmental theory of antisocial behavior. *Criminology*, *35*, 13–48.

Bechtoldt, H. P. (1951). Selection. In S. S. Stevens (Ed.), *Handbook of experimental psychology* (pp. 1237–1266). New York: Wiley.

Bell, M. D., Lysaker, P. H., Beam-Goulet, J. L., Milstein, R. M., & Lindenmayer, J. (1994). Five-component model of schizophrenia: Assessing the factorial invariance of the Positive and Negative Syndrome Scale. *Psychiatry Research*, *52*, 295–303.

Blumberg, A. E., & Feigl, H. (1931). Logical positivism. *Journal of Philosophy*, *28*, 281–296.

Borsboom, D., Mellenbergh, G. J., & van Heerden, J. (2003). The theoretical status of latent variables. *Psychological Review*, *110*, 203–219.

Braff, D. L., Freedman, R., Schork, N. J., & Gottesman, I. I. (2007) Deconstructing schizophrenia: An overview of the use of endophenotypes in order to understand a complex disorder. *Schizophrenia Bulletin*, *33*, 21–32.

Brinkley, C. A., Newman, J. P., Widiger, T. A., & Lynam, D. R. (2004). Two approaches to parsing the heterogeneity of psychopathy. *Clinical Psychology: Science and Practice*, *11*, 69–94.

Brown, T. A., Chorpita, B. F., & Barlow, D. H. (1998). Structural relationships among dimensions of the DSM-IV anxiety and mood disorders and dimensions of negative affect, positive affect, and autonomic arousal. *Journal of Abnormal Psychology*, *107*, 179–192.

Buchanan, R. W., & Carpenter, W. T. (1994). Domains of psychopathology: An approach to the reduction of heterogeneity in schizophrenia. *Journal of Nervous and Mental Disease*, *182*, 193–204.

Butcher, J. N. (1990). *Use of the MMPI-2 in treatment planning*. New York: Oxford University Press.

Campbell, D. T., & Fiske, D. W. (1959). Convergent and discriminant validation by the multitrait–multimethod matrix. *Psychological Bulletin*, *56*, 81–105.

Caspi, A., & Roberts, B. W. (2001). Personality development across the life course: The argument for change and continuity. *Psychological Inquiry*, *12*, 49–66.

Clark, L. A. (2007). Assessment and diagnosis of personality disorder: Perennial issues and an emerging reconceptualization. *Annual Review of Psychology*, *58*, 227–257.

Clark, L. A., & Watson, D. (1991). Tripartite model of anxiety and depression: Psychometric evidence and taxonomic implications. *Journal of Abnormal Psychology*, *100*, 316–336.

Cleckley, H. (1941). *The mask of sanity*. St. Louis, MO: Mosby.

Cooke, D. J., & Michie, C. (2001). Refining the construct of psychopathy: Towards a hierarchical model. *Psychological Assessment*, *13*, 171–188.

Costa, P. T., Jr., & McCrae, R. R. (1992). *Revised NEO Personality Inventory (NEO PI-R) and NEO Five-Factor Inventory (NEO-FFI) professional manual*. Odessa, FL: Psychological Assessment Resources.

Cox, A. C., Weed, N., & Butcher, J. (2009). The Minnesota Multiphasic Personality Inventory—MMPI-2. In J. N. Butcher (Ed.), *Oxford handbook of personality assessment* (pp. 250–276). New York: Oxford University Press.

Cronbach, L. J. (1988). Five perspectives on validation argument. In H. Wainer & H. Braun (Eds.), *Test validity* (pp. 3–17). Hillsdale, NJ: Erlbaum.

Cronbach, L. J., & Meehl, P. E. (1955). Construct validity in psychological tests. *Psychological Bulletin*, *52*, 281–302.

Dimidjian, S., Hollon, S. D., Dobson, K. S., Schmaling, K. B., Kohlenberg, R. J., Addis, M. E.,

et al. (2006). Randomized trial of behavioral activation, cognitive therapy and antidepressant medication in the acute treatment of adults with major depression. *Journal of Consulting and Clinical Psychology*, *74*, 658–670.

Edwards, J. R. (2001). Multidimensional constructs in organizational behavior research: An integrative analytical framework. *Organizational Research Methods*, *4*, 144–192.

Emmons, R. A. (1987). Narcissism: Theory and measurement. *Journal of Personality and Social Psychology*, *52*, 11–17.

First, M. B. (2005). Clinical utility: A prerequisite for the adoption of a dimensional approach in DSM. *Journal of Abnormal Psychology*, *114*, 560–564.

Flemming, E. G., & Flemming, C. W. (1929). The validity of the Mathews' revision of the Woodworth Personal Data Questionnaire. *Journal of Abnormal and Social Psychology*, *23*, 500–506.

Fossati, A., Citterio, A., Grazioli, F., Borroni, S., Carretta, I., Maffei, C., et al. (2005). Taxonic structure of schizotypal personality disorder: A multiple-instrument, multisample study based on mixture models. *Psychiatry Research*, *137*, 71–85.

Fossati, A., Maffei, C., Bagnato, M., Donati, D., Caterina, N., & Novella, L. (1999). Latent structure analysis of DSM-IV borderline personality disorder criteria. *Comprehensive Psychiatry*, *40*, 72–79.

Fried, R. E., Cyders, M. A., & Smith, G. T. (2008, June). *Longitudinal validation of the acquired preparedness model*. Paper presented at the annual meeting of the Research Society on Alcoholism, Washington, DC.

Garrett, H. E., & Schneck, M. R. (1928). A study of the discriminate value of the Woodworth Personal Data Sheet. *Journal of General Psychology*, *1*, 459–471.

Gough, H. G. (1996). *CPI manual*. Palo Alto, CA: Consulting Psychologists Press.

Greene, R. L. (2006). Use of the MMPI-2 in outpatient mental health settings. In J. Butcher (Ed.), *MMPI-2: A practitioner's guide* (pp. 253–272). Washington, DC: American Psychological Association.

Hare, R. D. (2003). *PCL-R technical manual*. North Tonawanda, NY: Multi-Health Systems.

Harpur, T. J., Hakstian, A. R., & Hare, R. D. (1988). Factor structure of the Psychopathy Checklist. *Journal of Consulting and Clinical Psychology*, *56*, 741–747.

Harpur, T. J., Hare, R. D., & Hakstian, A. R. (1989). Two-factor conceptualization of psychopathy: Construct validity and assessment implications. *Psychological Assessment*, *1*, 6–17.

Hough, L. M., & Schneider, R. J. (1996). Personality traits, taxonomies, and applications in organizations. In K. R. Murphy (Ed.), *Individuals and behavior in organizations* (pp. 31–88). San Francisco: Jossey-Bass.

Jang, K., Livesley, W., Taylor, S., Stein, M., & Moon, E. (2004). Heritability of individual depressive symptoms. *Journal of Affective Disorders*, *80*, 125–133.

Kane, M. T. (2001). Current concerns in validity theory. *Journal of Educational Measurement*, *38*, 319–342.

Kay, S. R., Fiszbein, A., & Opler, L. A. (1987). The Positive and Negative Syndrome Scale (PANSS) for schizophrenia. *Schizophrenia Bulletin*, *13*, 261–276.

King, D., Leskin, G., King, L., & Weathers, F. (1998). Confirmatory factor analysis of the Clinician-Administered PTSD Scale: Evidence for the dimensionality of posttraumatic stress disorder. *Psychological Assessment*, *10*, 90–96.

Kraepelin, E. (1981). *Clinical psychiatry* (A. R. Diendorf, Trans.). Delmar, NY: Scholar's Facsimiles and Reprints. (Original work published 1883)

Krueger, R. F. (2002). Psychometric perspectives on comorbidity. In J. E. Helzer & J.J. Hudziak (Eds.), *Defining psychopathology in the 21st century: DSM-V and beyond* (pp. 41–54). Washington, DC: American Psychiatric Press.

Krueger, R. F. (2005). Continuity of Axes I and II: Toward a unified model of personality, personality disorders and clinical disorders. *Journal of Personality Disorders, 19*, 233–261.

Krueger, R. F., & Markon, K. (2006). Reinterpreting comorbidity: A model-based approach to understanding and classifying psychopathology. *Annual Review of Clinical Psychology*, *2*, 111–133.

Kupfer, D., First, M., & Regier, D. (Eds.). (2002). *A research agenda for DSM-V*. Washington, DC: American Psychiatric Association.

Laird, D. A. (1925). A mental hygiene and vocational test. *Journal of Educational Psychology*, *16*, 419–422.

Leckman, J. F., Grice, D. E., Boardman, J., Zhang, H., Vitale, A., Bondi, C., et al. (1997). Symptoms of obsessive–compulsive disorder.

American Journal of Psychiatry, *154*, 911–917.

Levine, S. Z., & Rabinowitz, J. (2007). Revisiting the five dimensions of the Positive and Negative Syndrome Scale. *Journal of Clinical Psychopharmacology*, *27*, 431–436.

Linehan, M. M. (1993). *Cognitive-behavioral treatment of borderline personality disorder.* New York: Guilford Press.

Loevinger, J. (1957). Objective tests as instruments of psychological theory. *Psychological Reports*, *Monograph Supplement 3*, 635–694.

Lynam, D. R., & Widiger, T. A. (2007). Using a general model of personality to identify the basic elements of psychopathy. *Journal of Personality Disorders*, *21*, 160–178.

Markon, K. E., Krueger, R. F., & Watson, D. (2005). Delineating the structure of normal and abnormal personality: An integrative hierarchical approach. *Journal of Personality and Social Psychology*, *88*, 139–157.

Mathews, C. A., Jang, K. L., Hami, S., & Stein, M. B. (2004). The structure of obsessionality among young adults. *Depression and Anxiety*, *20*, 77–85.

McGrath, R. E. (2005). Conceptual complexity and construct validity. *Journal of Personality Assessment*, 112–124.

Megargee, E. (2009). The California Psychological Inventory. In J. N. Butcher (Ed.), *Oxford handbook of personality assessment* (pp. 323–335). New York: Oxford University Press.

Millon, T. (with Davis, R. D.). (1996). *Disorders of personality: DSM-IV and beyond* (2nd ed.). New York: Wiley.

Mizrahi, R., Kiang, M., Mamo, D. C., Arenovich, T., Bagby, R. M., Zipursky, R. B., et al. (2006). The selective effect of antipsychotics on the different dimensions of the experience of psychosis in schizophrenia spectrum disorders. *Schizophrenia Research*, *88*, 111–118.

Morey, L. (2003). Measuring personality and psychopathology. In Weiner (Series Ed.) & J. Schinka & W. Velicer (Vol. Eds.), *Handbook of psychology: Vol. 2, Research methods in psychology* (pp. 377–406). New York: Wiley.

Nakaya, M., Suwa, H., & Ohmori, K. (1999). Latent structure underlying schizophrenic symptoms: A five-dimensional model. *Schizophrenia Research*, *39*, 39–50.

Nichols, D. S., & Crowhurst, B. (2006). Use of the MMPI-2 in inpatient mental health settings. In J. N. Butcher (Ed.), *MMPI-2: A practitioner's guide* (pp. 195–252). Washington, DC: American Psychological Association.

Palmieri, P. A., Weathers, F. W., Difede, J., & King, D. W. (2007). Confirmatory factor analysis of the PTSD Checklist and the Clinician-Administered PTSD Scale in disaster workers exposed to the World Trade Center ground zero. *Journal of Abnormal Psychology*, *116*, 329–341.

Patrick, C. J., Bradley, M. M., & Lang, P. J. (1993). Emotion in the criminal psychopath: Startle reflex modulation. *Journal of Abnormal Psychology*, *102*, 82–92.

Patrick, C. J., Cuthbert, B. N., & Lang, P. J. (1994). Emotion in the criminal psychopath: Fear image processing. *Journal of Abnormal Psychology*, *103*, 523–534.

Paunonen, S. V. (1998). Hierarchical organization of personality and prediction of behavior. *Journal of Personality and Social Psychology*, *74*, 538–556.

Paunonen, S. V., & Ashton, M. C. (2001). Big Five factors and facets and the prediction of behavior. *Journal of Personality and Social Psychology*, *81*, 524–539.

Perry, J. N., Miller, K. B., & Klump, K. (2006). Treatment planning with the MMPI-2. In J. Butcher (Ed.), *MMPI-2: A practitioner's guide* (pp. 143–164). Washington, DC: American Psychological Association.

Raskin, R., & Hall, C. S. (1979). A Narcissistic Personality Inventory. *Psychological Reports*, *45*, 590.

Roberts, B. W., & DelVecchio, W. F. (2000). The rank-order consistency of personality traits from childhood to old age: A quantitative review of longitudinal studies. *Psychological Bulletin*, *126*, 3–25.

Rogers, R. (1995). *Diagnostic and structured interviewing.* Odessa, FL: Psychological Assessment Resources.

Rusch, K. M., Guastello, S. J., & Mason, P. T. (1992). Differentiating symptom clusters of borderline personality disorder. *Journal of Clinical Psychology*, *48*, 730–738.

Samuel, D. B., & Widiger, T. A. (2006). Clinicians' judgments of clinical utility: A comparison of the DSM-IV and five factor models. *Journal of Abnormal Psychology*, *115*, 298–308.

Sanislow, C. A., Grilo, C. M., & McGlashan, T. H. (2000). Factor analysis of the DSM-III-R borderline personality disorder criteria in psychiatric inpatients. *American Journal of Psychiatry*, *157*, 1629–1633.

Sanislow, C. A., Grilo, C. M., Morey, L. C., Bender, D. S., Skodol, A. E., Gunderson, J. G., et al. (2002). Confirmatory factor analysis of DSM-IV criteria for borderline personality disorder: Findings from the Collaborative Longitudinal Personality Disorders Study. *American Journal of Psychiatry*, *159*, 284–290.

Schneider, R. J., Hough, L. M., & Dunnette, M. D. (1996). Broadsided by broad traits: How to sink science in five dimensions or less. *Journal of Organizational Behavior*, *17*, 639–655.

Serretti, A., & Olgiati, P. (2004). Dimensions of major psychoses: A confirmatory factor analysis of six competing models. *Psychiatry Research*, *127*, 101–109.

Simms, L., Watson, D., & Doebbeling, B. (2002). Confirmatory factor analyses of posttraumatic stress symptoms in deployed and nondeployed veterans of the Gulf War. *Journal of Abnormal Psychology*, *111*, 637–647.

Smith, G. T. (2005). On construct validity: Issues of method and measurement. *Psychological Assessment*, *17*, 396–408.

Smith, G. T., Fischer, S., Cyders, M. A., Annus, A. M., Spillane, N. S., & McCarthy, D. M. (2007). On the validity and utility of discriminating among impulsivity-like traits. *Assessment*, *14*(2), 155–170.

Smith, G. T., Fischer, S., & Fister, S. M. (2003). Incremental validity principles in test construction. *Psychological Assessment*, *15*(4), 467–477.

Smith, G. T., & McCarthy, D. M. (1995). Methodological considerations in the refinement of clinical assessment instruments. *Psychological Assessment*, *7*, 300–308.

Smith, G. T., McCarthy, D. M., & Zapolski, T. C. B. (2009). *Elemental constructs and descriptive hierarchies: Toward increased validity of measurement and theory.* Manuscript submitted for publication.

Smith, G. T., & Zapolski, T. C. B. (2009). Construct validation of personality measures. In J. N. Butcher (Ed.), *Oxford handbook of personality assessment* (pp. 81–98). New York: Oxford University Press.

Stice, E. (2001). A prospective test of the dual-pathway model of bulimic pathology: Mediating effects of dieting and negative affect. *Journal of Abnormal Psychology*, *110*, 124–135.

Verheul, R. (2005). Clinical utility for dimensional models of personality pathology. *Journal of Personality Disorders*, *19*, 283–302.

Watson, D. (2005). Rethinking the mood and anxiety disorders: A quantitative hierarchical model for DSM-V. *Journal of Abnormal Psychology*, *114*, 522–536.

Watson, D., & Wu, K. (2005). Development and validation of the Schedule of Compulsions, Obsessions, and Pathological Impulses (SCOPI). *Assessment*, *12*, 50–65.

Whiteside, S. P., & Lynam, D. R. (2001). The five factor model and impulsivity: Using a structural model of personality to understand impulsivity. *Personality and Individual Differences*, *30*, 669–689.

Widiger, T. A., & Boyd, S. (2009). Assessing personality disorders. In J. N. Butcher (Ed.), *Oxford handbook of personality assessment* (pp. 336–363). New York: Oxford University Press.

Widiger, T. A., & Costa, P. T. (2002). Five factor model personality disorder research. In P. T. Costa & T. A. Widiger (Eds.), *Personality disorders and the five factor model of personality* (2nd ed., pp. 59–87). Washington, DC: American Psychological Association.

Widiger, T. A., & Lowe, J. R. (2008). A dimensional model of personality disorder: Proposal for DSM-V. *Psychiatric Clinics of North America*, *31*(3), 363–378.

Widiger, T. A., & Simonsen, E. (2005). Alternative dimensional models of personality disorder: Finding a common ground. *Journal of Personality Disorders*, *19*, 110–130.

Widiger, T. A., Simonsen, E., Krueger, R., Livesley, W. J., & Verheul, R. (2005). Personality disorder research agenda for the DSM-V. *Journal of Personality Disorders*, *19*, 317–340.

Widiger, T. A., & Trull, T. J. (2007). Plate tectonics in the classification of personality disorder: Shifting to a dimensional model. *American Psychologist*, *62*, 71–83.

CHAPTER 11

The Meaning of Comorbidity among Common Mental Disorders

NICHOLAS R. EATON
SUSAN C. SOUTH
ROBERT F. KRUEGER

Mental disorders are typically conceptualized as discrete, dichotomous entities, and the current nosological system, the DSM-IV-TR (American Psychiatric Association, 2000), loosely groups them under such rationally derived headings as "mood disorders" and "anxiety disorders" (Watson, 2005). However, high levels of comorbidity between individual disorders—and even across putatively distinct broader groupings—highlight the interrelatedness of many manifestations of psychopathology (Krueger & Markon, 2006). For example, major depressive disorder, panic disorder, and generalized anxiety disorder co-occur more frequently than would be expected by chance alone (Krueger, 1999; Krueger, Caspi, Moffitt, & Silva, 1998; Mineka, Watson, & Clark, 1998). This diagnostic comorbidity raises questions about why certain disorders seem to group together empirically. Do they share at least a portion of their etiologies (e.g., environmental circumstances or genetic predispositions)? Can they therefore be better conceptualized as distinguishable manifestations of more general underlying spectra? If so, what factors determine whether a latent spectrum will primarily manifest itself as one type of disorder (e.g., depression) instead of another (e.g., anxiety) at a specific point in an individual's life?

This chapter reviews research that attempts to answer these questions. We begin by briefly discussing issues relevant to comorbidity and statistical modeling. The early structural literature regarding the two principal factors that underlie common mental disorders—internalizing (INT) and externalizing (EXT)—are reviewed, followed by replications and expansions of these models. Next, we discuss the link between the INT and EXT factors. Several studies that have examined the invariance of the INT–EXT model in diverse populations (e.g., cultural invariance, gender invariance) are described. We also review the literature on the longitudinal stability of INT–EXT. Finally, we address some future directions for research on structural models of comorbidity.

Representations of Comorbidity

There are several ways to think about comorbidity, and it is worthwhile to discuss them briefly because they form the basis of the conceptualization and associated statistical modeling that follow. More in-depth dis-

cussions of comorbidity are presented elsewhere for the interested reader (e.g., Krueger & Markon, 2006; Lilienfeld, Waldman, & Israel, 1994). In general, for our purposes, these representations of comorbidity differ in terms of the type of variables analyzed and corresponding conceptual models. Three types of variables are discussed in terms of comorbidity modeling: categorical variables, continuous variables, and categorical variables that represent dichotomizations of underlying continua. We then explore the difference between co-occurrence and correlation of disorders in terms of comorbidity, which is illustrated with a real-world example.

Comorbidity of Putatively Distinct Categorical Disorders

The first way to conceptualize comorbidity is in terms of co-occurring diagnoses that are putatively distinct. Under the traditional medical model, disorders are typically conceived of as distinct categorical entities, with distinct etiopathophysiologies. For instance, a patient either has a tumor or does not, and that same patient may also either have diabetes or not (Feinstein, 1970). Mental disorder comorbidity is commonly conceptualized in this way, such that a person meets diagnostic criteria for two or more disorders at the same time.

Comorbidity of Continuous Disorders

Although the categorical disease system has prevailed for many years in the psychiatric community, more recent research has demonstrated the benefits of moving toward dimensional models of mental disorders (Helzer et al., 2008; Krueger & Piasecki, 2002; Widiger & Samuel, 2005). Under a dimensional system, comorbidity can be thought of differently than in the categorical disease model, and it is conceptualized in this dimensional way in some of the research to be discussed henceforth. Instead of calculating the proportion of individuals who either have major depression, generalized anxiety disorder, or both, dimensional models of comorbidity typically utilize covariation between *continuous symptom counts* for each disorder. For example, all individuals in a study may be given a structured clinical interview to determine whether or not each diagnostic criterion for major depression and generalized anxiety disorder is present or absent in their lives. The numbers of depression symptoms and of generalized anxiety symptoms assessed to be present are totaled separately for each individual, and a covariance between the two symptom counts is calculated. The size of this covariance—or, alternatively, the standardized covariance (i.e., correlation) between the two disorders—can be thought of as the degree of symptomatic comorbidity these two disorders show (e.g., Krueger, Chentsova-Dutton, Markon, Goldberg, & Ormel, 2003).

Comorbidity of Categorical Variables Modeled Continuously

One final means of understanding and modeling comorbidity germane to our purposes should be addressed. This approach combines the categorical diagnostic and dimensional approaches discussed above. Categorical yes–no diagnoses of disorders can be treated statistically as continuous dimensions by utilizing tetrachoric correlations. Tetrachoric correlations are indices of association that assume a liability threshold model (i.e., at a certain threshold point on the liability continuum of a disorder, the disorder switches from being "absent" to being "present"; see Kendler, 1993). Many recent studies examining the comorbidity between, and underlying structures of, common major mental disorders utilize tetrachoric correlations (e.g., Krueger, 1999; Krueger et al., 1998), and therefore an understanding of the use of tetrachoric correlations is beneficial.

On the most basic level, the key concept underlying the analysis of tetrachoric correlations is that manifest *dichotomous* variables (e.g., a yes–no categorical diagnosis of major depression) can be modeled in such a way that they reflect latent *dimensions*. For example, a researcher might assume that an underlying distribution of disease *X* symptomatology is continuously distributed. Individuals who do not receive a categorical diagnosis of disease *X* would fall below a certain diagnostic threshold on this continuum, and individuals who fall at or above the dimension's diagnostic threshold would receive a categorical diagnosis.

Comorbidity as Co-Occurrence versus Correlation

"Comorbidity" can be defined in a variety of ways. Two definitions of comorbidity with different implications for understanding disorder overlap are "co-occurrence" and "correlation" (Krueger & Markon, 2006). The simultaneous presentation of two (or more) disorders in one individual does not necessarily indicate that the disorders are related. Instead, individuals experiencing one disorder may have the other disorder by chance. This scenario, co-occurrence, simply implies that, due to base rates of each disorder, a particular number of individuals with one disorder will probably experience the other. The second scenario, correlation, is seen when two disorders relate more strongly than chance (i.e., their base rates) would dictate. Thus, when comorbidity is thought of in terms of disorder–disorder correlation, mental disorders are present simultaneously because of some association between them. We focus on this correlational view of comorbidity in this chapter because the evidence supports the existence of correlations among disorders (i.e., disorders do tend to co-occur more frequently than would be expected by chance).

The following example illustrates the difference between comorbidity as co-occurrence and as correlation; it utilizes the basic categorical diagnosis conceptualization of comorbidity discussed above. Under a categorical nosological system, comorbidity between dichotomous (i.e., yes–no) diagnoses of two disorders can be thought of in terms of two-by-two contingency tables, such as those depicted in Table 11.1. The two tables presented represent the difference between co-occurrence and correlation as bases for comorbidity between major depressive disorder and generalized anxiety disorder. Prevalence rates of the two disorders and observed cases of comorbidity in these tables are based on data from 7,108 individuals in the national probability sample of the Midlife Development in the United States (MIDUS) study (see Brim, Ryff, & Kessler, 2004). These rates have been rescaled to a sample of 1,000 individuals for simplicity of illustration and rounded to the nearest whole number.

The upper table represents the overlap of major depression and generalized anxiety disorder if the disorders showed no interrelation, and thus represents comorbidity conceptualized as co-occurrence (and not correlation). The number of individuals in each

TABLE 11.1. Two-by-Two Contingency Tables with Data Representing Frequency of Major Depression and Generalized Anxiety Disorder Diagnoses in Scenarios Where the Disorders Are Independent (Top Panel) and Correlated (Bottom Panel), Based on Rates from a National Probability Sample

Generalized anxiety disorder and major depression as independent disorders

		Generalized anxiety		
		Absent	Present	Marginals
Depression	Absent	844	23	867
	Present	129	**4**	133
	Marginals	973	27	*N* = 1,000

Generalized anxiety disorder and major depression as correlated disorders

		Generalized anxiety		
		Absent	Present	Marginals
Depression	Absent	858	10	867
	Present	115	**17**	133
	Marginals	973	27	*N* = 1,000

Note. Independent-presentation table represents *expected* values calculated from population prevalence rates of major depression and generalized anxiety disorder in 7,108 individuals from the Midlife Development in the United States (MIDUS) study, which utilized a national probability sample. Correlated-presentation table represents *observed* values of MIDUS disorder comorbidity. Values were scaled to *N* = 1,000 for simplicity of presentation.

cell are *expected values*, however, based on the prevalence rates of major depression and generalized anxiety disorder in the sample. For example, we see from the marginal values that 13.3% (i.e., [133/1,000] × 100 = 13.3%) of individuals in the sample experienced major depression, and 2.7% of individuals experienced generalized anxiety disorder. The expected frequencies of individuals in each cell were calculated by using these prevalence rates. We would expect to see approximately 4 individuals out of 1,000 who experienced comorbid major depression and generalized anxiety, on the basis of only these prevalence rates (i.e., .133 × .027 × 1,000 = 3.591, which rounds up to 4 individuals). Remember, this expected value is calculated under the assumption that major depression and generalized anxiety disorder have no association and simply *co-occur* by chance alone.

The lower table represents disorders that are comorbid not only because they co-occur, but also because they *correlate*; they are associated with one another at greater than chance levels. The frequencies in the lower table, unlike those in the upper table, are *observed values*, and thus each cell represents the actual frequencies of individuals seen in the MIDUS study. The marginal rates remain the same in the lower table, but the cell values differ from those in the upper table. Of most importance for our purposes are the cells with values in boldface: the numbers of individuals observed who experienced both major depression and generalized anxiety disorder. As mentioned above, we would expect four individuals to experience comorbidity of these disorders if they were unrelated. However, we see 17 individuals who have both disorders; this is more than 400% of the number of individuals with comorbidity we would expect if the disorders were in fact not associated. This marked increase in observed over expected values suggests that the disorders are correlated to some extent, and thus are seen in tandem more frequently than chance levels would dictate because of a relation between them (e.g., they may both be manifestations of the same latent construct, one disorder may "cause" the disorder, and etc.; see Klein & Riso, 1993; Neale & Kendler, 1995).

Once we have established, as we have in the example above, that disorders are comorbid because of a correlation (and not simply a co-occurrence level due to base rates), we begin to ask why this correlation exists. Numerous factors could account for this sort of comorbidity. It could be the case that one disorder commonly causes another, and this etiological pattern could result in comorbidity. HIV infection is commonly seen in tandem with AIDS-related medical complications, for instance, because the HIV infection leads to suppressed immunity, which allows for the proliferation of the medical complications.

In terms of psychopathology, we are unaware of compelling data for most major mental disorders that indicate a clean, causal etiological pathway from one disorder to the other (Krueger & Markon, 2006). Thus another hypothesis is needed to account for mental disorder comorbidity. A potentially compelling explanation for correlations between disorders is that the disorders are linked by a common latent spectrum. This hypothesis begins to take hold as more and more disorders are shown to interrelate to one another, and thus the presence of a psychologically meaningful underlying factor can be posited. We explore this line of thinking in the next section.

Comorbidity and Common Factors

As discussed above, many mental disorders show observed comorbidity levels that are higher than one would expect by chance alone. A hypothesis to account for the observed comorbidities between many forms of psychopathology is that seemingly distinct mental disorders may be manifestations of common underlying spectra. That is, an unobserved latent factor, or factors, would account for the observed covariations between disorders. This is an application of the common-factor model (Thurstone, 1947), which states that related observed variables are linear functions of one or more common factors and one unique factor per observed variable (these unique factors being typically understood as "psychometric error" or "uniqueness"). This common-factor hypothesis can also be tested statistically; indeed, results of such analyses are the primary focus of this chapter.

The common-factor model is depicted in Figure 11.1. In this hypothetical example, there are three manifest (i.e., observed) variables, depicted as rectangles by convention, and one latent common factor that links the three, depicted as ovular. A unique factor loading (denoted λ) links each manifest variable to the latent variable. Each observed variable also has a unique factor (denoted ε) that accounts for its specific variance, which is the variance in the observed variable not accounted for by the latent factor. Each individual has a score on the latent factor (commonly represented as η; not included in the figure). The observed scores for individuals on any given manifest variable is a linear composite of the factor loading and the unique factor for that variable. For example, the level of major depression observed (which we might denote y_{MD}) is $y_{MD} = \lambda_{MD}\,\eta + \varepsilon_{MD}$. The common-factor model utilizes observed variables, such as a symptom count for major depression, in exploratory or confirmatory factor analyses to clarify the way disorders relate to one another and the presence of any latent factors that could connect them.

If a researcher had collected data on major depression, generalized anxiety disorder, and panic disorder and noticed strong interrelations among the three disorders, he or she might be curious about what was driving this observed covariation. The model shown in Figure 11.1 depicts an answer to this question. If the model shown in this figure represented the true state of nature, it would imply that all individuals have a standing on a latent factor. This latent factor is related to each of the researcher's three variables in different ways (as represented by the three factor loadings); these three variables' relations to the common factor are what account for their observed comorbidity with one another. The researcher could then posit different models (e.g., a model with two latent factors) and, by comparing model fit indices, could determine which model best accounted for the observed covariances between disorders in a parsimonious way. This process, known as "confirmatory factor analysis" (as opposed to the more common "exploratory factor analysis"), has been used in the majority of the research we discuss subsequently. The interested reader is referred to Brown (2006) for a solid introduction to the theory and application of these techniques.

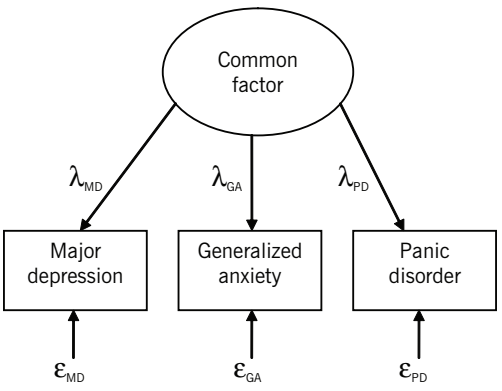


FIGURE 11.1. A theoretical example of the common-factor model.

The Structure of Common Mental Disorders: INT and EXT

Researchers have long considered the structure of mental health problems, especially in light of the levels of comorbidity between certain disorders. Studies of the structure of psychopathology have an especially strong history in the area of child mental health research. Indeed, this child-oriented research posited the notion that two factors could account well for the comorbidity between many common psychopathological syndromes. Achenbach and Edelbrock (1978, 1984) have reviewed this early thought about the structure of childhood psychopathology in detail.

Building upon the foundation of the child psychopathology literature, confirmatory factor analyses of the type described above have recently been applied to questions of psychiatric disorder comorbidity in adults. The results of these studies, by and large, have also indicated that the presence of two broad superordinate factors—INT and EXT—accounts best for the observed covariances between many common major mental disorders (Krueger, 1999; Krueger et al., 1998; see also Achenbach & Edelbrock, 1978, 1984). INT includes such disorders as major depression, generalized anxiety disorder, agoraphobia, panic disorder, and obsessive–compulsive disorder. EXT con-

sists of such disorders as antisocial personality disorder, conduct disorder, and alcohol and drug dependence. It is worth noting that the following analyses have focused on epidemiological data, and thus only common mental disorders are typically included in the statistical models. Disorders with low base rates (e.g., psychotic disorders) are not easily amenable to such analyses.

The General Structure of INT–EXT

The *structures* of INT and EXT are the best-understood aspects of the higher-order factors that account for comorbidity between syndromes. Krueger and colleagues (1998), drawing on previous research (e.g., Achenbach & Edelbrock, 1978), examined the relations between 10 common DSM-III-R (American Psychiatric Association, 1987) psychiatric disorders (i.e., major depressive episodes, dysthymia, generalized anxiety disorder, agoraphobia, social phobia, simple phobia, obsessive–compulsive disorder, conduct disorder, marijuana dependence, and alcohol dependence). Categorical diagnostic data were available for individuals followed longitudinally at age 18 (n = 930; we refer to this as time 1) and age 21 (n = 937; time 2). Due to the categorical nature of the data, the researchers adopted a liability threshold model, and thus analyzed tetrachoric correlations between the disorder diagnoses. Because this study has been cited by subsequent research in this area, was a relatively early contribution to the literature on this topic, and serves as a model for the reader of most studies to be reviewed subsequently, we discuss it here at some length.

Krueger and colleagues (1998) used confirmatory factor analysis to fit three different models to the data at times 1 and 2. Figure 11.2 illustrates several types of the common structural models tested in this and subsequent studies. Because studies frequently differ in the particular disorders (and number of disorders) included in the models that are tested, eight of the disorders examined by Krueger and colleagues were selected to illustrate the models in Figure 11.2. Simple phobia and obsessive–compulsive disorder were not included for the purpose of simplifying these illustrated models. The first model fitted by Krueger and colleagues was defined as all 10 of the disorders included in their study loading on a single common factor (i.e., a "general psychopathology" factor). Model fit indices indicated that this model fit the data reasonably well but left noticeable room for improvement. (Due to space limitations, these indices of model fit are not explored in detail; interested readers are referred to the original Krueger et al. [1998] paper, as well as the treatment of this topic by Brown [2006]. Briefly, however, fit indices gauge the degree to which a researcher-defined model reproduces the observed relations between disorders, and several of these indices favor more parsimonious models as well.) The second model fit was a two-factor model in which major depressive episodes, dysthymia, generalized anxiety disorder, agoraphobia, social phobia, simple phobia, and obsessive–compulsive disorder were indicators of one latent factor (i.e., INT), and conduct disorder, marijuana dependence, and alcohol dependence were indicators of a second latent factor (i.e., EXT). This model fit the data very well according to fit indices, and it also provided a markedly better fit than did the one-factor model; Figure 11.2 includes a simplified representation ("Two-factor INT–EXT model") to illustrate this model's two factor structure.

The time 1 data were fitted to one final model by Krueger and colleagues (1998): a four-factor model (see Figure 11.2's simplified "Four-factor model"). As mentioned above, DSM-IV-TR places disorders into rationally derived subgroups, and this model represented this approach. In this model, major depressive episodes and dysthymia made up one latent factor (i.e., "affective disorders"); generalized anxiety disorder, obsessive–compulsive disorder, social phobia, agoraphobia, and simple phobia made up another latent factor (i.e., "anxiety disorders"); alcohol and marijuana dependence made up a third latent factor (i.e., "substance dependence"); and conduct disorder served as an indicator for the fourth latent factor (i.e., "antisocial behavior"). Although this model fit the data well, it was clearly overparameterized, due to its large, unfavorable change in fit statistics versus the two-factor model. These results taken together highlighted that a two-factor INT-EXT model accounted most parsimoniously for the observed comorbidity between disorders.

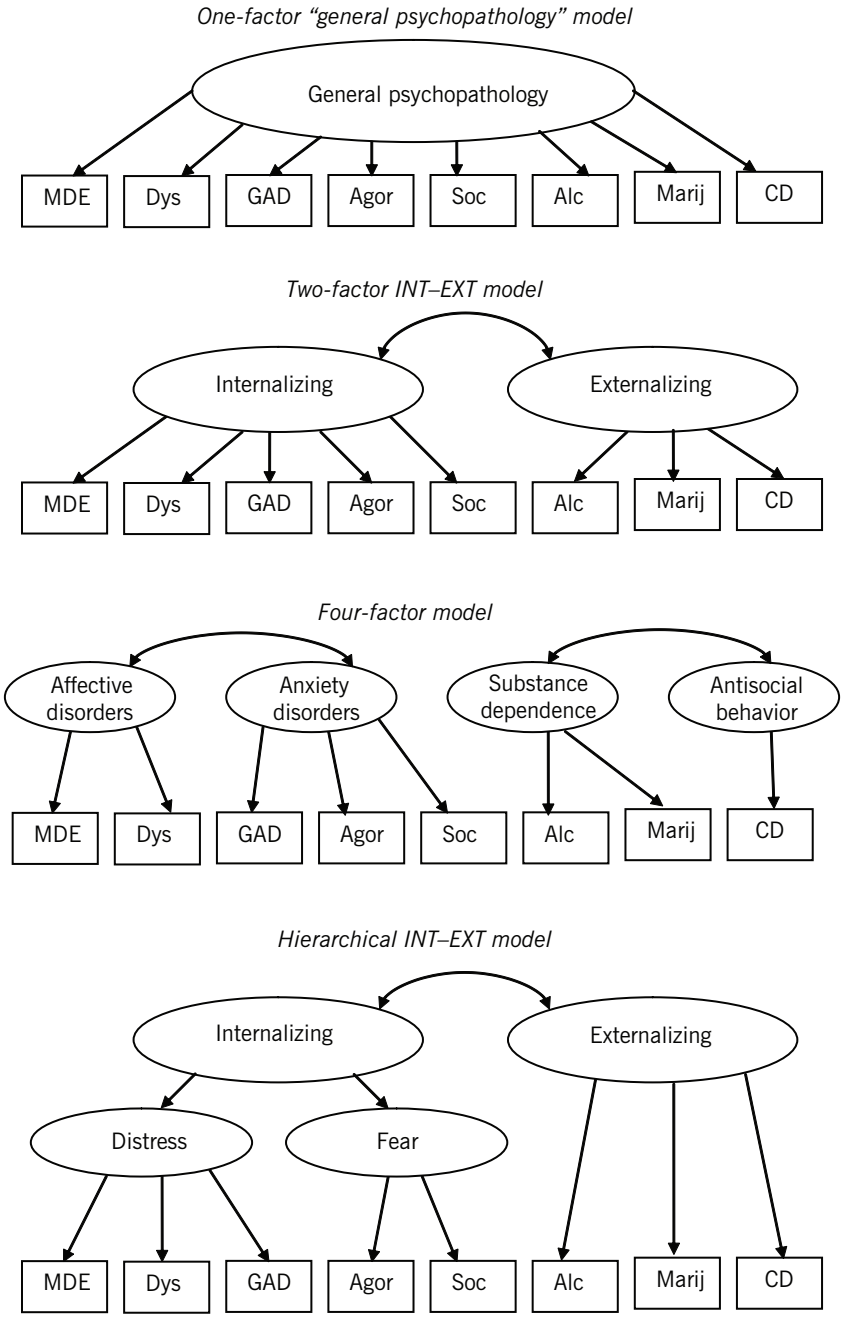


FIGURE 11.2. Simplified representations of commonly modeled structures. Ovals represent latent variables; rectangles represent observed variables; straight arrows represent factor loadings; and curved arrows represent factor correlations. Specific variances have been omitted for simplicity. MDE, major depressive episode; Dys, dysthymia; GAD, generalized anxiety disorder; Agor, agoraphobia; Soc, social phobia; Alc, alcohol dependence; Marij, marijuana dependence; CD, conduct disorder.

The time 2 data of individuals at age 21 years showed a similar pattern. The same two-factor model fit the data well. The one-factor model showed a worsening of fit compared to the two-factor model. The four-factor model again yielded a good fit, but fit indices again indicated that it was overparameterized and thus increased fit at the cost of parsimony. Thus the two-factor model was again preferred at this later time point. It was noteworthy that at both time 1 and time 2, the latent variables in the four-factor model tended to mimic the best-fitting, two-factor model. At time 1, the correlation between the anxiety and affective factors was estimated at 1.00, and the correlation between the antisocial behavior and substance dependence factors was estimated at .89. These correlations were .90 and .72 at time 2, respectively. Thus, even when INT and EXT were split into two separate factors (i.e., when INT was split into anxiety and affective factors, and EXT was split into antisocial behavior and substance dependence factors), those factors tended to correlate very highly together; this indicates the presence of two even higher-level factors to account for these factor–factor correlations (i.e., INT and EXT). This finding can be taken as further support for the hypothesis that the INT and EXT factors account for the observed comorbidity between numerous major mental disorders.

A study published the following year (Krueger, 1999) utilized diagnostic data from 8,098 individuals from the National Comorbidity Survey. Unlike the previous study, in which participants were in late adolescence and early adulthood, these individuals ranged in age from 15 to 45 years. Ten disorders were again modeled, and they were for the most part the same as those from the Krueger and colleagues (1998) study; however, panic disorder replaced obsessive–compulsive disorder, antisocial personality disorder replaced conduct disorder, and drug dependence replaced marijuana dependence. Four models were fitted to these disorders, three of which were the same as those in the earlier study (i.e., a one-factor, a two-factor, and a four-factor model, which divided both the INT and EXT factors in two). Exploratory factor analyses of the data, however, had revealed the presence of two subfactors for the INT factor: (1) an "anxious–misery" factor (referred to as "distress" in more recent literature; e.g., Krueger & Markon, 2006), with indicators of major depressive episodes, dysthymia, and generalized anxiety disorder; and (2) a "fear" factor, with indicators of social phobia, simple phobia, agoraphobia, and panic disorder (see Figure 11.2 for a simplified representation: "Hierarchical INT–EXT model").

The results of model-fitting analyses indicated that the three-factor model best balanced fit and parsimony in the total sample by a wide margin. The sample was then divided randomly in half, and the three-factor model fit best in both halves. Krueger (1999) took these results (and other subsample results to be discussed below) collectively to indicate the superiority of the three-factor model in general—especially due to the fact that this model provided the best fit in five groups, including the large total sample. Overall, this study provided strong evidence for the three-factor model, wherein EXT is unitary and INT is bifurcated into distress and fear factors. We refer to this model hereafter as the "hierarchical INT–EXT model."

Replications of the INT–EXT Model

The two studies described above converged on a two-factor model of psychopathology: The observed covariances between disorders could be conceptualized adequately, but also parsimoniously, by means of the INT and EXT latent factors. Although these studies had utilized different data drawn from different populations, further replication of this finding was warranted. Additional studies on this topic were also necessary to clarify whether INT was best conceptualized as a unitary factor or as having a hierarchical structure, with a higher-order factor consisting of two subfactors (i.e., distress and fear).

Several research groups have replicated and extended the INT–EXT model in the past decade. These replications further support the hypothesis that the INT and EXT factors link particular disorders. Vollebergh and colleagues (2001) modeled the structure of nine mental disorders in a community sample from the Netherlands. Diagnoses of DSM-III-R disorders (occurring in the previ-

ous 12 months, and thus not lifetime diagnoses) were analyzed via tetrachoric correlations and fitted to four models. The first was a one-factor, "general psychopathology" model, in which comorbidity between all nine disorders was accounted for by a single latent dimension. The second model reflected a two-factor structure of INT and EXT. A third model tested the structure outlined by Krueger (1999)—to wit, the hierarchical INT–EXT model. The fourth model placed conceptually similar disorders together. The authors of the study tested the fit of this fourth model by placing disorders into three DSM-based groups: mood disorders, anxiety disorders, and substance use disorders. Due to the longitudinal nature of their data (discussed below), these models were fitted to several subsets of the data.

The results of this study served as strong replication for previous INT–EXT research and the hierarchical INT–EXT model. The one-factor model tended to fit worst across all the analyses. Also providing a relatively poor fit was the three-factor, DSM-based model (i.e., mood disorders, anxiety disorders, and substance use factors). The two-factor INT–EXT model fit better. This INT–EXT model had a significantly better fit than the one-factor model, and did not show a worse fit than the more parameterized DSM-based three-factor model. However, the superior model was the three-factor hierarchical INT–EXT model. Across analyses, this model had the most favorable results on several fit statistics.

Other studies have also replicated the INT–EXT structure. Slade and Watson (2006) utilized 12-month DSM-IV and ICD-10 (World Health Organization, 1992) diagnoses collected in an Australian community sample. The four models they fitted were the same as those utilized by Vollebergh and colleagues (2001) discussed above, and the results were largely the same. For DSM-IV and ICD-10 disorders, the one-factor model provided the worst fit according to a variety of fit indices. The DSM-based three-factor model fit somewhat better, but generally not as well as the two-factor INT–EXT model. Again, in both diagnostic systems, the hierarchical INT–EXT model showed the most favorable fit index statistics.

The individual results of these studies are compelling, but a compilation could prove even more helpful. Indeed, Krueger and Markon (2006) undertook a meta-analysis of these findings. The authors utilized tetrachoric correlation matrices reported in five major studies of psychopathological comorbidity (Kendler, Prescott, Myers, & Neale, 2003; Kessler, Chiu, Demler, & Walters, 2005; Krueger, 1999; Krueger et al., 1998; Vollebergh et al., 2001), which represented a total of 23,557 participants. Several models were fitted to these data, and these analyses produced results largely congruent with those of previous studies: The one-factor "general psychopathology" model had a poor fit; a two-factor INT–EXT model fit better; and the best fit overall was provided by the hierarchical INT–EXT model. This study, with its very large sample size and aggregation of different data sets, provides the strongest evidence yet for the latent INT–EXT structure. In addition, these results support the bifurcation of the INT factor into two subfactors: distress and fear.

Several conclusions can be drawn from the convergence of the studies discussed up to this point. First, the overall latent structure of INT–EXT has been replicated by a number of independent research teams. When compared with other models, such as a one-factor "general psychopathology" model and a three-factor DSM-based model, INT–EXT has been found to account best for the observed covariances between many major mental disorders. Second, the structure of the INT factor seems to be hierarchical. The majority of studies have found that the hierarchical INT–EXT model fits the data best, which indicates that INT is best conceptualized as a higher-order INT factor with distress and fear subfactors.

The Expansion of the INT–EXT Model

The results of structural modeling depend to a large degree on the disorders included for analysis. The earlier work on the INT–EXT dimensions (e.g., Krueger, 1999; Krueger et al., 1998) found that a particular set of disorders could be accounted for by INT and EXT. The DSM-III-R disorders that related to INT were major depressive episodes, dysthymia, generalized anxiety disorder, social phobia, simple phobia, agoraphobia, panic

disorder, and obsessive–compulsive disorder. The disorders that related to EXT were alcohol dependence, drug (e.g., marijuana) dependence, antisocial personality disorder, and conduct disorder.

Subsequent research has expanded the disorders (DSM-IV and ICD-10) and syndromes associated with the INT factor. Krueger and colleagues (2003) parsed anxiety into "anxious worry" and "anxious arousal," both of which are associated with INT. They also found that neurasthenia, somatization, and hypochondriasis loaded on the INT factor. Slade and Watson (2006) demonstrated that posttraumatic stress disorder was related to the INT distress subfactor, and they replicated the inclusion of neurasthenia (also on the distress subfactor). Recent work has also indicated that the syndrome of bulimia/binge-eating disorder loads on the INT factor (Kramer, Krueger, & Hicks, 2008). Bulimia/binge-eating disorder may not be what some readers would intuitively identify as an internalizing syndrome. This study demonstrates that bulimia/binge-eating disorder has an INT factor loading similar in magnitude to that for panic disorder, which is about half of the loading of more traditionally conceived internalizing disorders (e.g., generalized anxiety disorder and social phobia); however, the loading of bulimia/binge-eating disorder is notably larger than that of hypochondriasis, and more than three times as large as the loading for obsessive–compulsive disorder in this study. Finally, in children and adolescents, it appears that separation anxiety disorder is related to INT (Lahey et al., 2008).

The disorders and syndromes included in EXT have remained largely similar for the past decade, and fewer have been identified for EXT than for INT. This is due in part to a greater focus of diagnostic systems on more internalizing-related disorders, as well as inclusion of fewer externalizing disorders in the data sets utilized for the statistical modeling. Adult antisocial behavior has been linked to EXT (Kramer, Krueger, & Hicks, 2008). In children and adolescents, inattention, hyperactivity–impulsivity, and oppositional defiant disorder seem related to EXT (Lahey et al., 2008). A study of categorical versus continuous liability models of externalizing disorders elaborated on the previously identified EXT substance-related disorders. This study found an externalizing-based interrelation between various forms of substance dependence: nicotine, alcohol, marijuana, cocaine, and other substances (Markon & Krueger, 2005). Finally, some externalizing-specific behaviors have been linked to EXT, such as relational, destructive, and physical aggression; boredom proneness; low empathy; alcohol, marijuana, and drug use; blame externalization; feelings of alienation; problematic impulsivity; low planful control; impatient urgency; theft; fraud; low honesty, irresponsibility; low dependability; rebelliousness; and excitement seeking (Krueger, Markon, Patrick, Benning, & Kramer, 2007). These studies taken together indicate that EXT, like INT, is a broad factor that underlies many temperamental and psychopathological constructs.

The Association between INT and EXT

As we have seen, numerous studies have uncovered and replicated the INT–EXT structure. This supports the notion that INT and EXT account for the comorbidity between many commonly diagnosed major mental disorders. However, up to this point, we have only discussed the covariances between manifest variables (e.g., between measured major depression symptom counts and generalized anxiety disorder symptom counts) and have largely neglected possible covariances between INT and EXT. It is not necessary that these factors be orthogonal (uncorrelated); they can be modeled obliquely (allowed to correlate). As mentioned above in passing, the factors in the four-factor model by Krueger and colleagues (1998) correlated highly. There is more to discuss on the topic of factor correlation: the relation between INT and EXT themselves. Many studies that have replicated the INT–EXT model have reported an association between INT and EXT. Krueger and colleagues reported correlations (standardized covariances) between INT and EXT of .454 and .417 when their participants were 18 and 21 years old, respectively. Other studies have indicated similar degrees of relationship between INT and EXT. For example, Krueger (1999) found an INT–EXT correlation of .51

in a different sample, which closely mirrored the correlation of .50 found via a large meta-analysis (Krueger & Markon, 2006). Correlations between INT and EXT estimated from two assessment waves in the Netherlands taken 2 years apart (.56 and .66) are consistent with the previously reported estimates as well (Vollebergh et al., 2001).

Although the first studies of INT–EXT tended to use DSM-III-R diagnostic criteria, converging INT–EXT relationships have been found in research using other classification systems as well. Slade and Watson (2006) found INT–EXT correlations of .65 when the factors were assessed via DSM-IV diagnostic criteria. The correlation between INT and EXT was almost identical (.61) when the ICD-10 criteria were utilized for assessment. Thus, even across classification systems, the INT–EXT correlation holds at relatively the same level of association.

To summarize, across studies, samples, diagnostic systems, and nations, correlations between INT and EXT have converged on a relatively small range of values. This moderate correlation has been reported to range from about .42 to about .66, with .50 seeming to be a reasonable compromise. This result indicates that approximately 25% of the variance (i.e., $.50^2 = .25$) in INT is accounted for by variance in EXT, and vice versa. It should be noted, however, that the common statistical parlance of "accounted for" should not be interpreted to mean "accounted for *causally*," but instead is a comment on the level of covariation between INT and EXT. In other words, this shared variance does not necessarily demonstrate that one factor causes the other.

This moderate relationship has several implications for research, theory, and practice regarding the INT and EXT latent factors of psychopathology. Most clearly, this correlation indicates that individuals with mental disorders do not always (or necessarily even usually) fall into an internalizing *or* an externalizing group. Indeed, the INT–EXT relation suggests that individuals who have internalizing psychopathology are likely to have some externalizing psychopathology; alternatively, individuals with externalizing behaviors or disorders are likely to have some internalizing ones as well. It is again important to note that this is not necessarily a causal relationship. For clarification of what this correlation might mean, consider the following example. Externalizing individuals might commit crimes and frequently use alcohol and drugs. This lifestyle, with its associated impulsivity, health problems, and mistreatment of others, could easily leave these individuals feeling alienated. These feelings of alienation and abandonment by family and friends could lead to feelings of depression. In addition, these individuals might have some anxiety arising out of fear of capture and incarceration. Thus, in this case, the internalizing symptoms (mood and anxiety) would "arise from" externalizing behaviors. This is only one interpretation, but it implies a longitudinal sequence of events (EXT problems antedating INT problems) that could be tested to help clarify the nature of the INT–EXT association.

Personality and Temperament as the Link between INT and EXT

One compelling hypothesis to account for the INT–EXT relationship incorporates a role for individuals' personality and temperament. If particular traits or predispositions are associated with both INT and EXT, these personality and temperamental constructs could serve as the bridge that links the two factors correlationally.

The notion that personality and temperament are related to psychopathology has a long history in clinical thought and nosology. Previous editions of the DSM, as well as the most recent edition, have included personality disorders in one form or another. Although personality disorders are often considered to be distinct from other forms of psychopathology (hence their placement on a separate axis from other psychopathology in DSM-IV), there is little compelling evidence to support this division. Indeed, reviews of the literature indicate that personality disorders are typically more similar to other mental disorders than they are different (Krueger, 2005). Normal (nonpathological) personality traits have also been linked to mental disorders (e.g., Trull & Durrett, 2005). Thus the hypothesis that personality and temperament may show associations with the two factors of psychopathology, and in fact may link them, is not without a priori empirical support.

Several studies have tested the role of personality and temperament in internalizing and externalizing disorders. This research has noted that both INT and EXT are associated with negative emotionality (or neuroticism), and that EXT is further linked with disinhibition (e.g., low conscientiousness, low control, high novelty seeking, high disagreeableness; Clark, 2005; Krueger & Markon, 2006). These findings suggest that negative emotionality may account for a large part of the relation between INT and EXT and may predispose one to internalizing and externalizing general psychopathology. The presence or absence of disinhibition, on the other hand, may play a role in determining whether this underlying propensity for psychopathology is manifested as externalizing (i.e., disinhibition present) or internalizing (i.e., disinhibition absent). In addition, several personality traits account for some lower-level disorder–disorder comorbidity. In one study, neuroticism appeared to account, to a strong degree, for the interrelations between the internalizing disorders; neuroticism and novelty-seeking trait levels accounted for a good deal of the observed comorbidity between the externalizing disorders (Khan, Jacobson, Gardner, Prescott, & Kendler, 2005).

The Genetic and Environmental Bases of INT–EXT

A number of studies have indicated that the hierarchical INT–EXT model accounts best for observed comorbidity between a variety of psychological disorders, and these factors seem to be related to some personality- and temperament-related predispositions. However, until this point, we have not discussed any studies that addressed the etiology of the INT and EXT factors. Indeed, the previous studies were observational in nature, and did not evaluate the risk factors that account for the observed comorbidity (Kendler et al., 2003). A determination of the origin of these disorders' comorbidity and higher-order factors could have implications for both conceptualization and treatment.

Etiological questions of this sort can be tested empirically by utilizing appropriate models and particular samples (e.g., twins). For readers unfamiliar with behavior genetic methodology, a brief description may prove helpful. Behavior genetic models, typically conducted with samples of identical (monozygotic) and fraternal (dizygotic) twins, parse variance in the observed variables into genetic, shared environmental, and nonshared environmental factors. Shared environment encompasses nongenetic factors that are shared by twins as they grow up, such as familial socioeconomic status, which serve to make the two twins more similar to one another. Nonshared environment is composed of nongenetic factors that differ between twins, such as one twin playing baseball while the other participates in a school orchestra, that serve to make the two twins less similar to one another. Error is also included in nonshared environment. Finally, it is important to note that the variance accounted for by genes, shared environment, and nonshared environment is estimable in these studies because monozygotic and dizygotic twin pairs differ in the proportion of genes shared between the twins (100% and 50%, respectively); monozygotic and dizygotic twins who were reared together do not differ in the amount of shared and nonshared environment between twins in a twin pair (all twins who were reared together, regardless of zygosity, share 100% of the shared environment and 0% of the nonshared environment, by definition).

Kendler and colleagues (2003) investigated the role of genetic, shared environmental, and nonshared environmental factors in the risk for developing many common disorders in a large sample (N = 5,600) of same-sex twin pairs. The authors modeled two different combinations of 10 disorders (major depression, generalized anxiety disorder, animal phobia, situational phobia, panic disorder, alcohol dependence, drug abuse/dependence, adult antisocial behavior, and conduct disorder) to address the origins of disorders within both INT and EXT, as well as between the distress and fear subfactors of INT. Their analyses utilized an independent-pathway model (a full description of which is beyond the scope of this chapter; interested readers are referred to the original paper). On a basic level, however, this model allowed for the estimation of the effects of genes, shared environment, and nonshared environment both (1) as higher-order, common factors that conferred risk for all relat-

ed disorders; and (2) as unique effects that conferred risk for each disorder separately. This could be illustrated in Figure 11.1, if the common factor were, for instance, "genetic effect," and each of the unique variances of the observed variables were disorder-specific genetic effects.

The results of this study indicated a strong genetic effect common to INT and EXT. In addition, genes also showed notable specific effects on alcohol dependence and drug abuse/dependence. Shared environment had effects for adult antisocial behavior and conduct disorder. Nonshared environment tended to show strong unique effects for each disorder.

These findings indicate that a high degree of genetic risk is associated with the origins of the INT and EXT factors. However, there is less of a genetic impact on the development of individual disorders. Genes are thus conferring a common liability for comorbidity among the internalizing disorders as well as the externalizing disorders. Kendler and colleagues (2003) concluded that the pattern of lifetime disorder comorbidity commonly observed in many major mental disorders occurs primarily through genetic risk factors, especially at the level of the INT and EXT factors.

Invariance of INT–EXT

In addition to structural replications, replications and investigations of the INT–EXT model in diverse samples are important. One issue that requires particular consideration in INT–EXT research is that of invariance. In the context of the present review, and on a basic level, invariance can be thought of as replication of the INT–EXT structure in persons (1) who come from different populations or (2) who vary from others on important individual difference variables. This is known as "configural invariance," and it is established by replications of the same INT–EXT structure across samples and studies. More stringent models of invariance can be tested as well, such as whether the loadings of the observed disorders on the latent factor are significantly different across groups. These more formal types of invariance require joint (simultaneous) modeling of two or more samples (e.g., younger adults and older adults), and testing of whether or not the model parameters for each group differ significantly from one another. For instance, major depression might have a very high factor loading on INT in younger adults (indicating that an individual's level of latent internalizing, or factor score, was highly predictive of his or her manifest level of major depression), but a lower loading in older adults (indicating that an individual's level of major depression was not strongly related to his or her overall level of latent internalizing). Space limitations do not permit an in-depth discussion of the levels of factorial invariance, and the interested reader is referred to other accounts for further reading (e.g., Kramer et al., 2008; Meredith, 1993).

Although the role of individual differences in the structure and invariance of INT–EXT is conceptually important, only now is it being examined. Previous research has addressed a few of the relevant possibilities for invariance, which we discuss below. At this time, attention has been paid primarily to cross-cultural and gender-based invariance. Some preliminary research touches on possible invariance in treatment-seeking populations and age invariance issues as well. In addition, most studies to date have not tested invariance formally; instead, structural invariance (i.e., the presence of a two-factor or hierarchical INT–EXT structure) has been investigated by comparing the results of separate studies conducted in different populations.

Cross-Cultural Invariance of INT–EXT

It is necessary to determine whether the INT–EXT model represents psychopathology adequately across cultures. If it were the case that other models superiorly accounted for the comorbidity of mental disorders in non-Western cultures, for instance, this would draw into question both the validity and the overall utility of the INT–EXT model outside the United States. Several studies have investigated the structure of common mental disorders in international samples, which allows us to address the concern of INT–EXT universality. These studies indicate that the INT–EXT structure does not appear to be a solely American, or even Western, phenomenon; it has been found in psychiatric data from a variety of cultures. We review

those studies briefly below to illustrate the breadth of the cross-cultural data, which have converged on the notion that the INT–EXT model of comorbidity shows configural invariance.

As discussed above, while Krueger (1999) utilized a U.S. sample, which resulted in a hierarchical INT–EXT model, Krueger and colleagues (1998) analyzed data from individuals in New Zealand. A similar hierarchical structural result was obtained with an Australian sample (Slade & Watson, 2006). In addition, a study conducted in the Netherlands also found that the hierarchical INT–EXT model provided the best fit to the data (Vollebergh et al., 2001).

The studies discussed above gave promising indications that an INT–EXT model of psychopathology—whether with a unitary or hierarchical INT factor—would be replicated cross-culturally. However, the New Zealand, Australian, and Dutch samples were all drawn from Western countries. Replications of INT–EXT from other countries might provide even stronger evidence about the cross-cultural invariance of this structure.

To address the latent factors of psychopathology to an even broader international degree, one study examined World Health Organization mental health data from 14 countries (Krueger et al., 2003). The countries represented a broad array of cultures and geographic locales: Brazil, Chile, China, France, Germany, Greece, India, Italy, Japan, the Netherlands, Nigeria, Turkey, the United Kingdom, and the United States. Four models, including a one-factor, a two-factor, and two three-factor models, were fitted separately to symptom count data from individuals within each country, thus yielding 14 best-fitting models. Even across these diverse cultures, the two-factor INT–EXT model tended to fit the data best; this model was superior in 12 of the 14 countries. In the remaining two countries (the United States and Germany), a three-factor model was superior, but even it resembled INT–EXT, with an alcohol-related EXT-type factor and two other factors (depression–anxiety and somatization) that were more INT-related.

These results highlight the apparent universality of the higher-order factors of psychopathology. These studies also emphasize the promise of the INT–EXT model as a conceptualization that functions well for many people regardless of cultural, geographic, socioeconomic, and other individual differences. Indeed, the replication of the two-factor structure from Chile to China, Italy to India, Nigeria to the Netherlands, may represent the strongest evidence yet in the search for the common underlying structure of numerous major mental disorders.

Gender Invariance of INT–EXT

In addition to cross-cultural structural invariance, gender invariance would be another worthwhile attribute of INT–EXT. Gender invariance would suggest that the INT–EXT structure captures a wide variety of psychopathology well in both men and women. If INT–EXT were not invariant, it is possible that men's mental disorders might best be captured by one model (e.g., a one-factor "general psychopathology" model), while women's might best be captured by another (e.g., a hierarchical INT–EXT model). Such an outcome could seriously limit the utility and universality of the two-factor model of psychopathology.

The structure of INT–EXT does seem to be invariant across genders. Krueger (1999) found the INT–EXT model to have a superior fit in the complete utilized data set; further analyses replicated the INT–EXT structure in several ways by creating subsamples of the data. One such analysis modeled the structure of mental disorders separately in male and female participant subsamples. When the sample was divided approximately in half by gender, the hierarchical three-factor model showed the best model fit in both groups. These results support the gender-based structural invariance of the INT–EXT model.

A recent study conducted a more stringent test of factorial invariance across gender (Kramer et al., 2008). This study tested increasingly strict levels of invariance (i.e., structural, metric, strong, and strict invariance; see the Kramer and colleagues (2000) paper for a detailed discussion of these models) to determine the highest level of invariance seen in INT–EXT across genders. A strong invariance model best fit the data, suggesting that gender differences in the mean levels of INT and EXT accounted for observed gender differences in symptom

levels. Strong invariance also implies that the structure (i.e., a two-factor INT–EXT model) and factor loadings of the disorders on the latent factors are not significantly different between men and women. Thus this analysis indicates that INT–EXT provides a gender-unbiased means of conceptualizing the underlying structure of many forms of psychopathology.

Age Invariance of INT–EXT

Another question of invariance relates to whether or not the INT–EXT structure functions well as a model of individuals' psychopathology, regardless of their age. As mentioned above, the child psychopathology research literature has long posited the existence of underlying factors to account for disorder comorbidity (Achenbach & Edelbrock, 1978, 1984). However, most recent INT–EXT research has utilized samples ranging from early adulthood to middle age to older adulthood. For example, Krueger (1999) utilized data from participants who ranged from 15 to 54 years of age; Vollebergh and colleagues' (2001) participants ranged from 18 to 64 years of age; and Kramer and colleagues (2008) analyzed data from participants ages 38–76. These studies thus included a very broad array of younger and older adults, but children tended not to be a focus of these analyses.

A few studies have drawn on the seminal factor-analytic work of Achenbach and Edelbrock (1978, 1984) and investigated childhood comorbidity and the INT–EXT model. In one such study, conducted by Lahey and colleagues (2008), mental disorders in children and adolescents were modeled via confirmatory factor analysis to determine the best-fitting underlying structure. Data on several commonly occurring mental disorders (including oppositional defiant disorder, separation anxiety disorder, and the hyperactivity–impulsivity and inattention symptoms of attention-deficit/hyperactivity disorder) from 4,409 twin children and adolescents, as well as caregiver report data, were analyzed to determine the best-fitting structure to account for observed comorbidity levels. Several dimensions emerged from these analyses, and these dimensions were found to be organized hierarchically in an INT–EXT model. Due to the complexity and number of the analyses conducted, interested readers are referred to the Lahey and colleagues paper for details. For our purposes, however, the major finding of this study is that even in youth, it seems that the INT–EXT model of mental disorders functions well as a structure for many forms of psychopathology.

Unlike the long tradition of internalizing and externalizing research in children, and the recent focus of structural psychopathology research on adults of widely varying ages, little attention has been paid to the structure of mental disorders in older adults. As discussed above, several of the INT–EXT studies' community samples included older adults. Even so, we are aware of no studies that have examined the structural invariance of INT–EXT over the lifespan. A few studies that have been conducted on related topics, however, obtained results that hold promise for possible invariance of INT–EXT in older adulthood. Teachman, Siedlecki, and Magee (2007) investigated the structural invariance of a hierarchical factor that resembled INT in several ways. The authors compared structural models of different age groups on state arousal, trait anxiety, general well-being, neuroticism, and state positive and negative affect. The best-fitting model—a hierarchical "tripartite" model, made up of a higher-order negative affect factor with anxious arousal and low positive affect as subfactors—bore some resemblance to the three-factor INT model derived from previous research. Further analyses suggested that this model was invariant across different age groups. Although these results are heartening in the search for an age-unbiased conceptualization of psychopathology and constitute an important step in this direction, the variables utilized can only serve as a proxy for psychopathology.

The Stability of INT–EXT

We now leave behind the notion of invariance to discuss a final topic relevant to INT–EXT conceptualization and research: stability over time. Stability of the INT–EXT factors could hold promise for notions of etiology, course, and remission of mental disorders. For example, if it were the case that an underlying predisposition to high levels

of internalizing were relatively constant over time, but the particular manifestations of this internalizing (e.g., major depression, generalized anxiety disorder) changed over time, researchers and clinicians could develop a better understanding of the emergence of manifest mental disorders.

To our knowledge, only two studies have examined the temporal stability of INT as individuals age. The first study investigated the stability of INT and EXT from age 18 to age 21 (Krueger et al., 1998). The correlation between INT factor scores at ages 18 and 21 was about .69, and the correlation between EXT factor scores over the same time period was around .86. This study indicates that over 3 years, both INT and EXT remain relatively stable. An individual's level of internalizing at age 18 accounted for approximately 48% ($.69^2$) of the variance in her level of internalizing at age 21. Externalizing showed an even higher level of stability: Externalizing at age 18 accounted for approximately three-quarters ($.86^2 = .74$) of the variance in externalizing at age 21.

The second study to investigate INT–EXT stability over time followed a large, nationally representative Dutch sample for 1 year (Vollebergh et al., 2001). This study, which replicated previous findings that INT has distress and fear subfactors, examined the stability of the INT subfactors individually. The stability of these subfactors was high: Distress over a year was stable at a correlation of .85, and fear showed a stability correlation of .89. Consistent with previous research (Krueger et al., 1999), EXT was significantly more stable than either of the INT subfactors, and it had a stability correlation of .96. Thus, over 1 year, an individual's level of latent externalizing remained almost perfectly stable. These results for INT–EXT stability are somewhat higher than those reported by Krueger and colleagues (1998), which is to be expected. The Netherlands results investigated stability for 1 year, which was high; the Krueger and colleagues study investigated stability over *3* years, which resulted in slightly less stability over a longer period of time. The results of these two studies indicate that INT and EXT are highly stable over at least relatively short periods of time. In addition, INT and its subfactors appear to be less stable than EXT.

Conclusion and Future Directions

This chapter has reviewed the empirical literature on underlying structure of many common major mental disorders. Two factors—INT and EXT—have emerged as the latent constructs that account for a great deal of the observed comorbidity between disorders. Across numerous studies, this model has shown superior fit to a variety of other models, including a one-factor "general psychopathology" model and models based on the DSM-IV's rationally derived disorder groupings. Most studies have supported a hierarchically organized INT factor, with subfactors of distress and fear. Expansions of the INT–EXT model have led to the inclusion of more disorders under each factor. Research has demonstrated that the INT–EXT structure remains largely invariant across numerous cultures and geographic regions, both genders, treatment-seeking versus nontreatment-seeking status, and (to a lesser extent) ages. The INT and EXT factors show notable relations to each other: INT–EXT correlations indicate that these two factors are moderately related and share a good deal of overlapping variance. Finally, over 1-year and 3-year periods, the stability of the INT and EXT factors was high, indicating that an individual's underlying levels of internalizing and externalizing remain relatively constant, at least over the short term.

The INT–EXT structure is a well-replicated way of modeling and understanding the underlying structure of a great deal of common mental disorders. Application of the INT–EXT model has several potential benefits. First, and on a most basic level, such a structure could be applied to nosological systems (e.g., DSM-V) as a more empirically derived means of grouping disorders under common headings. Second, INT-EXT allows us to conceptualize and explain comorbidity between disorders—some of which might appear conceptually unrelated, given their presentations. Third, this two-factor model can allow researchers to investigate the etiology of mental health issues, whether via longitudinal designs or via twin and family studies. For instance, it may be the case that the levels of latent internalizing and externalizing in an individual remain relatively stable over time, whereas

the manifestations of these underlying levels fluctuate markedly throughout the lifespan. Perhaps an individual with high internalizing would maintain this level from adolescence through adulthood, but in adolescence would show transitory anxiety-related symptoms and in adulthood would show recurrent mood-related symptoms. Twin and family studies would allow for the parsing of etiological influences (e.g., genes and environment) on INT and EXT, as well as on the observable disorders. Finally, INT–EXT has conceivable treatment-related applications: Interventions might target the underlying internalizing or externalizing predisposition, in conjunction with the particular psychopathological manifestation at any given time (e.g., an individual's level of internalizing might be treated in addition to his or her currently presenting major depression). Addressing the latent levels of internalizing and externalizing might allow for enhanced relapse prevention efforts and the like.

Although the two-factor INT–EXT model itself seems well established, more research is needed in related areas. First, psychotic disorders are not included in the current model. Limited previous research suggests that schizophrenia and other psychotic disorders are not associated with either INT or EXT, but instead may form a *third* higher-order factor of psychopathology (Wolf et al., 1988). More studies of this topic are necessary, but they will probably be difficult to conduct. Modeling the structure of psychotic disorders, while possible, presents several challenges. The low base rate of these disorders will require data sets that include them with sufficient statistical frequency (e.g., a large data set of individuals from a psychotic disorders clinic).

A second future direction involves the link between personality and psychopathology. Many researchers have attempted to integrate personality and psychopathology in recent years (see Krueger & Tackett, 2006, for recent theory in this area). The increased inclusion of normal and pathological personality (e.g., personality disorders) in the INT–EXT model could serve to enhance this effort and clarify the role that personality plays in the underlying structure of psychopathology. Several notable studies have already been conducted in this regard. Antisocial personality disorder has already been included in the modeling of EXT by some researchers, as discussed above. In addition, several personality traits and temperaments (e.g., negative emotionality, disinhibition) have been associated with INT–EXT as well as with the intrafactor disorder comorbidity (Khan et al., 2005). These findings are promising and indicate the need for further research in this area toward the creation of a unified model of personality and psychopathology. A third future direction would be to expand our understanding of INT–EXT invariance, especially with regard to aging and later life. Although some studies have addressed this issue tangentially, more research seems warranted, especially in light of a rapidly aging population in many countries.

References

Achenbach, T. M., & Edelbrock, C. S. (1978). The classification of child psychopathology: A review and analysis of empirical efforts. *Psychological Bulletin*, *85*(6), 1275–1301.

Achenbach, T. M., & Edelbrock, C. S. (1984). Psychopathology of childhood. *Annual Review of Psychology*, , *35*, 227–256.

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., rev.). Washington, DC: Author.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.

Brim, O. G., Ryff, C. D., & Kessler, R. C. (Eds.). (2004). *How healthy are we?: A national study of well-being at midlife*. Chicago: University of Chicago Press.

Brown, T. A. (2006). *Confirmatory factor analysis for applied research*. New York: Guilford Press.

Clark, L. A. (2005). Temperament as a unifying basis for personality and psychopathology. *Journal of Abnormal Psychology*, *114*, 505–521.

Feinstein, A. R. (1970). The pre-therapeutic classification of co-morbidity in chronic disease. *Journal of Chronic Disease*, *23*, 455–468.

Helzer, J. E., Kraemer, H. C., Krueger, R. F., Wittchen, H., Sirovatka, P. J., & Regier, D. A. (2008). *Dimensional approaches in diagnostic classification: Refining the research agenda*

for DSM-V. Arlington, VA: American Psychiatric Association.

Kendler, K. S. (1993). Twin studies of psychiatric illness: Current status and future directions. *Archives of General Psychiatry*, *50*, 905–915.

Kendler, K. S., Prescott, C. A., Myers, J., & Neale, M. C. (2003). The structure of genetic and environmental risk factors for common psychiatric and substance use disorders in men and women. *Archives of General Psychiatry*, *60*, 929–937.

Kessler, R. C., Chiu, W. T., Demler, O., & Walters, E. E. (2005). Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, *62*, 617–627.

Khan, A. A., Jacobson, K. C., Gardner, C. O., Prescott, C. A., & Kendler, K. S. (2005). Personality and comorbidity of common psychiatric disorders. *British Journal of Psychiatry*, *186*, 190–196.

Klein, D. N., & Riso, L. P. (1993). Psychiatric disorders: Problems of boundaries and comorbidity. In C. G. Costello (Ed.), *Basic issues in psychopathology* (pp. 19–26). New York: Guilford Press.

Kramer, M. D., Krueger, R. F., & Hicks, B. M. (2008). The role of internalizing and externalizing liability factors in accounting for gender differences in the prevalence of common psychopathological syndromes. *Psychological Medicine*, *38*, 51–61.

Krueger, R. F. (1999). The structure of common mental disorders. *Archives of General Psychiatry*, *56*, 921–926.

Krueger, R. F. (2005). Continuity of Axes I and II: Toward a unified model of personality, personality disorders, and clinical disorders. *Journal of Personality Disorders*, *19*, 233–261.

Krueger, R. F., Caspi, A., Moffitt, T. E., & Silva, P. A. (1998). The structure and stability of common mental disorders (DSM-III-R): A longitudinal–epidemiological study. *Journal of Abnormal Psychology*, *107*(2), 216–227.

Krueger, R. F., Chentsova-Dutton, Y. E., Markon, K. E., Goldberg, D., & Ormel, J. (2003). A cross-cultural study of the structure of comorbidity among common psychopathological syndromes in the general health care setting. *Journal of Abnormal Psychology*, *112*(3), 437–447.

Krueger, R. F., & Markon, K. E. (2006). Reinterpreting comorbidity: A model-based approach to understanding and classifying psychopathology. *Annual Review of Clinical Psychology*, *2*, 111–133.

Krueger, R. F., Markon, K. E., Patrick, C. J., Benning, S. D., & Kramer, M. D. (2007). Linking antisocial behavior, substance use, and personality: An integrative quantitative model of the adult externalizing spectrum. *Journal of Abnormal Psychology*, *116*(4), 645–666.

Krueger, R. F., & Piasecki, T. M. (2002). Toward a dimensional and psychometrically informed approach to conceptualizing psychopathology. *Behaviour Research and Therapy*, *40*, 485–500.

Krueger, R. F., & Tackett, J. L. (Eds.). (2006). *Personality and psychopathology*. New York: Guilford Press.

Lahey, B. B., Rathouz, P. J., Van Hulle, C., Urbano, R. C., Krueger, R. F., Applegate, B., et al. (2008). Testing structural models of DSM-IV symptoms of common forms of child and adolescent psychopathology. *Journal of Abnormal Child Psychology*, *36*, 187–206.

Lilienfeld, S. O., Waldman, I. D., & Israel, A. C. (1994). A critical examination of the use of the term and concept of *comorbidity* in psychopathology research. *Clinical Psychology: Science and Practice*, *1*, 71–83.

Markon, K. E., & Krueger, R. F. (2005). Categorical and continuous models of liability to externalizing disorders: A direct comparison in NESARC. *Archives of General Psychiatry*, *62*, 1352–1359.

Meredith, W. (1993). Measurement invariance, factor analysis, and factorial invariance. *Psychometrika*, *58*(4), 525–543.

Mineka, S., Watson, D., & Clark, L. A. (1998). Comorbidity of anxiety and unipolar mood disorders. *Annual Review of Psychology*, *49*, 377–412.

Neale, M. C., & Kendler, K. S. (1995). Models of comorbidity for multifactorial disorders. *American Journal of Human Genetics*, *57*, 935–953.

Slade, T., & Watson, D. (2006). The structure of common DSM-IV and ICD-10 mental disorders in the Australian general population. *Psychological Medicine*, *36*, 1593–1600.

Teachman, B. A., Siedlecki, K. L., & Magee, J. C. (2007). Aging and symptoms of anxiety and depression: Structural invariance of the tripartite model. *Psychology and Aging*, *22*(1), 160–170.

Thurstone, L. L. (1947). *Multiple-factor analysis*. Chicago: University of Chicago Press.

Trull, T. J., & Durrett, C. A. (2005). Categorical and dimensional models of personality disorder. *Annual Review of Clinical Psychology*, *1*, 355–380.

Vollebergh, W. A. M., Iedema, J., Bijl, R. V., de Graaf, R., Smit, F., & Ormel, J. (2001). The structure and stability of common mental disorders: The NEMESIS study. *Archives of General Psychiatry*, *58*, 597–603.

Watson, D. (2005). Rethinking the mood and anxiety disorders: A quantitative hierarchical model for DSM-V. *Journal of Abnormal Psychology*, *114*(4), 522–536.

Widiger, T. A., & Samuel, D. B. (2005). Diagnostic categories or dimensions? A question for the *Diagnostic and Statistical Manual of Mental Disorders*—Fifth Edition. *Journal of Abnormal Psychology*, *114*(4), 494–504.

Wolf, A. W., Schubert, D. S. P., Patterson, M. B., Grande, T. P., Brocco, K. J., & Pendleton, L. (1988). Associations among major psychiatric diagnoses. *Journal of Consulting and Clinical Psychology*, *56*(2), 292–294.

World Health Organization. (1992). *International statistical classification of diseases and related health problems* (10th rev.). Geneva: Author.

CHAPTER 12

The Connections between Personality and Psychopathology

Susan C. South
Nicholas R. Eaton
Robert F. Krueger

Personality and psychopathology are inextricably linked, as knowledge of one invariably complements our understanding of the other. This overlap is reflected in the numerous investigations of the associations between these two constructs (see Clark, 2005; Krueger & Tackett, 2003; Millon, 1996; Tackett, 2006; Widiger, Verheul, & van den Brink, 1999). The linkage has not always been recognized or acknowledged, however. Although closely connected in the developing years of the field of psychology, the study of personality and the study of psychopathology later broke into separate, unrelated domains, with little communication between personality/social psychologists on the one hand and clinical psychologists/psychiatrists on the other. In recent years, articles have appeared detailing both theoretical and empirical considerations of how personality and mental disorders are linked. However, there are still significant gaps in our understanding of the systematic ways in which personality and psychopathology transact.

In this chapter, we review three major types of connections between personality and psychopathology. First, we examine the associations between mental disorders as represented by Axis I of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV-TR; American Psychiatric Association [APA], 2000) and normal-range personality. In particular, we review the major domains of personality and their organization, as well as the connections between these domains and major forms of psychopathology. We then consider how the major domains of normal personality are related to Axis II personality disorders (PDs). In particular, we address the ways in which normal-range personality is currently being utilized to account for the variation in PD symptoms. Finally, we investigate the interplay between Axis I and Axis II disorders in light of evidence that supports several competing models.

Axis I Psychopathology and Normal-Range Personality

Theories of the Personality–Psychopathology Link

Currently, at least four models outline various ways in which personality and psychopathology may be linked (see Clark, Watson, & Mineka, 1994; Krueger & Tackett,

2003; Tackett, 2006; Watson & Clark, 1995; Widiger et al., 1999). These four models are known as the "vulnerability" model, the "complication/scar" model, the "pathoplasty" model, and the "spectrum" model. In the vulnerability model, preexisting personality traits or temperamental styles predispose an individual to develop mental illness. For instance, an individual who is high in the personality trait of neuroticism may be more likely to develop an anxiety disorder. The complication (or scar) model suggests that the experience of a certain form of psychopathology scars the individual's personality, changing it in key ways from premorbid functioning. An example of the scar model would be a decrease in openness following an episode of posttraumatic stress disorder (PTSD). According to the pathoplasty model, premorbid personality functioning affects the expression, course, severity, or treatment response of a disorder. An illustration of the pathoplasty model would be a heightened likelihood of suicidality in a depressed individual who also has low levels of constraint. Finally, the spectrum model would suggest that personality and psychopathology are both part of the same continuous latent dimension. The personality trait of disinhibition, for instance, is thought to be part of the same externalizing spectrum as substance use disorders (SUDs) and antisocial PD (Eaton, South, & Krueger, Chapter 11, this volume; Krueger et al., 2002).

There is probably no single correct model for the relation between personality and psychopathology. More than one of these models may be operating for a particular individual (Millon, 1996), and different models may be necessary to explain different disorders (Dolan-Sewell, Krueger, & Shea, 2001). Research results are often mixed as to which model best explains or accounts for a particular disorder. For instance, factor-analytic research finds that neuroticism falls within the same spectrum (internalizing) as major depression (South & Krueger, 2008b); other research suggests that premorbid neuroticism is a vulnerability factor for the development of depression (Clark et al., 1994), and that it affects depression levels after cognitive therapy (Clark, Vittengl, Kraft, & Jarrett, 2003). In fact, different studies have found support for all four models in relation to depression (Bagby, Joffe, Parker, Kalemba, & Harkness, 1995; Kendler, Neale, Kessler, Heath, & Eaves, 1993; Rothschild & Zimmerman, 2002; Scott, Williams, Brittlebank, & Ferrier, 1995).

Hierarchical Models of Normal Personality and Psychopathology

Although there are many different conceptualizations of personality (see Pervin & John, 1999), one in particular—trait theory—has gained the most support among researchers interested in psychopathology. According to this model of personality, patterns of thoughts, feelings, and behaviors are best captured by trait "descriptors." An ongoing question within the field of personality psychology is this: How many traits are needed to capture the full range of human personality functioning? A consensus is emerging around the notion of a hierarchical structure of personality, with a few broad factors or domains encompassing a larger number of "facets" (Goldberg, 1993; Markon, Krueger, & Watson 2005).

The most popular hierarchical model of personality, the "Big Five" model (BFM), originated from empirical studies of lexical descriptors found in the English language (Goldberg, 1990; John & Srivastava, 1999). It includes the following factors: extraversion (or surgency), or a person's characteristic tendency to seek out and desire communion with others; agreeableness, the tendency to be honest, straightforward, and cooperative in dealings with other people; conscientiousness, a reflection of how well-regulated a person is (from irresponsible, impulsive, and delinquent at one end to dutiful, disciplined, and goal-striving at the other); neuroticism, a reflection of how emotionally stable versus emotionally dysregulated a person is; and openness, generally thought to reflect a person's curiosity and interest in new and different things but has also been conceptualized as an "intellect" dimension. Similar types of factor analyses conducted on the adjective lexicons of other languages have consistently yielded factors resembling extraversion, agreeableness, conscientiousness, and neuroticism; openness has been more difficult to replicate (DeRaad, Perugini, Hrebiekova, & Szarota, 1998). Costa and McCrae (1992) later developed their own five-factor model (FFM) based on questionnaire studies. Their

model included the same five personality factors (or domains), as well as six subscales or "facets" for each domain. Their NEO Personality Inventory—Revised (NEO PI-R; Costa & McCrae, 1992) has become one of the most widely used measures of the FFM.

Two of the BFM factors, extraversion and neuroticism, appear in most major personality theories outside the BFM/FFM. Several models include three broad "superfactors" of personality. Eysenck's (1997) three-factor model included neuroticism (vs. emotional stability), extraversion (vs. introversion), and psychoticism (somewhat of a misnomer, in that it measures a disinhibition factor of personality). Following this work, Clark and Watson (1999b), proposed a model including neuroticism/negative emotionality, extraversion/positive emotionality, and disinhibition versus constraint. A similar prominent model includes negative emotionality, positive emotionality, and constraint (Tellegen, 1985). Evidence suggests that the BFM of personality is an expansion of these three basic personality domains, with neuroticism and extraversion from the BFM mapping to neuroticism/negative emotionality and extraversion/positive emotionality, respectively, while constraint combines agreeableness and conscientiousness from the BFM (Markon, Krueger, & Watson, 2005; Watson, Kotov, & Gamez, 2006).

Neuroticism is perhaps the personality factor most consistently identified with Axis I psychopathology. The connections between neuroticism and internalizing disorders (e.g., depression, anxiety), externalizing disorders (e.g., SUDs, antisocial behavior disorders), and even psychotic disorders have emerged so consistently (Goodwin & Gotlib, 2004; Krabbendam et al., 2002; Krueger, 1999; Krueger, Caspi, Moffitt, Silva, & McGee, 1996; Moffitt, Caspi, Dickson, Silva, & Stanton, 1996; Shiner, Masten, & Tellegen, 2002; van Os & Jones, 2001) that they have led some researchers to characterize neuroticism as a "noninformative marker" of psychopathology (Ormel, Rosmalen, & Farmer, 2004). Indeed, neuroticism could be thought of as nothing more than "a person's habitual level of distress." If such is the case, without a better understanding of the psychobiological underpinnings of neuroticism, the construct provides little information about the nature of psychopathology beyond simply indicating its presence (Ormel et al., 2004).

To illustrate the difficulty of interpreting the association between neuroticism and psychopathology, we use the example of major depression—the Axis I disorder studied most often in relation to neuroticism. Clark and colleagues (1994), in a review of the negative emotionality/neuroticism literature, concluded that there was support for three of the proposed models: vulnerability, pathoplasty, and scar. Since that review was published, several empirical studies have provided support for each of these models. For instance, there is considerable evidence that neuroticism is a risk factor for the development of later psychopathology, particularly the mood and anxiety disorders (Boyce, Parker, Barnett, Cooney, & Smith, 1991; Duberstein, Palsson, Waern, & Skoog, 2008; Kendler, Gatz, Gardner, & Pedersen, 2006; Kendler, Kessler, Neale, Heath, & Eaves, 1993; Ormel, Oldehinkel, & Brilman, 2001; Roberts & Kendler, 1999). Beevers, Rohde, Stice, and Nolen-Hoeksema (2007) found that adolescents who experienced an episode of major depression had higher levels of negative emotionality than a never-depressed group before, during, and after the episode, and that negative emotionality increased from baseline to the period of depression. They concluded that negative emotionality functions as a strong risk factor for the onset of depression, and stated that there was little evidence for depression having a scarring effect on personality. There is also evidence that, in support of the pathoplasty model, neuroticism affects the lifelong course and treatment of depression and dysthymia (Duggan, Lee, & Murray, 1990; Katon et al., 2002; Surtees & Wainwright, 1996).

Multivariate Models of Normal Personality and Axis I Psychopathology

More informative, perhaps, than individual studies that link one or more personality traits to a single clinical disorder are multivariate studies of the links between multiple traits (e.g., FFM domains) and multiple clinical disorders. In one of the first studies of this kind, Trull and Sher (1994) examined the patterns of interrelations between the FFM factors and clinical disorders (e.g.,

SUDs, major depression, PTSD) in a community sample of young adults. They found that the FFM distinguished between individuals who met diagnostic criteria and those who did not for several types of Axis I psychopathology. A general pattern of higher neuroticism and openness, and lower extraversion, agreeableness, and conscientiousness, was found across all forms of disorders. However, more specific patterns were found that distinguished between the disorders, particularly after comorbidity was controlled for. For example, nondepressed individuals with SUDs were distinguished by a pattern of low neuroticism, agreeableness, and conscientiousness, and high extraversion.

Malouff, Thorsteinsson, and Schutte (2005) conducted a meta-analytic study of all studies that compared the links between the BFM factors and Axis I disorders. They found that a pattern of high neuroticism, low agreeableness, low conscientiousness, and low extraversion was common across symptoms of all clinical disorders included in the analyses (i.e., mood and anxiety disorders, SUDs, eating disorders, somatic disorders, and psychotic disorders). Externalizing disorders showed the most distinct pattern of personality traits, with a combination of high extraversion, low agreeableness, and low neuroticism, which distinguished them from other types of psychopathology. Mood disorders (notable for lower extraversion than other disorders) and anxiety disorders (characterized by high levels of agreeableness) were also distinguishable by a distinct constellation of traits.

Other studies have attempted to flesh out the distinctions between different forms of clinical disorders based on FFM profiles. Ruiz, Pincus, and Schinka (2008) conducted a meta-analysis of the pattern of associations between the FFM domains and facets and externalizing psychopathology (defined as antisocial PD, SUDs, or the co-occurrence of the two disorders). Antisocial PD, SUDs, and their co-occurrence were characterized by low levels of agreeableness and conscientiousness; the co-occurrence and SUDs alone were also notable for moderate levels of neuroticism. Miller, Lynam, and Leukefeld (2003) also examined the relations between antisocial and aggressive behavior and the facets of the NEO PI-R (Costa & McCrae, 1992) neuroticism, agreeableness, and conscientiousness domains. The facets of low straightforwardness (a facet of agreeableness), low deliberation (conscientiousness), and low compliance (agreeableness) were particularly strong in their prediction of externalizing-type behaviors. Sellbom, Ben-Porath, and Bagby (2008) administered the Minnesota Multiphasic Personality Inventory–2 (MMPI-2) Restructured Clinical (RC) scales (Tellegen et al., 2003) and the NEO PI-R to patients in a psychiatric facility. A factor analysis of the RC scales revealed a three-factor solution of internalizing, externalizing, and thought disturbance. In addition, the internalizing factor was highly correlated with neuroticism and its facets; externalizing was negatively correlated with facets of conscientiousness and agreeableness; and thought disturbance was only moderately negatively correlated with two facets, values (openness) and trust (agreeableness).

Researchers have also examined how normal personality traits are related to a higher-order structure of the Axis I clinical disorders. Population-based studies of the comorbidity among Axis I clinical disorders have factor-analyzed diagnostic information from structured diagnostic interviews. Evidence has converged on a two-factor internalizing (mood and anxiety disorders) and externalizing (SUDs and conduct disorder) model of psychopathology (Eaton et al., Chapter 11, this volume; Krueger & Markon, 2006). This work follows logically from research in the child psychopathology field, which has consistently identified an internalizing–externalizing pattern of clinical pathology. Importantly for understanding the links between personality and psychopathology, studies have found that normal personality traits fit within these factor models in predictable and expected ways. The personality trait of neuroticism fits well within the internalizing spectrum (Kendler, Prescott, Myers, & Neale, 2003; South & Krueger, 2008b), while the personality dimension of disinhibition–constraint is subsumed under the externalizing factor (Krueger et al., 2002). In further support of a spectrum model of psychopathology, O'Connor (2002) found that the factor structures of both clinical disorders and normal personality traits were similar across clinical and nonclinical populations, supporting a continuum of psychopathology rather than discrete categorical distinctions.

Other Individual Difference Variables

In addition to broad-based personality traits or dimensions, researchers have examined cognitive factors that are related to psychopathology. Biased forms of information processing have been observed across the emotional disorders (Mathews & MacLeod, 2005), although how they are manifested tends to differ, depending on the type of pathology. Attentional biases, for instance, are present in depressive and anxiety disorders, although attention to relevant threatening stimuli begins at an earlier, nonconscious processing stage in anxiety disorders, whereas attention is consciously directed toward mood-congruent stimuli in depression. Evidence for memory biases is more mixed, but there is generally solid evidence in favor of both a negative interpretive bias (e.g., identifying a putatively neutral stimuli as negative) and repeated negative ideation (e.g., worrying about worry) across mood and anxiety disorders.

Based on the different cognitive processing biases observed across various internalizing disorders, researchers have proposed cognitive theories to explain almost all of the mood and anxiety disorders. In particular, there are many rich and detailed cognitive theories of depression. According to the hopelessness theory of depression, people who make negative inferences about self, causality, and consequences in response to negative events are more likely to develop depression in the face of such negative events (Abramson, Metalsky, & Alloy, 1989). A prospective longitudinal study of college students found that participants with negative cognitive styles were more likely to have an onset of depression than participants without a negative cognitive style (Alloy et al., 2006). Support for the interaction between hopelessness as a cognitive vulnerability and negative life events as a stressor in the development of depression has been found for adolescents and adults (see Hyde, Mezulis, & Abramson, 2008, for a review). Similarly, the theory of ruminative response style suggests that people who think persistently and negatively about the emotions elicited by a negative event are more likely to develop depression (Nolen-Hoeksema, 2000). Rumination predicts the development of later depressive episodes, even after prior history of depression is controlled for; it also predicts anxiety disorders.

Importantly, most cognitive theories of internalizing disorders propose that the cognitive bias is a "diathesis"—that is, an inborn characteristic that precedes and elevates the risk for a subsequent disorder. This cognitive diathesis is presumed to interact with environmental events (e.g., adverse life circumstances) and to trigger the development of psychopathology, in line with the vulnerability model of the personality–psychopathology relationship. Unfortunately, the research conducted at this point has only begun to tease apart the developmental timing of the relationships between/among cognitive biases, psychopathology, and other related variables (e.g., personality traits, affective styles). Cole and colleagues (2008), for instance, found that the structure of attributional style changed over the course of childhood and acted as a diathesis for depressive symptoms only after the emergence of early adolescence.

Evidence from Behavior Genetics

Behavior genetics, or the study of the genetic and environmental influences on individual differences, is an important means by which to examine the shared etiology of personality and psychopathology. Typically, behavior genetic research has been used to parse the variance in personality traits or mental disorders into the proportion due to (1) genetic effects (h^2), or heritability; (2) shared or "common" environmental effects (c^2), or those experiences shared by members of the same family that make the individuals more similar; and (3) unique environmental effects (e^2), or those events that are not shared by family members and that make the individuals unique (Plomin, DeFries, McClearn, & McGuffin, 2008). Research has consistently shown that almost every individual difference variable, including both normal and pathological personality and psychopathology, has a genetic component (Turkheimer, 1998). More recently, behavior genetic modeling has gone beyond simple estimates of heritability; researchers are beginning to use these methods to examine the etiology of the links between personality traits and psychopathology.

One line of research from behavior genetic modeling of personality and psychopathology has examined shared genetic and environmental influences. To conduct these analyses, researchers make use of a biometric model called a "bivariate Cholesky decomposition," which estimates the genetic and environmental variance shared between two phenotypes (e.g., a personality trait and a mental disorder) and unique to each. For example, it is possible to determine whether the genetic variance in variable *A* (e.g., neuroticism) is correlated with the genetic variance in variable *B* (e.g., depression) by estimating the "genetic correlation" (*rG*) between variables *A* and *B*. (It is also possible to examine whether the environmental variance in variable *A* is correlated with the environmental variance in variable *B* by estimating the "environmental correlation," *rE*, between variables *A* and *B*.) Numerous studies have now been conducted showing substantial shared genetic influences between personality and different forms of psychopathology, including neuroticism and mood/anxiety disorders (Hettema, Neale, Myers, Prescott, & Kendler, 2006; Kendler, Prescott, Myers, & Neale, 2003) as well as constraint and externalizing disorders (Krueger et al., 2002; Young, Stallings, Corley, Krauter, & Hewitt, 2000). This work, unfortunately, is relatively uninformative in the pattern of temporal relations between personality and psychopathology delineated by the different causal theories; however, it does tell us that the personality–psychopathology link is at least partly genetic.

In addition to showing shared genetic influences between personality traits and various forms of psychopathology, research has provided evidence that personality traits influence the environments people choose. This idea is known as "gene–environment correlation," or the degree to which a genotype influences the likelihood of exposure to a specific environment (Plomin et al., 2008). As in the research described above, a Cholesky decomposition can be conducted with a personality trait and an environmental measure to determine whether or not there are any shared genetic influences. This is possible because research has shown that putatively "environmental" measures have a genetic basis (Kendler & Baker, 2007). The finding of genetic influences on the environment has been interpreted as suggesting that genetically based personality traits influence the types of environments that people seek out. South, Krueger, Johnson, and Iacono (2008) found genetic correlations between adolescents' reports of the Multidimensional Personality Questionnaire (MPQ; Tellegen & Waller, in press) higher-order personality factors of positive emotionality, negative emotionality, and constraint, and adolescent–parent relationship quality, providing support for the role of personality in shaping the nature of environment. If it could then be shown that these personality-influenced environments moderate genetic susceptibility to psychopathology—an idea known as "gene–environment interaction"—these results would constitute strong evidence for the vulnerability model of personality–psychopathology links.

Gene–environment interactions have been difficult to study empirically until recently, but new types of biometric moderation models (Purcell, 2002) estimate the heritability of psychopathology at different levels of the environmental moderator variable; thus, instead of determining a heritability estimate averaged over the entire sample-specific population, it is possible to determine the heritability estimate for subgroups within the population who differ on the environmental moderator variable. For example, South and Krueger (2008b) used a nationwide U.S. twin sample to examine gene–environment interaction in the study of internalizing psychopathology (composed of depression, generalized anxiety disorder, panic disorder, and neuroticism). They found that the heritability of an internalizing factor score was highest ($h^2 = 29$) at the lowest levels of marital quality, while heritability was actually quite low ($h^2 = .05$) when marital quality was high. Similar instances of this type of gene–environment interaction have begun appearing in the literature. In these studies, greater genetic influences on psychopathology have been found at higher levels of environmental adversity. This is true of delinquent peer groups and conduct (Button et al., 2007); higher parental conflict and adolescent antisocial behavior (Feinberg, Button, Neiderhiser, Reiss, & Heatherington, 2007); lower levels of parental monitoring and adolescent smoking (Dick et al., 2007); and negative life events and higher maternal

discipline and adolescent depression (Lau & Eley, 2008).

Behavior genetic modeling has also been used to examine another viable model of the personality–psychopathology link: the spectrum model. Much as the basic univariate twin model has been extended to the bivariate decomposition model to examine genetic influences common to personality and psychopathology, other useful extensions of biometric modeling support the hypothesis that personality lies on a similar dimensional spectrum with psychopathology. In particular, "multivariate biometric modeling" of personality and psychopathology variables allows a researcher to decompose not only the variance within phenotypes, but also the covariance between phenotypes, into genetic and environmental influences. To examine the spectrum model of personality and psychopathology, researchers can compare two types of multivariate biometric models. The model which would support the spectrum hypothesis is called the "common-pathway" model. In this model, a single latent phenotype explains all the covariation in a set of variables, and it is influenced by one set of additive genetic, shared environmental, and nonshared environmental influences. In essence, this model is an extension of a phenotypic factor analysis, where the personality and psychopathology variables are subsumed under one overall factor, and the differences between people on this latent factor can be decomposed into genetic and environmental influences. A competing model to explain the covariation between personality and psychopathology is the "independent-pathway" model, which specifies direct links to the personality and psychopathology phenotypes from one or more additive genetic and shared and nonshared environmental influences common to all the variables and unique to each individual variable. Thus the independent-pathway model does not require that the covariation between the personality variable(s) and the psychopathology variable(s) be mediated through a common phenotypic factor.

Only recently have researchers begun to test these competing models directly. Krueger and colleagues (2002), using a twin sample of more than 600 adolescents, found that a common-pathway model best explained the covariation between adolescent antisocial behavior, conduct disorder, alcohol dependence, drug dependence, and the personality trait of constraint (reverse-scored). Other research groups have also found support for a common-factor model of externalizing behavior (Kendler et al., 2003; Young et al., 2000). These results closely parallel large-scale phenotypic factor analyses of the comorbidity among different forms of psychopathology (Krueger & Markon, 2006). There have been only three biometric studies of the internalizing disorders, which have yielded slightly different results. In an earlier study, Kendler and colleagues (2003) found a genetic structure that closely paralleled the phenotypic structure of internalizing. Hettema and colleagues (2006) later conducted a similar analysis, with the addition of the personality trait of neuroticism, and found two overlapping genetic factors; one of these was shared largely between neuroticism, major depression, generalized anxiety, panic disorder, and the phobias, and the other factor accounted for covariation between major depression, generalized anxiety, and panic disorder, independent of neuroticism. Finally, South and Krueger (2008b) found that one factor accounted for the genetic influences on symptoms of depression, generalized anxiety, and panic attacks and levels of neuroticism.

Axis II Personality Disorders and Normal-Range Personality

DSM-IV-TR lists 10 personality disorders (PDs), grouped into three clusters according to broad, overarching descriptors. Cluster A is composed of the odd, eccentric disorders (paranoid, schizoid, schizotypal); Cluster B is composed of the dramatic/emotional/erratic disorders (antisocial, borderline, histrionic, narcissistic); and Cluster C is composed of the anxious, fearful disorders (avoidant, dependent, obsessive–compulsive). Many of these PDs have been codified in the diagnostic nomenclature since the first edition of DSM (APA, 1952), but it was with the publication of DSM-III (APA, 1980) that PDs were moved to a distinct axis and each individual PD was defined according to its own polythetic set of criteria. The PDs originated from myriad clinical and theoretical formulations, including a conceptualization

as formes frustes of more acute, debilitating pathology (e.g., schizotypal PD and schizophrenia) and Freudian notions of poor ego development (e.g., narcissistic PD) (Frances & Widiger, 1986; Livesley, 2001b). None of the current PDs originated in more broad-based empirical research focusing on adaptive, normal personality functioning.

There are numerous difficulties with the current diagnostic conceptualizations of the PDs, which have been elaborated at length elsewhere. These include excessive comorbidity, both among the PDs and between PDs and Axis I psychopathology (see below), poor reliability, poor convergent and discriminant validity, and inadequate coverage (Clark, Livesley, & Morey, 1997; First et al., 2002; Livesley, 2003; Millon, 2002). As a result, many commentators have suggested revisions to the current diagnostic system. Arguments have converged on a dimensional assessment of personality pathology (Widiger & Trull, 2007). The reliability and stability of personality assessment are better with dimensional measures; research does not generally support a discrete boundary between normal and abnormal personal functioning; and empirical evidence suggests that personality structure is similar across clinical and nonclinical samples (Clark & Watson, 1999a; Livesley, 2003; Livesley, Jang, & Vernon, 1998; O'Connor, 2002). Thus most of the research on Axis II pathology and normal personality has focused on what systems of normal personality might best account for the variance in personality pathology. We discuss the two general classes of these studies: those linking DSM-defined PDs with normal personality, and those studying the associations between normal personality and dimensional models of personality pathology.

DSM-Defined PDs and Normal Personality

Researchers have attempted to characterize the individual differences in the DSM-defined PDs by using various measures of normal personality. These studies have utilized one of three general types of methodologies: comparing the structure of PDs across clinical and nonclinical samples, determining the percentage of variance in PDs that could be accounted for by normal personality traits, and determining profiles of the different PDs by using normal personality traits. Most of the work examining associations between PDs and normal personality has utilized the FFM to assess normal personality, so we focus our review on this literature.

Much of this research has found stable and reliable relations between FFM traits and PD features (Ball, Tennen, Poling, Kranzler, & Rounsaville, 1997; Costa & McCrae, 1990; Trull, 1992; Wiggins & Pincus, 1989). In a review of this research, Widiger and Costa (2002) concluded that PD traits can be thought of as maladaptive variants of the FFM traits, which account for moderate proportions of variance in PD symptoms. A recent meta-analysis of 15 independent samples confirmed that the 10 DSM-IV-TR PDs are related to the five higher-order factors in predictable and meaningful ways (Saulsman & Page, 2004). For instance, borderline PD is strongly correlated with high neuroticism and low agreeableness. In fact, most of the PDs lie in a quadrant formed by high neuroticism and low agreeableness. The FFM domains have been used to code the PD criteria in both the DSM-III-R (Widiger, Trull, Clarkin, Sanderson, & Costa, 1994) and DSM-IV (Widiger, Trull, Clarkin, Sanderson, & Costa, 2002), and recent research has extended this work to the FFM facets (Axelrod, Widiger, Trull, & Corbitt, 1997; Dyce & O'Connor, 1998; Morey et al., 2002; Trull et al., 1998). Research also suggests that the FFM can account for the diagnostic co-occurrence among the PDs (Lynam & Widiger, 2001). PD researchers (Lynam & Widiger, 2001) and practicing clinicians (Samuel & Widiger, 2004) have used FFM descriptors to provide ratings of the PD criteria. There is good agreement among the FFM ratings of the PDs across the different type of raters; most differences arise from the clinicians' tendency to present a more detailed description of the PDs by using all of the possible FFM facets, instead of limiting their ratings to facets suggested by each specific PD (Mullins-Sweatt & Widiger, 2006).

The longitudinal association between PDs and normal personality is also worthy of consideration. PDs were originally separated from Axis I clinical disorders because the latter were presumed to be more episodic, while the former was thought to be chronic,

enduring, and stable. Normal personality is thought to be relatively stable (Costa & McCrae, 1994), particularly in terms of rank-order consistency after middle age (Roberts & DelVecchio, 2000). Although the stability of PD diagnoses per se has varied considerably (see McDavid & Pilkonis, 1996, for a review), the correlational stability of PD symptom counts over time is higher (Shea et al., 2002). In the first longitudinal study of the relationship between the FFM traits and DSM-defined PDs, Warner and colleagues (2004), using the same data from the Collaborative Longitudinal Study of Personality Disorders as Shea and colleagues (2002), examined whether or not stability of PD symptoms was attributable to the stability of normal personality traits underlying the disorders. For three PDs, schizotypal, borderline, and avoidant, the authors found that FFM traits thought to underlie the disorders (as measured by an expert consensus approach; Lynam & Widiger, 2001) predicted change in the disorders over time, but that change in the disorders did not predict change in the FFM factors. Warner and colleagues concluded that normal personality traits form the core of personality pathology. For obsessive–compulsive PD, there were few significant effects across time between the personality traits and the disorder, possibly suggesting that FFM traits do not adequately capture this particular PD.

Some have argued that the FFM factors (as assessed by the NEO PI-R) are not useful (Block, 1995) or comprehensive enough (Livesley, 2001a; Morey et al., 2002) to capture the maladaptive nature of PDs. In particular, researchers have suggested that the FFM does not distinguish adequately among the different DSM-defined PDs (Coolidge et al., 1994; Zweig-Frank & Paris, 1995). Haigler and Widiger (2001) reworded some of the NEO items to capture more adequately the extreme, undesirable, maladaptive variants of the scales, which increased correlations between the PDs and the NEO scales. Other research has confirmed that the FFM facets account for the differences between PDs better than the higher-order factors do (Axelrod et al., 1997; Dyce & O'Connor, 1998; Morey et al., 2002; Trull et al., 1998). However, future research is needed to determine whether the FFM can completely account for the variance in PDs, or whether, as has been suggested, measures of abnormal personality are needed above and beyond the FFM (Reynolds & Clark, 2001; Schroeder, Wormworth, & Livesley, 1992).

Dimensional Models of Personality Pathology and Normal Personality

As noted above, there has been a growing consensus that PDs should be conceptualized dimensionally (Widiger & Simonsen, 2005). At the same time, there is strong support for incorporating both pathological and normal personality within the same integrative, hierarchical structure of personality (e.g., Markon et al., 2005). Thus recent research has been focused on finding a common factor structure across the myriad dimensional measures of personality pathology, many of which actually incorporate both adaptive and maladaptive personality traits within the same framework. Several dimensional measures of personality pathology have now been developed, including the Dimensional Assessment of Personality Pathology—Basic Questionnaire (DAPP-BQ; Livesley & Jackson, 2002); three editions of the Millon Clinical Multiaxial Inventory (MCMI, Millon, 1983; MCMI-II, Millon, 1987; MCMI-III, Millon, 1994); the MMPI-2 (Butcher, Dahlstrom, Graham, Tellegen, & Kaemmer, 1989); and the Schedule for Nonadaptive and Adaptive Personality (SNAP; Clark, 1993). These instruments vary in the ways that they conceptualize personality pathology. The MCMI, for instance, uses the DSM-defined PD constructs, but scores are presented dimensionally. The DAPP-BQ and the SNAP were developed through an iterative structural approach, in which personality traits thought to cover the range of personality pathology were subjected to structural modeling to develop subordinate and superordinate factors.

Across these alternative dimensional models, a common hierarchical framework has begun to appear. This is not surprising, as the goal of these different personality measures has been to identify the key dimensions of maladaptive functioning that can explain the variation in the existing PD categories. Factor-analytic structures of the various measures appear to be converging on a four-factor structure (Widiger & Simonsen, 2005). The DAPP-BQ has four higher-order

factors of emotional dysfunction, dissocial behavior, inhibitedness, and compulsivity (Livesley et al., 1998); the SNAP has three higher-order domains of positive affectivity, negative affectivity, and constraint (Clark, 1993). Joint factor analyses of the DAPP-BQ and the SNAP have found four factors: positive affectivity/extraversion, negative affectivity/neuroticism, antagonism, and constraint (Clark & Livesley, 2002; Clark, Livesley, Schroeder, & Irish, 1996). Across various dimensional personality measures, four domains consistently appear: extraversion (vs. introversion), antagonism (vs. compliance), constraint (vs. impulsivity), and emotional dysregulation (vs. emotional stability; see Widiger & Simonsen, 2005, for a review). Covered by the extraversion domain are subscales of stimulus seeking, exhibitionism, assertiveness, sociability, and detachment. Lower-order facets of antagonism include mistrust, aggression, suspiciousness, altruism, compliance, and submissiveness. Subscales covered by the constraint domain include dutifulness, achievement, ambitiousness, responsibility, and self-discipline. Finally, emotional dysregulation subsumes the lower-order facets of self-harm, dependency, alienation, depressiveness, and vulnerability.

In two different studies, Markon and colleagues (2005) empirically evaluated the higher-order structure of normal and abnormal personality. First, they conducted a meta-analysis of the joint factor structure of five of the most widely used measures of normal and abnormal personality: the DAPP-BQ (Livesley & Jackson, in press); the Eysenck Personality Questionnaire (EPQ and EPQ-R; Eysenck & Eysenck, 1975; Eysenck, Eysenck, & Barrett, 1985); the MPQ (Tellegen, 1985); variants of the NEO PI broad domain scales (NEO PI, NEO PI-R, and NEO-FFI; Costa & McCrae, 1985, 1992); and the Temperament and Character Inventory and its predecessor (Cloninger, 1987; Cloninger, Svrakic, & Przybeck, 1993). Results indicated that there were no more than five interpretable factors, and that the five-factor model strongly resembled the FFM found in the normal personality literature. Furthermore, the four-factor model was consistent with both empirical (Livesley et al., 1998) and theoretical (Widiger & Simonsen, 2005) accounts of a four-factor model of normal and abnormal personality. Three- and two-factor models were also tenable, and resembled the three-factor (positive emotionality, negative emotionality, and constraint) models of Clark and Watson (1999b), Tellegen (1985), and Eysenck (1994), and Digman's (1997) model of alpha (neuroticism, agreeableness, and conscientiousness) and beta (extraversion and openness). Markon and colleagues obtained essentially the same results when they conducted a factor analysis on a separate sample of data collected from undergraduate students using the NEO PI-R facet scales (Costa & McCrae, 1992), the EPQ-R (Eysenck & Eysenck, 1975; Eysenck et al., 1985), the SNAP (Clark, 1993), and the Big Five Inventory (John, Donahue, & Kentle, 1991).

Evidence from Behavior Genetics

Heritability estimates of the DSM-defined PDs have varied considerably, depending on sample and type of assessment instrument utilized (see Livesley & Jang, 2008, for a recent review). Torgersen and colleagues (2000) reported heritabilities ranging from 28% to 79% across all 10 DSM-IV PDs based on a structured interview. More recent analyses, also using a structured interview, found heritabilities at the lower end of the estimates Torgersen and colleagues found for the Cluster A and Cluster C disorders (Kendler et al., 2006; Reichborn-Kjennerud et al., 2007).

Given the extensive arguments in favor of viewing PDs as dimensional concepts, researchers have turned to examining genetic and environmental influences on dimensional measures of personality pathology. Livesley, Jang, Jackson, and Vernon (1993) conducted univariate behavior genetic models, using four higher-order traits found in factor analyses of the DAPP-BQ. They reported heritabilities of 53% for emotional dysfunction, 50% for dissocial behavior, 51% for inhibitedness, and 38% for compulsivity. Heritabilities for the 18 lower-order DAPP-BQ traits varied from 35% to 56%. Jang, Livesley, and Vernon (1998) later compared the genetic and environmental influences on the DAPP-BQ scales across gender. They found heritabilities of 52%, 66%, 62%, and 46% for emotional dysfunction, dissocial behavior, inhibitedness, and compulsivity

for men, and 64%, 55%, and 44% for emotional dysfunction, inhibitedness, and compulsivity for women (dissocial behavior had a nonsignificant heritability for women).

Livesley and colleagues (1998) later extended this work and determined that the phenotypic factor structure of the DAPP-BQ mapped well onto the genetic factor structure. They also found that after they controlled for the genetic influences on the four higher-order factors, there were genetic effects specific to the traits making up each of the general factors. This would suggest that there are general genetic influences shared by the broader factors (e.g., emotional dysfunction), as well as genetic influences specific to the traits that each factor subsumes.

Axis I Psychopathology and Axis II Personality Disorders

Patterns of Comorbidity

Moving the personality disorders to a separate axis in DSM-III was prompted by the pull that clinicians often felt to assign a diagnosis of *either* a more acute, transient condition (e.g., depression, anxiety), or a chronic, relatively stable character defect. Placing the PDs on Axis II did encourage greater research and understanding of these diagnoses. With this greater attention to and investment in research on the PDs came an unforeseen finding: high levels of comorbidity. Research has consistently shown that the Axis II PDs occur with each other (Bornstein, 1998; Lilienfeld, Waldman, & Israel, 1994; Livesley, 2003) and with Axis I disorders at rates greater than would be expected by chance alone (Dolan-Sewell et al., 2001; Fabrega, Ulrich, Pilkonis, & Mezzich, 1992; McGlashan, 1987; Skodol, Oldham, & Gallagher, 1999).

In a review, Dolan-Sewell and colleagues (2001) concluded that between 66% and 97% of patients with PDs also meet criteria for an Axis I disorder; similarly, between 13% and 81% of patients with an Axis I disorder can also be diagnosed with a PD. Of course, certain patterns of comorbidity are more common; these include borderline PD and major depressive disorder (Zanarini et al., 1998), as well as Cluster B PDs and SUDs (Trull, Sher, Minks-Brown, Durbin, & Burr, 2000), particularly antisocial PD and SUDs (e.g., Sher & Trull, 1994). Trull, Waudby, and Sherr (2004) demonstrated that alcohol use disorders in a nonclinical sample of 395 young adults were significantly related to Cluster B PD symptoms (particularly antisocial and borderline). Researchers have found considerable comorbidity between avoidant PD and generalized social phobia (Alden, Laposa, Taylor, & Ryder, 2002). Chambless, Fydrich, and Rodebaugh (2008) compared participants with generalized social phobia who were and were not comorbid for avoidant PD, and concluded that avoidant PD might best be considered a severe form of generalized social phobia.

The significant degree of co-occurrence of the Axis I and Axis II disorders suggests that whatever rationale exists for separating clinical disorders from PDs may not be justified. PDs are presumed to be more stable and to have an earlier age of onset than clinical disorders; as Krueger's (2005) review makes clear, there is little evidence to support either presumption.

Evidence for Models of the Relationship between Axis I and Axis II Pathology

The four theoretical models of the relationship between Axis I psychopathology and "normal" personality traits apply equally well to the links between Axis I and Axis II psychopathology. In the case of the predisposition/vulnerability model, the presence of a certain PD is thought to be a risk factor for the development of Axis I pathology. For instance, antisocial PD may be a risk factor for the development of SUDs. Alternatively, the experience of an Axis I disorder may "scar" an individual's preexisting personality, leading to the development of a PD. An anxiety disorder like agoraphobia, for example, may precede the development of dependence on a close individual that is so debilitating it reaches the level of a diagnosable PD.

According to the pathoplasty model, an individual might develop both an Axis I and an Axis II disorder (which are etiologically distinct), but the Axis II disorder will affect the course, severity, or treatment response to the Axis I disorder. Research shows that PDs do influence the course and treatment of

Axis I disorders (Dolan-Sewell et al., 2001; Gunderson, Triebwasser, Phillips, & Sullivan, 1999; Klein, Wonderlich, & Shea, 1993; Reich, 2003; Widiger & Seidlitz, 2002). For instance, one study showed that individuals with comorbid PDs responded less well to cognitive treatment for depression than individuals without PD pathology did (Fournier et al., 2008). Cluster C personality traits have also been related to poor treatment outcome in depressed patients (Hardy et al., 1995) and in those with obsessive–compulsive symptoms (Cavedini, Erzegovesi, Ronchi, & Bellodi, 1997). A diagnosis of borderline PD has also been linked with less improvement from treatment in patients with PTSD (Feeny, Zoellner, & Foa, 2002) and depression (Goodman, Hull, Clarkin, & Yeomans, 1998; Meyer, Pilkonis, Proietti, Heape, & Egan, 2001). However, reviews of the available research have shown that support for the negative effect of PDs on depression (Mulder, 2002) and anxiety (Dreessen & Arntz, 1998) is mixed at best.

Finally, the spectrum model would suggest that both PDs and Axis I disorders lie on the same dimensions, ranging from more to less severe expressions of psychopathology. The comorbidity between certain combinations of PDs and Axis I disorders have led to various proposals for spectrums of psychopathology. For instance, it has been suggested that Cluster A PDs and Axis I psychotic disorders (e.g., schizophrenia) all lie on a continuum of severity (Tyrer, Gunderson, Lyons, & Tohen, 1997). As evidence in support of this idea, research has shown that that schizophrenia is genetically related to paranoid and schizotypal PDs (Kendler, McGuire, et al., 1993). Some have suggested eliminating PDs from Axis II of DSM and instead replacing these concepts with early-onset, chronic variants of Axis I disorders (First et al., 2002; Siever & Davis, 1991). In an example of research examining the feasibility of the spectrum concept, Klein and Schwartz (2002) compared different models of the relationship between depressive symptoms in dysthymic disorder and symptoms of borderline PD over time. They concluded that the best-fitting model to explain the data was a spectrum model, with a fixed common factor underlying the two disorders and specific influences unique to each.

Clark (2005) has suggested that the interplay between the genetically influenced dimensions of temperament is what links personality and psychopathology. "Temperament" is often defined as core, biologically based individual differences in predispositions or tendencies toward emotional, motor, and attentional activity and self-regulation (Rothbart & Bates, 1998). These tendencies are apparent very early in life and form the basis for the development of later personality, which is assumed to include a greater differentiation of individual characteristics (e.g., Caspi, Roberts, & Shiner, 2005). Three major temperament dimensions are identifiable from childhood to adulthood: positive activation (or positive emotionality/affectivity), negative activation (negative emotionality/affectivity), and disinhibition (vs. constraint). The temperament dimensions of neuroticism/negative affectivity and extraversion/positive affectivity have been linked to internalizing disorders (e.g., depression, anxiety), while the third dimension of disinhibition/constraint has been linked to externalizing psychopathology (see Clark, 2005, for a review).

Clark (2005) argues that associating personality and psychopathology through temperament, and not in a causal direction from temperament to personality to psychopathology (Hyde et al., 2008), explains the increasing stability of personality over time (Roberts & DelVecchio, 2000), the hierarchical structure of personality (Markon et al., 2005), and the way that multiple models of the personality–psychopathology link can be equally valid (e.g., both spectrum and vulnerability). In an illustrative example of this type of research, Casillas and Clark (2002) studied whether dependency, impulsivity, and self-harm (lower-order traits of the temperament/personality dimensions of negative emotionality and constraint) would explain the links between Cluster B PDs and SUDs. They found that impulsivity and self-harm were strongly related to both the PDs and SUDs. Impulsivity in particular played a strong role in the association between the two types of disorders. When impulsivity and self-harm were partialed out, this reduced the PD–SUD relation in many cases; in fact, the correlation between narcissistic PD and SUDs was no longer significant.

Evidence from Molecular Genetics

Molecular genetics is an area of research that holds great promise for the understanding of the etiology of personality and psychopathology, particularly for understanding the interplay between these phenotypes. Certainly the field of molecular genetics has not delivered as quickly on the promise of discovery as originally had been hoped (e.g., Plomin & Crabbe, 2000), although the expectations were admittedly quite high. Attempts to replicate early findings on links between specific genes and normal personality traits have been frustratingly inconsistent (see South & Krueger, 2008a, for a review). However, recent findings provide intriguing evidence for new ways of identifying specific genes that may explain, at least in some small part, the variance in personality and psychopathology. First, measured-gene–measured-environment interactions have now been found for both psychopathology (see Caspi et al., 2002, 2003) and personality traits (Keltikangas-Jarvinen, Raikkonen, Ekelund, & Peltonen, 2004). In these studies, the combination of a specific stressor (e.g., childhood rearing environment) and the presence of a specific gene was manifested in the expression of the phenotypic personality trait or psychopathological syndrome. Second, building on the spectrum model of externalizing discussed above, Dick and colleagues (2008) found an association between variations in the CHRM2 gene and a latent factor of externalizing behavior (composed of SUDs, conduct disorder, antisocial PD, and the personality traits of novelty seeking and sensation seeking). This finding would support the conclusion that normal personality, pathological personality, and psychopathological syndromes are part of one spectrum that is due, at least in small part, to genetic influences.

Conclusions

In this chapter, we have reviewed evidence for the four models of interplay between personality and psychopathology, as they apply to (1) Axis I psychopathology and normal personality; (2) Axis II PD psychopathology and normal personality; and (3) Axis I clinical disorders and Axis II PDs. It is certainly still too early to draw a definitive conclusion about which model best fits the data for each type of personality–psychopathology relationship. There is consistent evidence, however, that multivariate, integrative, hierarchical models do provide important information about the relationships between personality and psychopathology. The research reviewed here has consistently supported the value of a higher-order structure in the conceptualization of normal personality traits, pathological personality traits, and clinical disorders. As we have noted, the structure of normal personality seems best accounted for by five factors or domains (neuroticism, extraversion, agreeableness, openness, and conscientiousness); the structure of pathological personality traits by four higher-order factors (e.g., the DAPP-BQ factors of emotional dysfunction, dissocial behavior, inhibitedness, and compulsivity); and the structure of common forms of psychopathology by two factors (internalizing and externalizing).

A promising area of future research, which has already begun, is to articulate how these three higher-order structures fit together. PD symptoms are already well represented by the FFM (Lynam & Widiger, 2001); in turn, normal personality traits (e.g., neuroticism, constraint) have been included in the internalizing–externalizing model of psychopathology (e.g., Krueger et al., 2002; South & Krueger, 2008b). It may be possible to construct a metamodel that incorporates aspects of normal personality, pathological personality, and Axis I psychopathology, which may all be variants of core temperament dimensions (e.g., Clark, 2005). Such a metastructure could provide key information about which types of behaviors are likely to occur together. Even more informative would be to examine this personality–psychopathology metamodel over time. Longitudinal research is probably the best approach to understanding the development of personality and psychopathology. Much work is still necessary to uncover how core temperament dimensions may be manifested as different personality or psychopathology presentations. Behavior and molecular genetic methods will be particularly helpful in determining how environmental influences interact with genetic dispositions in the development of personality and psycho-

pathology. Undoubtedly, not only do environmental factors shape the development of personality and psychopathology, but genetically influenced personality and psychopathology have an impact on an individual's environment, thus creating a dynamic, fluctuating interaction between the person and his or her world.

References

Abramson, L. Y., Metalsky, G., & Alloy, L. (1989). Hopelessness depression: A theory-based subtype of depression. *Psychological Review*, *96*, 358–372.

Alden, L. E., Laposa, J. M., Taylor, C. T., & Ryder, A. G. (2002). Avoidant personality disorder: Current status and future directions. *Journal of Personality Disorders*, *16*, 1–29.

Alloy, L. B., Abramson, L. Y., Whitehouse, W. G., Hogan, M. E., Panzarella, C., & Rose, D. T. (2006). Prospective incidence of first onsets and recurrences of depression in individuals at high and low cognitive risk for depression. *Journal of Abnormal Psychology*, *115*, 145–156.

American Psychiatric Association (APA). (1952). *Diagnostic and statistical manual of mental disorders*. Washington, DC: Author.

American Psychiatric Association (APA). (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

American Psychiatric Association (APA). (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

American Psychiatric Association (APA. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.

Axelrod, S. R., Widiger, T. A., Trull, T. J., & Corbitt, E. M. (1997). Relations of five-factor model antagonism facets with personality disorder symptomatology. *Journal of Personality Assessment*, *67*, 297–313.

Bagby, R. M., Joffe, R. T., Parker, J. D. A., Kalemba, V., & Harkness, K. L. (1995). Major depression and the five-factor model of personality. *Journal of Personality Disorders*, *9*, 224–234.

Ball, S. A., Tennen, H., Poling, J. C., Kranzler, H. R., & Rounsaville, B. J. (1997). Personality, temperament, and character dimensions and the DSM-IV personality disorders in substance abusers. *Journal of Abnormal Psychology*, *106*, 545–553.

Beevers, C. G., Rohde, P., Stice, E., & Nolen-Hoeksema, S. (2007). Recovery from major depressive disorder among female adolescents: A prospective test of the scar hypothesis. *Journal of Consulting and Clinical Psychology*, *75*, 888–900.

Block, J. (1995). A contrarian view of the five-factor approach to personality description. *Psychological Bulletin*, *117*, 187–215.

Bornstein, R. F. (1998). Reconceptualizing personality disorder diagnosis in the DSM-V: The discriminant validity challenge. *Clinical Psychology: Science and Practice*, *5*, 333–343.

Boyce, P., Parker, G., Barnett, B., Cooney, M., & Smith, F. (1991). Personality as a vulnerability factor for depression. *British Journal of Psychiatry*, *159*, 106–114.

Butcher, J. N., Dahlstrom, W. G., Graham, J. R., Tellegen, A., & Kaemmer, B. (1989). *MMPI-2: Manual for administering and scoring.* Minneapolis: University of Minnesota Press.

Button, T. M. M., Corley, R. P., Rhee, S. H., Hewitt, J. K., Young, S. E., & Stallings, M. C. (2007). Delinquent peer affiliation and conduct problems: A twin study. *Journal of Abnormal Psychology*, *116*, 554–564.

Casillas, A., & Clark, L. A. (2002). Dependency, impulsivity, and self-harm: Traits hypothesized to underlie the association between Cluster B personality and substance use disorders. *Journal of Personality Disorders*, *16*, 424–436.

Caspi, A., McClay, J., Moffitt, T., Mill, J., Martin, J., Craig, I. W., et al. (2002). Role of genotype in the cycle of violence in maltreated children. *Science*, *297*, 752–754.

Caspi, A., Roberts, B. W., & Shiner, R. L. (2005). Personality development: Stability and change. *Annual Review of Psychology*, *56*, 453–484.

Caspi, A., Sugden, K., Moffitt, T., Taylor, A., Craig, I. W., Harrington, H., et al. (2003). Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. *Science*, *301*, 386–389.

Cavedini, P., Erzegovesi, S., Ronchi, P., & Bellodi, L. (1997). Predictive value of obsessive–compulsive personality disorder in antiobsessional pharmacological treatment. *European Neuropsychopharmacology*, *7*, 45–49.

Chambless, D. L., Fydrich, T., & Rodebaugh, T. L. (2008). Generalized social phobia and avoidant personality disorder: Meaningful distinction or useless duplication? *Depression and Anxiety*, *25*, 8–19.

Clark, L. A. (1993). *Manual for the Schedule for Nonadaptive and Adaptive Personality (SNAP)*. Minneapolis: University of Minnesota Press.

Clark, L. A. (2005). Temperament as a unifying basis for personality and psychopathology. *Journal of Abnormal Psychology*, *114*, 505–521.

Clark, L. A., & Livesley, W. J. (2002). Two approaches to identifying the dimensions of personality disorder: Convergence on the five-factor model. In P. T. Costa & T. A. Widiger (Eds.), *Personality disorders and the five-factor model of personality* (2nd ed., pp. 161–176). Washington, DC: American Psychological Association.

Clark, L. A., Livesley, W. J., & Morey, L. C. (1997). Personality disorder assessment: The challenge of construct validity. *Journal of Personality Disorders*, *11*, 205–231.

Clark, L. A., Livesley, W. J., Schroeder, M. L., & Irish, S. L. (1996). Convergence of two systems for assessing specific traits of personality disorder. *Psychological Assessment*, *8*, 294–303.

Clark, L. A., Vittengl, J., Kraft, D., & Jarrett, R. B. (2003). Separate personality traits from states to predict depression. *Journal of Personality Disorders*, *17*, 152–172.

Clark, L. A., & Watson, D. (1999a). Personality, disorder, and personality disorder: Towards a more rational conceptualization. *Journal of Personality Disorders*, *13*, 142–151.

Clark, L. A., & Watson, D. (1999b). Temperament: A new paradigm for trait psychology. In L. A. Pervin & O. P. John (Eds.), *Handbook of personality: Theory and research* (2nd ed., pp. 399–423). New York: Guilford Press.

Clark, L. A., Watson, D., & Mineka, S. (1994). Temperament, personality, and the mood and anxiety disorders. *Journal of Abnormal Psychology*, *103*, 103–116.

Cloninger, C. R. (1987). A systematic method for clinical description and classification of personality variants. *Archives of General Psychiatry*, *44*, 573–588.

Cloninger, C. R., Svrakic, D. M., & Przybeck, T. R. (1993). A psychobiological model of temperament and character. *Archives of General Psychiatry*, *50*, 975–990.

Cole, D. A., Ciesla, J. A., Dallaire, D. H., Jacquez, F. M., Pineda, A. Q., LaGrange, B., et al. (2008). Emergence of attributional style and its relation to depressive symptoms. *Journal of Abnormal Psychology*, *117*, 16–31.

Coolidge, F. L., Becker, L. A., DiRito, D. C., Durham, R. L., Kinlaw, M. M., & Philbrick, P. B. (1994). On the relationship of the five-factor personality model to personality disorders: Four reservations. *Psychological Reports*, *75*, 11–21.

Costa, P. T., & McCrae, R. R. (1985). *The NEO Personality Inventory manual*. Odessa, FL: Psychological Assessment Resources.

Costa, P. T., Jr., & McCrae, R. R. (1990). Personality disorders and the five-factor model of personality. *Journal of Personality Disorders*, *4*, 362–371.

Costa, P. T., & McCrae, R. R. (1992). *Revised NEO Personality Inventory (NEO-PI-R) and NEO Five-Factor Inventory (NEO-FFI) professional manual*. Odessa, FL: Psychological Assessment Resources.

Costa, P. T., & McCrae, R. R. (1994). Set like plaster?: Evidence for the stability of adult personality. In T. F. Heatherton & J. L. Weinberger (Eds.), *Can personality change?* (pp. 21–40). Washington, DC: American Psychological Association.

DeRaad, B., Perugini, M., Hrebiekova, M., & Szarota, P. (1998). Lingua franca of personality: Taxonomies and structures based on the psycholexical approach. *Journal of Cross-Cultural Psychology*, *29*, 212–232.

Dick, D. M., Aliev, F., Wang, J. C., Grucza, R. A., Schuckit, M., Kuperman, S., et al. (2008). Using dimensional models of externalizing psychopathology to aid in gene identification. *Archives of General Psychiatry*, *65*, 310–318.

Dick, D. M., Viken, R., Purcell, S., Kaprio, J., Pulkkinen, L., & Rose, R. J. (2007). Parental monitoring moderates the importance of genetic and environmental influences on adolescent smoking. *Journal of Abnormal Psychology*, *116*, 213–218.

Digman, J. M. (1997). Higher-order factors of the Big Five. *Journal of Personality and Social Psychology*, *73*, 1246–1256.

Dolan-Sewell, R. T., Krueger, R. F., & Shea, M. T. (2001). Co-occurrence with syndrome disorders. In W. J. Livesley (Ed.), *Handbook of personality disorders: Theory, research, and treatment* (pp. 84–106). New York: Guilford Press.

Dreessen, L., & Arntz, A. (1998). The impact of personality disorders on treatment outcome of anxiety disorders: Best-evidence synthesis. *Behaviour Research and Therapy*, *36*, 483–504.

Duberstein, P. R., Palsson, S. P., Waern, M., & Skoog, I. (2008). Personality and risk for depression in a birth cohort of 70-year-olds fol-

lowed for 15 years. *Psychological Medicine*, *38*, 663–671.

Duggan, C. F., Lee, A. S., & Murray, R. M. (1990). Does personality predict long-term outcome in depression? *British Journal of Psychiatry*, *157*, 19–24.

Dyce, J. A., & O'Connor, B. P. (1998). Personality disorders and the five-factor model: A test of facet-level predictions. *Journal of Personality Disorders*, *12*, 31–45.

Eysenck, H. J. (1994). Normality–abnormality and the three-factor model of personality. In S. Strack & M. Lorr (Eds.), *Differentiating normal and abnormal personality* (pp. 3–25). New York: Springer.

Eysenck, H. J. (1997). Personality and experimental psychology: The unification of psychology and the possibility of a paradigm. *Journal of Personality and Social Psychology*, *73*, 1224–1237.

Eysenck, H. J., & Eysenck, S. B. G. (1975). *Manual of the Eysenck Personality Questionnaire*. London: Hodder & Stoughton.

Eysenck, S. B. G., Eysenck, H. J., & Barrett, P. (1985). A revised version of the Psychoticism scale. *Personality and Individual Differences*, *6*, 21–29.

Fabrega, H., Ulrich, R., Pilkonis, P., & Mezzich, J. E. (1992). Pure personality disorders in an intake psychiatric setting. *Journal of Personality Disorders*, *6*, 153–161.

Feeny, N. C., Zoellner, L. A., & Foa, E. B. (2002). Treatment outcome for chronic PTSD among female assault victims with borderline personality characteristics: A preliminary examination. *Journal of Personality Disorders*, *16*, 30–40.

Feinberg, M. E., Button, T. M. M., Neiderhiser, J. M., Reiss, D., & Heatherington, E. M. (2007). Parenting and adolescent antisocial behavior and depression: Evidence of genotype × parenting environment interaction. *Archives of General Psychiatry*, *64*, 457–465.

First, M. B., Bell, C. B., Cuthbert, B., Krystal, J. H., Malison, R., Offord, D. R., et al. (2002). Personality disorders and relational disorders: A research agenda for addressing crucial gaps in DSM. In D. J. Kupfer, M. B. First, & D. A. Regier (Eds.), *A research agenda for DSM-V* (pp. 123–199). Washington, DC: American Psychiatric Association.

Fournier, J. C., DeRubeis, R. J., Shelton, R. C., Gallop, R., Amsterdam, J., & Hollon, S. D. (2008). Antidepressant medications v. cognitive therapy in people with depression with or without personality disorder. *British Journal of Psychiatry*, *192*, 124–129.

Frances, A., & Widiger, T. A. (1986). Methodological issues in personality disorder diagnosis. In T. Millon & G. L. Klerman (Eds.), *Contemporary directions in psychopathology: Toward the DSM-IV* (pp. 381–400). New York: Guilford Press.

Goldberg, L. R. (1990). An alternative "description of personality": The Big-Five-factor structure. *Journal of Personality and Social Psychology*, *59*, 1216–1229.

Goldberg, L. R. (1993). The structure of phenotypic personality traits. *American Psychologist*, *48*, 26–34.

Goodman, G., Hull, J. W., Clarkin, J. F., & Yeomans, F. E. (1998). Comorbid mood disorders as modifiers of treatment response among inpatients with borderline personality disorder. *Journal of Nervous and Mental Disease*, *186*, 616–622.

Goodwin, R. D., & Gotlib, I. H. (2004). Gender differences in depression: The role of personality factors. *Psychiatry Research*, *126*, 135–142.

Gunderson, J. G., Triebwasser, J., Phillips, K. A., & Sullivan, C. N. (1999). Personality and vulnerability to affective disorders. In C. R. Cloninger (Ed.), *Personality and psychopathology* (pp. 3–32). Washington, DC: American Psychiatric Association.

Haigler, E. D., & Widiger, T. A. (2001). Experimental manipulation of NEO PI-R items. *Journal of Personality Assessment*, *77*, 339–358.

Hardy, G. E., Barkham, M., Shapiro, D. A., Stiles, W. B., Rees, A., & Reynolds, S. (1995). Impact of Cluster C personality disorders on outcomes of contrasting brief psychotherapies for depression. *Journal of Consulting and Clinical Psychology*, *63*, 997–1004.

Hettema, J. M., Neale, M. C., Myers, J. M., Prescott, C., & Kendler, K. S. (2006). A population-based twin study of the relationship between neuroticism and internalizing disorders. *American Journal of Psychiatry*, *163*, 857–864.

Hyde, J. S., Mezulis, A. H., & Abramson, L. Y. (2008). The ABCs of depression: Integrating affective, biological, and cognitive models to explain the emergence of the gender difference in depression. *Psychological Review*, *115*, 291–313.

Jang, K. L., Livesley, W. J., & Vernon, P. A. (1998). A twin study of genetic and environmental contributions to gender differences in

traits delineating personality disorder. *European Journal of Personality*, *12*, 331–344.

John, O. P., Donahue, E. M., & Kentle, R. L. (1991). *The Big Five Inventory—Versions 4a and 54*. Berkeley: University of California, Institute of Personality and Social Research.

John, O. P., & Srivastava, S. (1999). The Big Five trait taxonomy: History, measurement, and theoretical perspectives. In L. A. Pervin & O. P. John (Eds.), *Handbook of personality: Theory and research* (2nd ed., pp. 102–138). New York: Guilford Press.

Katon, W., Russo, J., Frank, E., Barrett, J. A., Williams, J. W., Oxman, T., et al. (2002). Predictors of nonresponse to treatment in primary care patients with dysthymia. *General Hospital Psychiatry*, *24*, 20–27.

Keltikangas-Jarvinen, L., Raikkonen, K., Ekelund, J., & Peltonen, L. (2004). Nature vs. nurture in novelty seeking. *Molecular Psychiatry*, *9*, 308–311.

Kendler, K. S., & Baker, J. H. (2007). Genetic influences on measures of the environment: A systematic review. *Psychological Medicine*, *37*, 615–626.

Kendler, K. S., Gatz, M., Gardner, C. O., & Pedersen, N. L. (2006). Personality and major depression: A Swedish longitudinal, population-based study. *Archives of General Psychiatry*, *63*, 1113–1120.

Kendler, K. S., Kessler, R. C., Neale, M. C., Heath, A. C., & Eaves, L. J. (1993). The prediction of major depression in women: Toward an integrated etiologic model. *American Journal of Psychiatry*, *150*, 1139–1148.

Kendler, K. S., McGuire, M., Gruenberg, A. M., O'Hare, A., Spellman, M., & Walsh, D. (1993). The Roscommon Family Study: III. Schizophrenia-related personality disorders in relatives. *Archives of General Psychiatry*, *50*, 781–788.

Kendler, K. S., Neale, M. C., Kessler, R. C., Heath, A. C., & Eaves, L. J. (1993). A longitudinal twin study of personality and major depression in women. *Archives of General Psychiatry*, *50*, 853–862.

Kendler, K. S., Prescott, C. A., Myers, J., & Neale, M. C. (2003). The structure of genetic and environmental risk factors for common psychiatric and substance use disorders in men and women. *Archives of General Psychiatry*, *60*, 929–937.

Klein, D. N., & Schwartz, J. E. (2002). The relation between depressive symptoms and borderline personality disorder features over time in dysthymic disorder. *Journal of Personality Disorders*, *16*, 523–535.

Klein, M. H., Wonderlich, S., & Shea, M. T. (1993). Models of relationship between personality and depression: Toward a framework for theory and research. In M. H. Klein, D. J. Kupfer, & M. T. Shea (Eds.), *Personality and depression* (pp. 1–54). New York: Guilford Press.

Krabbendam, L., Janssen, I., Bak, M., Bijl, R. V., de Graaf, R., & van Os, J. (2002). Neuroticism and low self-esteem as risk factors for psychosis. *Social Psychiatry and Psychiatric Epidemiology*, *37*, 1–6.

Krueger, R. F. (1999). The structure of common mental disorders. *Archives of General Psychiatry*, *56*, 921–926.

Krueger, R. F. (2005). Continuity of Axes I and II: Toward a unified model of personality, personality disorders, and clinical disorders. *Journal of Personality Disorders*, *19*, 233–261.

Krueger, R. F., Caspi, A., Moffitt, T. E., Silva, P. A., & McGee, R. (1996). Personality traits are differentially linked to mental disorders: A multitrait–multidiagnosis study of an adolescent birth cohort. *Journal of Abnormal Psychology*, *105*, 299–312.

Krueger, R. F., Hicks, B. M., Patrick, C. J., Carlson, S. R., Iacono, W. G., & McGue, M. (2002). Etiologic connections among substance dependence, antisocial behavior, and personality: Modeling the externalizing spectrum. *Journal of Abnormal Psychology*, *111*, 411–424.

Krueger, R. F., & Markon, K. E. (2006). Reinterpreting comorbidity: A model-based approach to understanding and classifying psychopathology. *Annual Review of Clinical Psychology*, *2*, 111–133.

Krueger, R. F., & Tackett, J. L. (2003). Personality and psychopathology: Working toward the bigger picture. *Journal of Personality Disorders*, *17*, 109–128.

Lau, J. Y. F., & Eley, T. C. (2008). Disentangling gene–environment correlations and interactions on adolescent depressive symptoms. *Journal of Child Psychology and Psychiatry*, *49*, 142–150.

Lilienfeld, S. O., Waldman, I. D., & Israel, A. C. (1994). A critical examination of the use of the term "comorbidity" in psychopathology research. *Clinical Psychology: Science and Practice*, *1*, 71–83.

Livesley, W. J. (2001a). Commentary on recon-

ceptualizing personality disorder categories using trait dimensions. *Journal of Personality*, *69*, 277–286.

Livesley, W. J. (2001b). Conceptual and taxonomic issues. In W. J. Livesley (Ed.), *Handbook of personality disorders: Theory, research, and treatment* (pp. 3–38). New York: Guilford Press.

Livesley, W. J. (2003). Diagnostic dilemmas in classifying personality disorder. In K. A. Phillips, M. B. First, & H. A. Pincus (Eds.), *Advancing DSM: Dilemmas in psychiatric diagnosis* (pp. 153–190). Washington, DC: American Psychiatric Association.

Livesley, W. J., & Jackson, D. N. (2002). *Manual for the Dimensional Assessment of Personality Pathology—Basic Questionnaire (DAPP)*. London, ON, Canada: Research Psychologists' Press.

Livesley, W. J., & Jackson, D. (in press). *Manual for the Dimensional Assessment of Personality Pathology—Basic Questionnaire*. Port Huron, MI: Sigma Press.

Livesley, W. J., & Jang, K. L. (2008). The behavioral genetics of personality disorder. *Annual Review of Clinical Psychology*, *4*, 247–274.

Livesley, W. J., Jang, K. L., Jackson, D. N., & Vernon, P. A. (1993). Genetic and environmental contributions to dimensions of personality disorder. *American Journal of Psychiatry*, *150*, 1826–1831.

Livesley, W. J., Jang, K. L., & Vernon, P. A. (1998). Phenotypic and genetic structure of traits delineating personality disorder. *Archives of General Psychiatry*, *55*, 941–948.

Lynam, D. R., & Widiger, T. A. (2001). Using the five-factor model to represent the DSM-IV personality disorders: An expert consensus approach. *Journal of Abnormal Psychology*, *110*, 401–412.

Malouff, J. M., Thorsteinsson, E. B., & Schutte, N. S. (2005). The relationship between the five-factor model of personality and symptoms of clinical disorders: A meta-analysis. *Journal of Psychopathology and Behavioral Assessment*, *27*, 101–114.

Markon, K. E., Krueger, R. F., & Watson, D. (2005). Delineating the structure of normal and abnormal personality: An integrative hierarchical approach. *Journal of Personality and Social Psychology*, *88*, 139–157.

Mathews, A., & MacLeod, C. (2005). Cognitive vulnerability to emotional disorders. *Annual Review of Clinical Psychology*, *1*, 167–195.

McDavid, J. D., & Pilkonis, P. A. (1996). The stability of personality disorder diagnoses. *Journal of Personality Disorders*, *10*, 1–15.

McGlashan, T. H. (1987). Borderline personality disorder and unipolar affective disorder: Long-term effects of comorbidity. *Journal of Nervous and Mental Disease*, *175*, 467–473.

Meyer, B., Pilkonis, P. A., Proietti, J. M., Heape, C. L., & Egan, M. (2001). Attachment styles and personality disorders as predictors of symptom course. *Journal of Personality Disorders*, *15*, 371–389.

Miller, J. D., Lynam, D., & Leukefeld, C. (2003). Examining antisocial behavior through the lens of the five factor model of personality. *Aggressive Behavior*, *29*(6), 497–514.

Millon, T. (1983). *Millon Clinical Multiaxial Inventory: Manual for the MCMI*. Minneapolis, MN: National Computer Systems.

Millon, T. (1987). *Millon Clinical Multiaxial Inventory II: Manual for the MCMI-II*. Minneapolis, MN: National Computer Systems.

Millon, T. (1996). *Personality and psychopathology: Building a clinical science: Selected papers of Theodore Millon*. New York: Wiley.

Millon, T. (2002). Assessment is not enough: The SPA should participate in constructing a comprehensive clinical science of personality. *Journal of Personality Assessment*, *78*, 209–218.

Millon, T. (with Davis, R.). (1996). *Disorders of personality: DSM-IV and beyond* (2nd ed.). New York: Wiley.

Millon, T. (with Millon, C., & Davis, R.). (1994). *MCMI-III manual*. Minneapolis, MN: National Computer Systems.

Moffitt, T. E., Caspi, A., Dickson, N., Silva, P., & Stanton, W. (1996). Childhood-onset versus adolescent-onset antisocial conduct problems in males: Natural history from ages 3 to 18 years. *Development and Psychopathology*, *8*, 399–424.

Morey, L. C., Gunderson, J. G., Quigley, B. D., Shea, M. T., Skodol, A. E., McGlashan, T. H., et al. (2002). The representation of borderline, avoidant, obsessive–compulsive, and schizotypal personality disorders by the five-factor model. *Journal of Personality Disorders*, *16*, 215–234.

Mulder, R. T. (2002). Personality pathology and treatment outcome in major depression: A review. *American Journal of Psychiatry*, *159*, 359–371.

Mullins-Sweatt, S. N., & Widiger, T. A. (2006). The five-factor model of personality disorder: A translation across science and practice. In R. F. Krueger & J. L. Tackett (Eds.), *Personality*

and psychopathology (pp. 39–70). New York: Guilford Press.

Nolen-Hoeksema, S. (2000). The role of rumination in depressive disorders and mixed anxiety/depressive symptoms. *Journal of Abnormal Psychology*, *109*, 504–511.

O'Connor, B. P. (2002). The search for dimensional structure differences between normality and abnormality: A statistical review of published data on personality and psychopathology. *Journal of Personality and Social Psychology*, *83*, 962–982.

Ormel, J., Oldehinkel, A. J., & Brilman, E. I. (2001). The interplay and etiological continuity of neuroticism, difficulties and life events in the etiology of major and subsyndromal, first and recurrent depressive episodes in later life. *American Journal of Psychiatry*, *158*, 885–891.

Ormel, J., Rosmalen, J., & Farmer, A. (2004). Neuroticism: A non-informative marker of vulnerability to psychopathology. *Social Psychiatry and Psychiatric Epidemiology*, *39*, 906–912.

Pervin, L. A., & John, O. P. (Eds.). (1999). *Handbook of personality: Theory and research* (2nd ed.). New York: Guilford Press.

Plomin, R., & Crabbe, J. (2000). DNA. *Psychological Bulletin*, *126*, 806–828.

Plomin, R., DeFries, J. C., McClearn, G. E., & McGuffin, P. (2008). *Behavioral genetics*. New York: Worth.

Purcell, S. (2002). Variance components models for gene–environment interaction in twin analysis. *Twin Research*, *5*, 554–571.

Reich, J. (2003). The effects of Axis II disorders on the outcome of treatment of anxiety and unipolar depressive disorders: A review. *Journal of Personality Disorders*, *17*, 387–405.

Reichborn-Kjennerud, T., Czajkowski, N., Neale, M. C., Ørstavik, R. E., Torgersen, S., Tambs, K., et al. (2007). Genetic and environmental influences on dimensional representations of DSM-IV Cluster C personality disorders: A population-based multivariate twin study. *Psychological Medicine*, *37*, 645–653.

Reynolds, S. K., & Clark, L. A. (2001). Predicting dimensions of personality disorder from domains and facets of the five-factor model. *Journal of Personality*, *69*, 199–222.

Roberts, B. W., & DelVecchio, W. F. (2000). The rank-order consistency of personality traits from childhood to old age: A quantitative review of longitudinal studies. *Psychological Bulletin*, *126*, 3–25.

Roberts, S. B., & Kendler, K. S. (1999). Neuroticism and self-esteem as indices of the vulnerability to major depression in women. *Psychological Medicine*, *29*, 1101–1109.

Rothbart, M. K., & Bates, J. E. (1998). Temperament. In W. Damon (Series Ed.) & N. Eisenberg (Vol. Ed.), *Handbook of child psychology: Vol. 3. Social, emotional, and personality development* (5th ed., pp. 105–176). New York: Wiley.

Rothschild, L., & Zimmerman, M. (2002). Personality disorders and the duration of depressive episode: A retrospective study. *Journal of Personality Disorders*, *16*, 293–303.

Ruiz, M. A., Pincus, A. L., & Schinka, J. (2008). Externalizing pathology and the five-factor model: A meta-analysis of personality traits, associated with antisocial personality disorder, substance use disorder, and their co-occurrence. *Journal of Personality Disorders*, *22*, 365–388.

Samuel, D. B., & Widiger, T. A. (2004). Clinicians' personality descriptions of prototypic personality disorders. *Journal of Personality Disorders*, *18*, 286–308.

Saulsman, L. M., & Page, A. C. (2004). The five-factor model and personality disorder empirical literature: A meta-analytic review. *Clinical Psychology Review*, *23*, 1055–1085.

Schroeder, M. L., Wormworth, J. A., & Livesley, W. J. (1992). Dimensions of personality disorder and their relationships to the Big Five dimensions of personality. *Psychological Assessment*, *4*, 47–53.

Scott, J., Williams, J. M. G., Brittlebank, A., & Ferrier, I. N. (1995). The relationship between premorbid neuroticism, cognitive dysfunction and persistence of depression. *Journal of Affective Disorders*, *33*, 167–172.

Sellbom, M., Ben-Porath, Y., & Bagby, R. M. (2008). Personality and psychopathology: Mapping the MMPI-2 Restructured Clinical (RC) scales onto the five factor model of personality. *Journal of Personality Disorders*, *22*, 291–312.

Shea, M. T., Stout, R. L., Gunderson, J. G., Morey, L. C., Grilo, C. M., McGlashan, T., et al. (2002). Short-term diagnostic stability of schizotypal, borderline, avoidant, and obsessive–compulsive personality disorders. *American Journal of Psychiatry*, *159*, 2036–2041.

Sher, K. J., & Trull, T. J. (1994). Personality and disinhibitory psychopathology: Alcoholism and antisocial personality disorder. *Journal of Abnormal Psychology*, *103*, 92–102.

Shiner, R. L., Masten, A. S., & Tellegen, A. (2002). A developmental perspective on personality in emerging adulthood: Childhood antecedents and concurrent adaptation. *Journal of Personality and Social Psychology*, *83*, 1165–1177.

Siever, L. J., & Davis, K. L. (1991). A psychobiological perspective on the personality disorders. *American Journal of Psychiatry*, *148*, 1647–1658.

Skodol, A. E., Oldham, J. M., & Gallagher, P. E. (1999). Axis II comorbidity of substance use disorders among patients referred for treatment of personality disorders. *American Journal of Psychiatry*, *156*, 733–738.

South, S. C., & Krueger, R. F. (2008a). An interactionist perspective on genetic and environmental contributions to personality. *Social and Personality Psychology Compass*, *2*, 929–948.

South, S. C., & Krueger, R. F. (2008b). Marital quality moderates genetic and environmental influences on the internalizing spectrum. *Journal of Abnormal Psychology*, *117*, 826–837.

South, S. C., Krueger, R. F., Johnson, W., & Iacono, W. G. (2008). Adolescent personality moderates genetic and environmental influences on relationships with parents. *Journal of Personality and Social Psychology*, *94*, 899–912.

Surtees, P. G., & Wainwright, N. W. J. (1996). Fragile states of mind: Neuroticism, vulnerability and the long-term outcome of depression. *British Journal of Psychiatry*, *169*, 338–347.

Tackett, J. L. (2006). Evaluating models of the personality–psychopathology relationship in children and adolescents. *Clinical Psychology Review*, *26*, 584–599.

Tellegen, A. (1985). *Brief manual for the Multidimensional Personality Questionnaire*. Unpublished manuscript, University of Minnesota.

Tellegen, A., Ben-Porath, Y. S., McNulty, J. L., Arbisi, P. A., Graham, J. R., & Kaemmer, B. (2003). *MMPI-2 Restructured Clinical (RC) scales: Development, validation, and interpretation*. Minneapolis: University of Minnesota Press.

Tellegen, A., & Waller, N. G. (in press). *Exploring personality through test construction: Development of the Multidimensional Personality Questionnaire (MPQ)*. Minneapolis: University of Minnesota Press.

Torgersen, S., Lygren, S., Øien, P. A., Skre, I., Onstad, S., Edvardsen, J., et al. (2000). A twin study of disorders. *Comprehensive Psychiatry*, *41*(6), 416–425.

Trull, T. J. (1992). DSM-III-R personality disorders and the five-factor model of personality: An empirical comparison. *Journal of Abnormal Psychology*, *101*, 553–560.

Trull, T. J., & Sher, K. J. (1994). Relationship between the five-factor model of personality and Axis I disorders in a nonclinical sample. *Journal of Abnormal Psychology*, *103*, 350–360.

Trull, T. J., Sher, K. J., Minks-Brown, C., Durbin, J., & Burr, R. (2000). Borderline personality disorder and substance use disorders: A review and integration. *Clinical Psychology Review*, *20*, 235–253.

Trull, T. J., Waudby, C. J., & Sher, K. J. (2004). Alcohol, tobacco, and drug use disorders and personality disorder symptoms. *Experimental and Clinical Psychopharmacology*, *12*, 65–75.

Trull, T. J., Widiger, T. A., Useda, J. D., Holcomb, J., Doan, D.-I., Axelrod, S. R., et al. (1998). A structured interview for the assessment of the five-factor model of personality. *Psychological Assessment*, *10*, 229–240.

Turkheimer, E. (1998). Heritability and biological explanation. *Psychological Review*, *105*, 782–791.

Tyrer, P., Gunderson, J., Lyons, M., & Tohen, M. (1997). Special feature: Extent of comorbidity between mental state and personality disorders. *Journal of Personality Disorders*, *11*, 242–259.

van Os J., & Jones, P. B. (2001). Neuroticism as a risk factor for schizophrenia. *Psychologial Medicine*, *31*, 1129–1134.

Warner, M. B., Morey, L. C., Finch, J. F., Gunderson, J. G., Skodol, A. E., Sanislow, C. A., et al. (2004). The longitudinal relationship of personality traits and disorders. *Journal of Abnormal Psychology*, *113*, 217–227.

Watson, D., & Clark, L. A. (1995). Depression and the melancholic temperament. *European Journal of Personality*, *9*, 351–366.

Watson, D., Kotov, R., & Gamez, W. (2006). Basic dimensions of temperament in relation to personality and psychopathology. In R. F. Krueger & J. L. Tackett (Eds.), *Personality and psychopathology* (pp. 7–38). New York: Guilford Press.

Widiger, T. A., & Costa, P. T., Jr. (2002). Five-factor model personality disorder research. In P. T. Costa, Jr. & T. A. Widiger (Eds.), *Personality disorders and the five-factor model of*

personality (2nd ed., pp. 59–88). Washington, DC: American Psychological Association.

Widiger, T. A., & Seidlitz, L. (2002). Personality, psychopathology, and aging. *Journal of Research in Personality*, *36*, 335–362.

Widiger, T. A., & Simonsen, E. (2005). Alternative dimensional models of personality disorder: Finding a common ground. *Journal of Personality Disorders*, *19*, 110–130.

Widiger, T. A., & Trull, T. J. (2007). Plate tectonics in the classification of personality disorder: Shifting to a dimensional model. *American Psychologist*, *62*, 71–83.

Widiger, T. A., Trull, T. J., Clarkin, J. F., Sanderson, C., & Costa, P. T., Jr. (1994). A description of the DSM-III-R and DSM-IV personality disorders with the five-factor model of personality. In P. T. Costa, Jr. & T. A. Widiger (Eds.), *Personality disorders and the five-factor model of personality* (pp. 41–56). Washington, DC: American Psychological Association.

Widiger, T. A., Trull, T. J., Clarkin, J. F., Sanderson, C., & Costa, P. T., Jr. (2002). A description of the DSM-IV personality disorders with the five-factor model of personality. In P. T. Costa, Jr. & T. A. Widiger (Eds.), *Personality disorders and the five-factor model of personality* (2nd ed., pp. 89–99). Washington, DC: American Psychological Association.

Widiger, T. A., Verheul, R., & van den Brink, W. (1999). Personality and psychopathology. In L. A. Pervin & O. P. Johns (Eds.), *Handbook of personality: Theory and research* (2nd ed., pp. 347–366). New York: Guilford Press.

Wiggins, J. S., & Pincus, A. L. (1989). Conceptions of personality disorders and dimensions of personality. *Psychological Assessment*, *1*, 305–316.

Young, S. E., Stallings, M. C., Corley, R. P., Krauter, K. S., & Hewitt, J. K. (2000). Genetic and environmental influences on behavioral disinhibition. *American Journal of Medical Genetics (Neuropsychiatric Genetics)*, *96*, 684–695.

Zanarini, M. C., Frankenburg, F. R., Dubo, E. D., Sickel, A. E., Trikha, A., Levin, A., et al. (1998). Axis I comorbidity of borderline personality disorder. *American Journal of Psychiatry*, *155*, 1733–1739.

Zweig-Frank, H., & Paris, J. (1995). The five-factor model of personality in borderline and nonborderline personality disorders. *Canadian Journal of Psychiatry*, *40*, 523–526.

CHAPTER 13

Is It True That Mental Disorders Are So Common, and So Commonly Co-Occur?

MARIO MAJ

The World Health Organization (WHO, 2001) estimates that more than 25% of individuals worldwide develop one or more mental disorders during their lifetimes. This estimate has been recently supported by the World Mental Health Surveys (Kessler et al., 2007), a series of face-to-face community surveys conducted in 17 countries in Africa, Asia, the Americas, Europe, and the Middle East, with a total number of 85,052 respondents. The lifetime prevalence of any mental disorder was found to be higher than 25% in 11 countries (with the highest rates in the United States, 47.4%; New Zealand, 39.3%; Colombia, 39.1%; France, 37.9%; and Ukraine, 36.1%), and higher than 15% in all countries but two (China and Nigeria). Anxiety disorders were the most prevalent disorders in 10 out of 17 countries (with the highest rates in the United States, 31.0%; Colombia, 25.3%; New Zealand, 24.6%; and France, 22.3%), and mood disorders in 6 (with the highest rates in the United States, 21.4%; France, 21.0%; New Zealand, 20.4%; and the Netherlands, 17.9%).

The WHO (2001) also reports that it is common for an individual to have two or more mental disorders in his or her lifetime. This notion is supported by several population studies. For instance, in the U.S. National Comorbidity Survey (NCS), 79% of people with a psychiatric diagnosis had a lifetime history of two or more mental disorders (Kessler et al., 1994), and 51% of patients with a current DSM-III-R diagnosis of major depression had at least one concomitant ("comorbid") anxiety disorder (Kessler et al., 1996).

The evidence concerning both the common occurrence of mental disorders (particularly anxiety and mood disorders), and the frequent concomitance of two or more mental disorders in the same individual, is now regarded as consolidated and has represented the basis for several public health policy recommendations. However, this evidence is unfortunately somewhat weak, in that it reflects two serious current problems of diagnosis in psychiatry: (1) the difficulty of differentiating between mental disorders and homeostatic reactions to adverse life events, (2) the questionable validity of the concept of "psychiatric comorbidity." The present chapter deals briefly with these two problems.

Differentiating between Mental Disorders and Homeostatic Reactions to Adverse Life Events

"Determining when relatively common experiences such as anxiety or sadness . . . should be considered evidence of some disorder requires the setting of boundaries that are largely arbitrary, not scientific, unlike setting the boundaries for what constitutes cancer or pneumonia." "However unpleasant or painful your experiences, you intuitively reject the idea that these common hassles are truly signs of mental disorder." These are two representative statements taken from a vitriolic book entitled *Making Us Crazy: DSM: The Psychiatric Bible and the Creation of Mental Disorders* (Kutchins & Kirk, 1997). To the average psychiatrist, these statements will probably appear incorrect and ideologically biased. He or she has learned to believe that the current systems for the diagnosis and classification of mental disorders have received convincing empirical validation, and that these systems reliably differentiate between true mental disorders and common hassles of everyday life.

"It is not always easy to establish a threshold for a mental disorder, particularly in light of how common symptoms of mental distress are and the lack of objective, physical symptoms." This is a statement by the U.S. Surgeon General (U.S. Department of Health and Human Services, 1999). In this case, ignorance and ideological bias are not likely to be involved. However, this view from outside the psychiatric field may still reflect to some degree a prejudice against the profession.

"Based on the high prevalence rates identified in both the ECA [Epidemiologic Catchment Area study] and the NCS, it is reasonable to hypothesize that some syndromes in the community represent transient homeostatic responses to internal or external stimuli that do not represent true psychopathologic disorders" (Regier et al., 1998). "The criteria diagnosed many individuals who were exhibiting normal reactions to a difficult environment as having a mental disorder" (Spitzer & Wakefield, 1999). This time the statements come from within the psychiatric field, and from very authoritative sources; therefore, they will probably convince the average psychiatrist that the problem is a real one. At the moment, we do not have a clear idea of how to fix the threshold for the diagnosis of some mental disorders (particularly of those with the highest prevalence in the community—i.e., depression and anxiety disorders), or of how to differentiate between these disorders and homeostatic reactions to adverse life events. We have to address this situation urgently because it may damage the credibility of psychiatry as a discipline and a profession.

Why is this problem becoming so visible only now? One contributing factor has certainly been the recent broadening of the scope of psychiatric intervention from traditional hospital settings, where the issue of whether admitted patients had a mental disorder or not was not really relevant, to community settings, where this issue is a sensitive one in many cases. A second factor has been the increased presence and influence in the mental health field of several other professions, whose members' perceptions of mental health problems are often different from those of psychiatrists. A third factor has been the higher levels of information and awareness about mental disorders among service users, families, and the public at large. A fourth factor has probably been difficulty with the translation of traditional descriptive definitions of mental disorders into current operational diagnostic criteria. Traditional definitions seemed to convey a gestalt, a structure for each disorder, whereas current operational definitions seem less able to do so. Therefore, the thresholds for the diagnosis of some mental disorders appear today more clearly arbitrary than they appeared in the past (Maj, 2007).

Yet another factor has been the evolution of psychiatric treatments. There was a time, about 30 years ago, when it seemed that the targets of psychiatric treatments could be easily defined. Two items of the so-called "neo-Kraepelinian credo," according to Blashfield (1984), were these: "Psychiatry treats people who are sick and who require treatment for mental illness," and "There is a boundary between the normal and the sick." A corollary to these two items was the statement that "depression, when carefully defined as a clinical entity, is qualitatively different from the mild episodes of depression that everyone experiences at some point

in his or her life" (Blashfield, 1984). Apparently in line with this statement was the observation that tricyclic antidepressants were active only in people who were clinically depressed. Administered to persons who were not clinically depressed, these drugs did not act as stimulants and did not alter their mood.

Today, the picture appears much less clear. It has been observed that "before various reproductive techniques . . . were developed, infertility was simply a fact of nature; now that it can be treated, it is a medical problem" (Elliott, 1999a). Something similar can be certainly stated for several categories of mental disorders. Moreover, today medicine does not only treat illnesses or disorders. There are medical interventions (e.g., the use of estrogen by postmenopausal women; cosmetic surgery in people who are not satisfied with their looks) that do not aim to treat diseases, but to enhance individuals' capacities or characteristics (Elliott, 1999b). Analogously, the concept of "cosmetic psychopharmacology" has been introduced; it refers to the use of psychotropic drugs not in order to move a person from a pathological to a normal state, but in order to move him or her from a state that is normal but unrewarded to a state that is equally normal but better rewarded (Kramer, 1993). Both the concept and the practice of cosmetic psychopharmacology raise obvious ethical issues. However, in order to be able to differentiate between clinical and cosmetic psychopharmacology, we are supposed to be able to differentiate between mental states that are normal and those that are pathological. If the boundary between the pathological and the normal is unclear, the concept of cosmetic psychopharmacology itself becomes questionable (Bjorklund, 2005).

If we acknowledge that mental suffering may be a reasonable reaction to an adverse life event or situation, is it inappropriate to try to lessen that suffering by using a drug, if that drug may be effective? The answer to this question will depend on people's views about the nature of mental suffering and its role in the human condition (Sperry & Prosen, 1998). One view will be that externally elicited, mild to moderate, time-limited depressive states have an adaptive role that has developed through the evolution of the human species (McGuire & Troisi, 1998). They may warn an individual that his or her past or present coping strategies have failed and that new strategies are needed. Psychomotor slowing and social withdrawal may remove the individual from high-cost, low-benefit social interactions, and these and other depressive symptoms and signs may elicit help from the environment (McGuire & Troisi, 1998). In this light, the experience of mental suffering is developmentally useful and even necessary. By medicalizing this condition and treating it with drugs, we may undermine the individual's coping strategies. Moreover, as Wakefield, Schmitz, First, and Horwitz (2007) have recently pointed out, "false-positive diagnoses can potentially lead to stigmatization, inappropriate care, and inflated epidemiological prevalence rates that undermine the credibility of the diagnostic system."

The opposite view, however, will be that prolonged mental suffering in itself does not promote any personal growth, and that what makes sense from a universal philosophical standpoint—explaining why mental pain has developed through the evolution of the human species—becomes unacceptable when one tries to maintain that the suffering of a specific individual in real time and space should not be alleviated because it exists for his or her own good (Elliott, 1999b). In this light, the relief of mental suffering becomes a legitimate medical purpose, exactly like the relief of physical pain. Furthermore, we cannot exclude the possibility that some states that appear today to be merely part of the human condition actually reflect a biological vulnerability that may represent a legitimate target for pharmacological intervention. This is, if you wish, the philosophical component of the issue. The scientific and clinical component will be that the efficacy of specific pharmacological interventions in states of demoralization or grief will have to be empirically documented, and that the balance between their benefits and risks will have to be found favorable.

Thus the problem of setting the boundary between some common mental disorders and homeostatic reactions to adverse life events is a real and crucial one from several different perspectives. Let's now review some possible approaches to this problem.

A first approach is the one emphasizing the importance of the context in which the

symptoms occur. DSM-III and its successors have deliberately tried to exclude the assessment of the context from the diagnostic process: "Because assessing the causal role of context is messy and likely to be unreliable in its results, the need for reliability has been taken to imply that references to context must be minimized" (Wakefield, 1997). However, the context in which the symptoms occur may be crucial for the distinction between a true mental disorder and a homeostatic reaction in general, and for the distinction between a true case of depression and an understandable reaction to a severe adverse event in particular. Wakefield (1997) has proposed that either the diagnosis of depression should be "excluded if the sadness response is caused by a real loss that is proportional in magnitude to the intensity and duration of the response," or this diagnosis should require that "despite there being no real recent loss (or only losses of minor magnitude), the individual nonetheless experiences a sufficient number and intensity of symptoms."

This approach may sound reasonable, but it has several limitations (Maj, 2008). First, the decisions of whether or not there is a causal relationship between the event and the response, and of whether the response is proportional or not to the event, would be left to the subjective judgment of the clinician, with a high risk of low reliability. Even worse, the ideological orientation of the clinician may sometimes become decisive; for example, there are psychiatrists who do believe that every psychopathological manifestation can be explained by looking at the individual's environmental conditions. Second, a large proportion of individuals with definite major depression report that the onset of their condition occurred in a context of psychosocial difficulties (Kendler, 1999). Third, although Wakefield's proposal aims to extend the current DSM-IV exclusion criterion concerning bereavement to other significant losses, some research evidence seems to suggest that even this current exclusion criterion may not be valid (Zisook, Shear, & Kendler, 2001). For instance, a study focusing on individuals who met diagnostic criteria for a major depressive episode during the first 2 months of bereavement found a high rate of response to antidepressant medication, similar to that seen in major depression without bereavement (Zisook, Shuchter, Pedrelli, Sable, & Deaciuc, 2001). Finally, even when confronted with the most severe life events, only a small minority of individuals develop major depression, raising the issue of what is meant by a "normal" reaction to stressors (Kendler, 1999).

A second approach to our boundary issue is the one emphasizing the importance of possible qualitative differences between true mental disorders and homeostatic reactions to adverse life events. The underlying idea here is that the fairly recent translation of traditional concepts of mental disorders into operational terms has sometimes involved an oversimplification of psychopathology and a reduction of complex phenomena to their least common denominators. On the one hand, "the gestalt characteristic of psychopathology, which tells us that the structure of symptoms is more than their sum, is largely ignored in modern classification systems" (Helmchen & Linden, 2000). In particular, "syndromal definition has disappeared from the diagnosis of depression" (van Praag, 1998). On the other hand, the need for specific professional skills to differentiate psychopathological manifestations from other expressions of impaired well-being has been deemphasized. Helmchen and Linden (2000) note that "the question of whether a person feels [or, even more, has felt in the past] 'sad or depressed', presented in a standardized interview by a lay interviewer with only limited psychopathological expertise, will necessarily generate many false positives." They add: "Normal forms of negative mood such as despair or sadness must not be mistaken as depressed mood, characterized by a lack of holothymia and being an emotional feeling only known to depressed persons" (Helmchen & Linden, 2000).

Indeed, in a study (Healy, 1993) in which patients with a clinical diagnosis of major depression were asked to describe in their own words their depressed mood,

> the commonest primary description was of the experience of lethargy and inability to do things, whether because of tiredness, a specific inability to summon up effort, a feeling of being inhibited or an inability to envisage the future . . . the next most common description was of a sense of detachment from the environment . . . [and] the next most common descriptor

> was of physical changes that were described in terms of feeling that the subject was coming down with a viral illness, either influenza or glandular fever, along with descriptions of aches and pains and, in particular, headaches or numbness of the head or tight bands around the head.

The experience of sadness or unhappiness did not appear in those descriptions (Healy, 1993).

Along the same line, some studies carried out in patients with severe or chronic physical illness have described features differentiating between clinical depression and demoralization. According to these studies, a depressed person has lost the ability to experience pleasure generally, whereas a demoralized individual is able to experience pleasure normally when he or she is distracted from thoughts concerning the demoralizing circumstance or event (de Figueiredo, 1993). Moreover, the demoralized person feels helpless, incompetent, and inhibited in action because of not knowing what to do; by contrast, the depressed individual has lost motivation and drive, and is unable to function even if an appropriate direction of action is clear in his or her mind (Clarke & Kissane, 2002). Finally, persons with clinical depression often report psychomotor, neurovegetative, and cognitive symptoms, which are not typically present in demoralization.

Although this second approach to our boundary issue is certainly of interest, it has two main weaknesses. The first is its limited research support; the second is the fact that it probably applies only to some forms of depression, rather than to clinical depression as a whole. Actually, some taxometric analyses of depression carried out in large clinical samples have suggested that major depression is not qualitatively distinguishable from less severe mood states (e.g., Ruscio & Ruscio, 2000), while some other investigations using taxometric methods in clinical populations have detected a latent depression taxon corresponding to "endogenous" or "nuclear" depression (e.g., Haslam & Beck, 1994).

The third approach to our boundary issue is the one assuming that the threshold for the diagnosis of common mental disorders is unavoidably arbitrary and has to be decided on pragmatic grounds (Kendell & Jablensky, 2003). It will be regarded as clinically valid if it has significant predictive implications in terms of response to treatment and clinical outcome. However, if prediction of treatment response is going to be one of our validating criteria, in the case of depression it is unlikely that the threshold for the diagnosis will be the same for all currently available treatment modalities. The threshold for response to interpersonal psychotherapy, for instance, may be different from the threshold for response to a selective serotonin reuptake inhibitor, which may be different from the threshold for response to a tricyclic antidepressant. This provides a rationale for the recent proposal of sequential stepwise treatment algorithms for people with depressive symptoms (e.g., Linden, Helmchen, Mackert, & Müller-Oerlinghausen, 1994). Should we ignore the issue of the differential diagnosis between true depression and understandable sadness, and simply apply one of these algorithms to all people presenting with depressive symptoms?

Of course, the principle that the boundary between normality and disorder can be decided only arbitrarily on pragmatic grounds seems to support the notion that this boundary "depends on time, space, cultural context, landscapes of care and the particularities of individual lives (say, any given patient's moral framework and any particular prescriber's educational background)" (Elliott, 1999b), while the proposal of sequential stepwise treatment algorithms appears in line with the statement that "one of psychiatry's ugly little secrets is that so much of what it does is still trial and error" (Bjorklund, 2005).

Thus, in conclusion, the question of whether we are able to discriminate between some common mental disorders (particularly depression) and homeostatic reactions to adverse life events (particularly normal sadness or an existential ailment) does not seem to have a positive answer at present. There are several approaches to the problem, but none of them seems to be supported by convincing empirical evidence. This should be a priority for future research because, as we have seen, the clinical, scientific, ethical, and political implications of this issue are very significant; they involve the image and the credibility of the psychiatric discipline and profession.

The Validity of the Concept of "Psychiatric Comorbidity"

Current diagnostic systems explicitly encourage the practice of diagnosing multiple psychiatric disorders in the same patient. According to the "Use of the Manual" section of DSM-IV (American Psychiatric Association, 1994), "The general convention in DSM-IV is to allow multiple diagnoses to be assigned for those presentations that meet criteria for more than one DSM-IV disorder" (p. 6). This strategy diverges from the one adopted in DSM-I and DSM-II, which encouraged the clinician to use a single, all-encompassing psychiatric diagnosis for each patient, with qualifying phrases to describe complex cases—for example, "phobic reaction, manifested by claustrophobia, with obsessive–compulsive symptoms, counting and recurring thoughts" (American Psychiatric Association, 1952; see also Pincus, Tew, & First, 2004).

Actually, the structure of current diagnostic systems, especially that of DSM-IV, is such that the vast majority of patients will qualify for multiple psychiatric diagnoses. I have recently reviewed the characteristics of current diagnostic systems—particularly those of DSM-III and its successors—that have contributed to the emergence of the phenomenon called "psychiatric comorbidity" (Maj, 2005a, 2005b).

The first of these characteristics is represented by a rule that has never been made explicit (as far as I know) in any DSM-related publication, but is mentioned by Lee Robins (1994) in one of her papers: "the rule laid down in the construction of DSM-III that the same symptom could not appear in more than one disorder." What Robins probably means is that in the construction of DSM-III, it was agreed that some symptoms regarded as characteristic of some classes of mental disorders (e.g., anxiety for anxiety disorders and delusions for psychotic disorders) could not appear in the diagnostic criteria for disorders belonging to other classes. This is most probably the reason why the symptom of anxiety does not appear in the diagnostic criteria for major depression, although it is very common in patients with this disorder. This situation obviously contributes to the frequent comorbidity between major depression and anxiety disorders, particularly panic disorder. In fact, panic attacks, as well as somatic and psychic anxiety, regularly appear in factor analyses of the depressive syndrome (Sato, Bottlender, Kleindiest, & Möller, 2005) and of the manic syndrome (Dilsaver, Chen, Shoaib, & Swann, 1999). Therefore, the extremely common comorbidity of anxiety disorders with major depression and bipolar I disorder largely reflects the fact that anxiety is an integral part of the phenomenology of both major depression and mania. Not surprisingly, Robins states: "I thought then, as I still do, that the rule was not a good one, because it deviates from practice in the rest of medicine, where many diseases share symptoms."

The second characteristic of DSM-III and its successors that has contributed to the emergence of psychiatric comorbidity as a common phenomenon has been the proliferation of diagnostic categories. If demarcations are introduced where they do not exist in nature, the probability increases that several diagnoses will have to be made for the same patient. This applies in particular to anxiety and personality disorders. It is indeed very rare to find a patient with a DSM-IV anxiety disorder who does not also fulfill the diagnostic criteria for at least one more anxiety disorder, or a patient with a DSM-IV personality disorder who does not also fulfill the diagnostic criteria for at least one more personality disorder (see, e.g., DiNardo & Barlow, 1990; Grove & Tellegen, 1991).

A third characteristic of DSM-III and its successors that has played a role in the rise of psychiatric comorbidity is the limited number of "hierarchical rules" (i.e., rules not permitting a clinician to make a diagnosis in the presence of another diagnosis at a higher hierarchical level). It may be useful to remind readers that in the 1970s a model was proposed according to which, in the presence of a disorder at a given hierarchical level, at least one disorder at each of the lower levels would be present. For instance, in a patient with a diagnosis of schizophrenia, this model would dictate that at least one neurotic disorder would be present (Foulds & Bedford, 1975). One could of course argue that the possibility of diagnosing today, for instance, panic disorder in a patient with a diagnosis of schizophrenia represents a useful development, because it allows a clinician to record some additional information that may be useful for management purposes. But

are we really sure that the panic of a patient with schizophrenia, of a patient with agoraphobia, of a patient with depression, and of a patient with mania is exactly the same psychopathological entity, which simply co-occurs with the other four and requires the same treatment in all four cases? I am not aware of any empirical study addressing this issue, and I doubt that this issue can be addressed with the currently available assessment instruments, due to their limited degree of psychopathological sophistication.

A fourth characteristic of current diagnostic systems that has contributed to the emergence of psychiatric comorbidity is the fact itself that these systems are based on operational diagnostic criteria as opposed to traditional descriptive definitions. Of course, operational diagnostic criteria are more precise and reliable, but the fact is that traditional descriptive definitions tended to convey a gestalt of the various mental disorders, whereas current operational definitions seem to be less able to do so. This may be due at least in part to the fact that descriptive definitions placed different degrees of emphasis on the various clinical aspects of each disorder, whereas current operational definitions tend to give the same weight to the various symptoms; it may also be attributable in part to the fact that traditional definitions included some clinical aspects (e.g., autism in the case of schizophrenia) that have been deleted from current operational definitions because they have been regarded as insufficiently reliable. Whether the gestalt conveyed by a traditional descriptive definition was a fact or an illusion remains open to debate. I raised this issue some years ago concerning the concept of schizophrenia (Maj, 1998): Have we lost the character of schizophrenia as a qualitatively distinct psychosis in the translation of the traditional concept into operational terms? Or was that character as conveyed by the traditional definition actually an illusion that operational criteria, being more precise, have revealed? In any case, the fact is that because traditional descriptive definitions tended to provide a gestalt of the various mental disorders, they encouraged the differential diagnosis between those disorders, whereas operational definitions are less able to convey that gestalt and thus tend to encourage multiple diagnoses.

A fifth characteristic of our current diagnostic systems that has contributed to the emergence of psychiatric comorbidity is the emphasis on the cross-sectional picture of the various mental disorders. Two mental disorders may happen to coexist at a certain point, or an additional disorder may have been present in the lifetime of an individual with a given diagnosis—but only a longitudinal follow-up will clarify whether the two conditions are truly independent of each other, as the general concept of comorbidity (Feinstein, 1970) requires.

The impact of the above-mentioned factors (or at least some of them) in contributing to the emergence of psychiatric comorbidity as a phenomenon, so that comorbidity represents to some extent an artifact of the structure of our current diagnostic systems, is acknowledged from time to time in the literature. However, two main arguments are put forward to advocate the utility of making multiple psychiatric diagnoses in the same patient. These arguments have been summarized by Pincus and colleagues (2004). The first one is that the practice of making multiple psychiatric diagnoses in the same patient "maximizes the communication of diagnostic information." The second argument is that this practice "can provide a more complete appreciation of the complexity of the patient's clinical presentation, which has the potential to result in more appropriate treatment planning."

However, does the practice of making multiple psychiatric diagnoses really increase the quantity of information collected in ordinary clinical settings? And does this practice really result in a more comprehensive approach to treatment? The answers to both these questions remain to be documented by empirical research.

First, we lack convincing evidence that this practice of making multiple psychiatric diagnoses in the same patient is commonly implemented in ordinary clinical settings. Actually, the limited research evidence available seems to point in the opposite direction. Zimmerman and Mattia (1999) have reported that clinicians routinely underdetect psychiatric comorbidity, and that in clinical practice, more often than not, only one diagnosis is made. Adler, Drake, and Teague (1990) found that when a group of psychiatrists were given case histories of patients who met criteria for four personality disorders, two-thirds of them diagnosed only one of the disorders, and no psychiatrist

diagnosed all four. Moreover, we have anecdotal information from several countries (especially low-income ones) suggesting that the information systems adopted in those countries do not have the capacity to incorporate all comorbidities, and sometimes explicitly require the use of just one diagnosis for each patient (Njenga, 2004). Thus the widespread use of DSM-IV as a diagnostic system in clinical settings in many countries does not seem to be paralleled by an equally widespread implementation of the DSM-IV strategy of making multiple psychiatric diagnoses in an individual patient. As a consequence of this, the diagnostic pie is being cut into small slices, to use the metaphor adopted by Pincus and colleagues (2004)—but only one of these slices is currently taken by a clinician confronted with an individual patient, and this slice may not be the same when several clinicians see the same patient, so that a powerful new source of diagnostic unreliability is emerging.

For example, an ordinary patient who would have received in the past a diagnosis of severe neurotic disorder, and who has recurrent mild depressive episodes, panic attacks, social phobia, and obsessions and compulsions, will usually now receive just one of the four diagnoses for which he or she meets the current criteria—and this single diagnosis may not be the same if the patient is seen by various clinicians. One of these clinicians may diagnose major depression, whereas others may diagnose panic disorder, social phobia, or obsessive–compulsive disorder. So the quantity of information that is actually collected may be even less than in the past, and a new source of diagnostic unreliability may be created. In the absence of codified hierarchical rules, several clinicians apply their own idiosyncratic hierarchical rules when making their diagnoses, and these rules differ from one clinician to the other. So, before we can state that the strategy of encouraging multiple psychiatric diagnoses really results in the collection of a greater quantity of diagnostic information in ordinary clinical practice, we certainly need further research evidence.

Let's try now to answer the second question—that is, whether the practice of making multiple psychiatric diagnoses in the same patient, when implemented, results in a more comprehensive treatment approach. Of course, we are not dealing here with those cases in which there is co-occurrence of a mental disorder and a substance use disorder, or of a mental disorder and a physical disease. We are dealing with those cases in which a patient is diagnosed with multiple mental disorders.

Let's take the example of the comorbidity of an anxiety disorder with bipolar I disorder. This is currently reported to be one of the most common forms of psychiatric comorbidity, with a lifetime prevalence of any anxiety disorder in people with bipolar I disorder in the general population of 92.9% (Kessler, 1999), and a reported current prevalence of any anxiety disorder in outpatients with bipolar I disorder of 31.9% (Otto et al., 2006).

It has been repeatedly documented that the anxiety disorder comorbidity has significant prognostic implications in patients with bipolar I disorder. In all cases, these implications are negative—a finding that recurs throughout the literature on psychiatric comorbidity (and that may be regarded to some extent as tautological because it is not surprising that a more complex psychiatric condition will have a poorer prognosis). Anxiety disorder comorbidity has been found to be associated with poorer symptomatic and functional recovery, more frequent suicidal behavior, higher recurrence rate, poorer quality of life, an unfavorable course and outcome, and a reduced acute response to pharmacological treatment (e.g., Bauer et al., 2005; McElroy et al., 2001).

These negative prognostic implications, now repeatedly documented for many years, should have encouraged the conduction of specific drug trials aimed to inform therapeutic decisions in these cases of bipolar I–anxiety comorbidity. Unfortunately, this has not been the case. There is not a single double-blind controlled trial assessing the efficacy of any pharmacological treatment in bipolar I disorder with a comorbid anxiety disorder. Moreover, the few treatment guidelines that provide any advice in this respect usually suggest not treating the comorbid anxiety disorder. "It is appropriate in the majority of the psychiatric comorbidities to defer starting new pharmacotherapy for panic disorder, obsessive–compulsive disorder, post-traumatic stress disorder, [or] eating disorder comorbidity until the benefit of the mood stabilizer can be assessed" (Singh & Zarate, 2006). "The initial goal in

pharmacologic management of patients with bipolar disorder and a co-occurring anxiety disorder is mood stabilization" (Keck, Strawn, & McElroy, 2006).

The current uncertainty and predominantly cautious attitude about the therapeutic implications of the anxiety–bipolar I comorbidity are commonly ascribed to the "routine exclusion of patients with bipolar disorder and comorbidity from pivotal randomized clinical trials for all phases of bipolar illness" (Goldberg, 2001). However, several additional factors probably have to be considered. One of these factors is that almost all the available studies dealing with anxiety disorder comorbidity in bipolar I disorder have been cross-sectional and have focused on lifetime comorbidity. The information that a patient with bipolar I disorder has a lifetime comorbidity of an anxiety disorder is not sufficient to guide treatment. What is the temporal relationship between the occurrence of the anxiety disorder and the course of the bipolar illness? Of course, treatment implications will be very different, depending on whether the anxiety disorder predominantly occurs when the patient is manic, depressed, or euthymic, or whether it has no relationship to the different phases of bipolar illness. The fact is, however, that virtually all the available studies on the bipolar I–anxiety comorbidity do not provide this information.

MacQueen and colleagues (2003) reported that the prevalence of any anxiety disorder was higher in patients with bipolar I disorder who were in a subsyndromal status (80.6%) than in those who were in a syndromal status (53.4%) or in those who were euthymic (38.6%). This is a useful piece of information, but the study does not provide data concerning the relationship between any of the anxiety disorders and depressive versus manic episodes or subsyndromal symptomatology. Strakowski and colleagues (1998) reported that obsessive–compulsive disorder had a course frequently mirroring that of bipolar I disorder, whereas posttraumatic stress disorder showed a separate course from bipolar I disorder in 64% of patients. Again, this information is very useful, but the study did not differentiate among manic, depressive, and mixed phases. Perugi, Akiskal, Toni, Simonini, and Gemignani (2001), in a retrospective study, provided the only available preliminary data on the temporal relationship between individual anxiety disorders and manic and depressive phases. Very interestingly, the onset of panic disorder was concomitant with that of mania or hypomania in 28.6% of cases, versus 4.3% of cases of obsessive–compulsive disorder and no case of social phobia. The authors commented that "many cases of panic disorder in the context of bipolar disorder might reflect mixed manic or hypomanic symptomatology." On the other hand, social phobia may represent a depressive equivalent (Perugi et al., 2001). This is in line with the statement by Himmelhoch (1998) that "the induction of significant hypomania in the majority of social phobics responding to RIMA's [reversible inhibitors of monoamine oxidase type A] and MAOI's [monoamine oxidase inhibitors] suggests that social anxiety itself is part of the bipolar spectrum."

This last observation points to a further reason for the current reluctance in providing any additional treatment for anxiety disorders comorbid with bipolar I disorder: the fear that antidepressant treatment for the anxiety disorder may precipitate mania and exacerbate the bipolar illness. But a further reason for this reluctance is certainly the underlying, usually unexpressed feeling that comorbid anxiety disorders are actually an intrinsic part of the clinical picture of bipolar I disorder and therefore do not require independent treatment. This is probably the main rationale for the suggestion to defer any additional treatment until the benefit of the mood stabilizer can be assessed. It is probably also the explanation for statements that appear in some treatment guidelines and that would otherwise seem unacceptable, like the following one: "Management of bipolar disorder with comorbid panic disorder. The important distinction here is whether the patient has two psychiatric disorders, i.e., bipolar disorder and a comorbid anxiety disorder, or whether the anxiety symptoms are simply a manifestation within the bipolar symptom complex" (Singh & Zarate, 2006). This is, of course, a distinction that a clinician cannot make in most cases in ordinary clinical practice.

Thus, although the prognostic implications of making an additional diagnosis of an anxiety disorder in a patient with bipolar I disorder are clear, the therapeutic implications of this additional diagnosis are at present uncertain. This is the case for many

other forms of psychiatric comorbidity. This situation provides some support to the argument that the practice of making multiple psychiatric diagnoses in the same patient "can obscure the focus of our treatments by 'losing the forest for the trees' " (Pincus et al., 2004), so that "the next editions of the DSM and the ICD might add a provision that only those diagnoses that are included as target symptoms in the current treatment plan should be listed" (Pincus et al., 2004).

However, if we are to understand the full therapeutic implications of psychiatric comorbidity, what we need is further research evidence. Patients with comorbid psychiatric conditions should not be excluded any more from major clinical trials, so that subanalyses focusing on these patients can be carried out. Prospective longitudinal studies, exploring the temporal relationship between the various comorbid disorders, should have priority over retrospective studies providing further data on lifetime comorbidity between these disorders. More detailed assessment of the phenomenology of panic attacks, obsessions and compulsions, social avoidance, and so forth, when they occur in patients with bipolar I disorder, major depression, or schizophrenia, should be another research priority. Finally, a revision of current operational diagnostic criteria, diagnostic interviews, and assessment instruments may be needed to reflect the emerging and evolving phenomenology of some complex syndromes, such as mania and major depression.

If the construct of psychiatric comorbidity has any merit or any meaning, it has it as a starting point and a stimulus for further research work. Meanwhile, it is probably preferable that diagnostic criteria reflect clinical observations rather than theoretical assumptions. For instance, if anxiety is a common symptom of major depression, it should appear in the diagnostic criteria for that disorder.

Conclusion

This chapter has focused on two current problems of psychiatric diagnosis: the question of discriminating between some common mental disorders and homeostatic reactions to adverse life events, and the issue of so-called psychiatric comorbidity. Both of these problems are shaking the foundations of our diagnostic systems today because they emphasize some fundamental drawbacks of these systems: the tendency toward psychopathological oversimplification; the incomplete utilization of the potential of the operational approach; the focus on the reliability of diagnosis at the expense of its validity; and the persisting tendency to conceptualize mental disorders as discrete disease entities. On the other hand, the increasing awareness of these problems is now starting to stimulate new research efforts, which are likely to be useful for the future revision of our classification systems.

References

Adler, D. A., Drake, R. E., & Teague, G. B. (1990). Clinicians' practices in personality assessment: Does gender influence the use of DSM-III Axis II? *Comprehensive Psychiatry*, *31*, 125–133.

American Psychiatric Association. (1952). *Diagnostic and statistical manual of mental disorders*. Washington, DC: Author.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Bauer, M. S., Altshuler, L. L., Evans, D. R., Beresford, T., Williford, W. O., & Hauger, R., et al. (2005). Prevalence and distinct correlates of anxiety, substance and combined comorbidity in a multi-site public sector sample with bipolar disorder. *Journal of Affective Disorders*, *85*, 301–315.

Bjorklund, P. (2005). Can there be a "cosmetic" psychopharmacology? Prozac unplugged: The search for an ontologically distinct cosmetic psychopharmacology. *Nursing Philosophy*, *6*, 131–143.

Blashfield, R. K. (1984). *The classification of psychiatry: Neo-Kraepelinian and quantitative approaches*. New York: Plenum Press.

Clarke, D. M., & Kissane, D. W. (2002). Demoralization: Its phenomenology and importance. *Australian and New Zealand Journal of Psychiatry*, *36*, 733–742.

de Figueiredo, J. M. (1993). Depression and demoralisation: Phenomenologic differences and research perspectives. *Comprehensive Psychiatry*, *34*, 308–311.

Dilsaver, S. C., Chen, Y. R., Shoaib, A. M., & Swann, A. C. (1999). Phenomenology of

mania: Evidence for distinct depressed, dysphoric, and euphoric presentations. *American Journal of Psychiatry*, *156*, 426–430.

DiNardo, P. A., & Barlow, D. H. (1990). Syndrome and symptom co-occurrence in the anxiety disorders. In J. D. Maser & C. R. Cloninger (Eds.), *Comorbidity of mood and anxiety disorders* (pp. 205–230). Washington, DC: American Psychiatric Press.

Elliott, C. (1999a). *A philosophical disease: Bioethics, culture and identity*. London: Routledge.

Elliott, C. (1999b). Prozac and the existential novel: Two therapies. In C. Elliott & J. Lantos (Eds.), *The last physician: Walker Percy and the moral life of medicine* (pp. 59–69). Durham, NC: Duke University Press.

Feinstein, A. R. (1970). The pre-therapeutic classification of co-morbidity in chronic disease. *Journal of Chronic Diseases*, *23*, 455–468.

Foulds, G. A., & Bedford, A. (1975). Hierarchy of classes of personal illness. *Psychological Medicine*, *5*, 181–192.

Goldberg, J. F. (2001). Bipolar disorder with comorbid substance abuse: Diagnosis, prognosis, and treatment. *Journal of Psychiatric Practice*, *7*, 109–122.

Grove, W. M., & Tellegen, A. (1991). Problems in the classification of personality disorders. *Journal of Personality Disorders*, *5*, 31–41.

Haslam, M., & Beck, A. T. (1994). Subtyping major depression: A taxometric analysis. *Journal of Abnormal Psychology*, *103*, 686–692.

Healy, D. (1993). Dysphoria. In C. G. Costello (Ed.), *Symptoms of depression* (pp. 24–45). Chichester, UK: Wiley.

Helmchen, H., & Linden, M. (2000). Subthreshold disorders in psychiatry: Clinical reality, methodological artefact, and the double-threshold problem. *Comprehensive Psychiatry*, *41*, 1–7.

Himmelhoch, J. M. (1998). Social anxiety, hypomania and the bipolar spectrum: Data, theory and clinical issues. *Journal of Affective Disorders*, *50*, 203–213.

Keck, P. E., Jr., Strawn, J. R., & McElroy, S. L. (2006). Pharmacologic treatment considerations in co-occurring bipolar and anxiety disorders. *Journal of Clinical Psychiatry*, *67*(Suppl. 1), 8–15.

Kendell, R., & Jablensky, A. (2003). Distinguishing between the validity and utility of psychiatric diagnoses. *American Journal of Psychiatry*, *160*, 4–12.

Kendler, K. S. (1999). Setting boundaries for psychiatric disorders. *American Journal of Psychiatry*, *156*, 1845–1848.

Kessler, R. C. (1999). Comorbidity of unipolar and bipolar depression with other psychiatric disorders in a general population survey. In M. Tohen (Ed.), *Comorbidity in affective disorders* (pp. 1–25). New York: Dekker.

Kessler, R. C., Angermeyer, M., Anthony, J. C., de Graaf, R., Demyttenaere, K., Gasquet, D., et al. (2007). Lifetime prevalence and age-of-onset distributions of mental disorders in the World Health Organization's World Mental Health Survey initiative. *World Psychiatry*, *6*, 168–176.

Kessler, R. C., McGonagle, K. A., Zhao, S., Nelson, C. B., Hughes, M., Eshleman, S., et al. (1994). Lifetime and 12-time prevalence of DSM-III-R psychiatric disorders in the United States: Results from the National Comorbidity Survey. *Archives of General Psychiatry*, *51*, 8–19.

Kessler, R. C., Nelson, C. B., McGonagle, K. A., Liu, J., Swartz, M., & Blazer, D. G. (1996). Comorbidity of DSM-III-R major depressive disorder in the general population: Results from the US National Comorbidity Survey. *British Journal of Psychiatry*, *168*(Suppl. 30), 17–30.

Kramer, P. (1993). *Listening to Prozac*. New York: Penguin Books.

Kutchins, H., & Kirk, S. A. (1997). *Making us crazy: DSM: The psychiatric bible and the creation of mental disorders*. New York: Free Press.

Linden, M., Helmchen, H., Mackert, A., & Müller-Oerlinghausen, B. (1994). Structure and feasibility of a standardized stepwise drug treatment regimen (SSTR) for depressed patients. *Pharmacopsychiatry*, *27*, 51–53.

MacQueen, G. M., Marriott, M., Begin, H., Robb, J., Joffe, R. T., & Young, L. T. (2003). Subsyndromal symptoms assessed in longitudinal, prospective follow-up of a cohort of patients with bipolar disorder. *Bipolar Disorders*, *5*, 349–355.

Maj, M. (1998). Critique of the DSM-IV operational diagnostic criteria for schizophrenia. *British Journal of Psychiatry*, *172*, 458–460.

Maj, M. (2005a). The aftermath of the concept of "psychiatric comorbidity." *Psychotherapy and Psychosomatics*, *74*, 67–68.

Maj, M. (2005b). "Psychiatric comorbidity": An artefact of current diagnostic systems? *British Journal of Psychiatry*, *186*, 182–184.

Maj, M. (2007). Are we able to differentiate be-

tween true mental disorders and homeostatic reactions to adverse life events? *Psychotherapy and Psychosomatics*, *76*, 257–259.

Maj, M. (2008). Depression, bereavement, and "understandable, intense sadness: Should the DSM-IV approach be revised? *American Journal of Psychiatry*, *165*, 1373–1375.

McElroy, S. L., Altshuler, L. L., Suppes, T., Keck, P. E., Jr., Frye, M. A., Denicoff, K. D., et al. (2001). Axis I psychiatric comorbidity and its relationship to historical illness variables in 288 patients with bipolar disorder. *American Journal of Psychiatry*, *158*, 420–426.

McGuire, M. T., & Troisi, A. (1998). *Darwinian psychiatry*. New York: Oxford University Press.

Njenga, F. (2004). Comorbidity: The African perspective. *World Psychiatry*, *3*, 30–31.

Otto, M. W., Simon, N. M., Wisniewski, S. R., Miklowitz, D. J., Kogan, J. N., Reilly-Harrington, N. A., et al. (2006). Prospective 12-month course of bipolar disorder in outpatients with and without comorbid anxiety disorders. *British Journal of Psychiatry*, *189*, 20–25.

Perugi, G., Akiskal, H. S., Toni, C., Simonini, E., & Gemignani, A. (2001). The temporal relationship between anxiety disorders and (hypo) mania: A retrospective examination of 63 panic, social phobic and obsessive–compulsive patients with comorbid bipolar disorder. *Journal of Affective Disorders*, *67*, 199–206.

Pincus, H. A., Tew, J. D., Jr., & First, M. B. (2004). Psychiatric comorbidity: Is more less? *World Psychiatry*, *3*, 18–23.

Regier, D. A., Kaelber, C. T., Rae, D. S., Farmer, M. E., Knauper, B., Kessler, R. C., et al. (1998). Limitations of diagnostic criteria and assessment instruments for mental disorders: Implications for research and policy. *Archives of General Psychiatry*, *55*, 109–115.

Robins, L. (1994). How recognizing 'comorbidities' in psychopathology may lead to an improved research nosology. *Clinical Psychology: Science and Practice*, *1*, 93–95.

Ruscio, J., & Ruscio, A. M. (2000). Informing the continuity controversy: A taxometric analysis of depression. *Journal of Abnormal Psychology*, *109*, 473–487.

Sato, T., Bottlender, R., Kleindiest, N., & Möller, H.-J. (2005). Irritable psychomotor elation in depressed inpatients: A factor validation of mixed depression. *Journal of Affective Disorders*, *84*, 187–196.

Singh, J. B., & Zarate, C. A., Jr. (2006). Pharmacological treatment of psychiatric comorbidity in bipolar disorder: A review of controlled trials. *Bipolar Disorders*, *8*, 696–709.

Sperry, L., & Prosen, H. (1998). Contemporary ethical dilemmas in psychotherapy: Cosmetic psychopharmacology and managed care. *American Journal of Psychotherapy*, *52*, 54–63.

Spitzer, R. L., & Wakefield, J. C. (1999). DSM-IV diagnostic criterion for clinical significance: Does it help solve the false positives problem? *American Journal of Psychiatry*, *156*, 1856–1864.

Strakowski, S. M., Sax, K. W., McElroy, S. L., Keck, P. E., Jr., Hawkins, J. M., & West, S. A. (1998). Course of psychiatric and substance abuse syndromes co-occurring with bipolar disorder after a first psychiatric hospitalization. *Journal of Clinical Psychiatry*, *59*, 465–471.

U.S. Department of Health and Human Services. (1999). *Mental health: A report of the Surgeon General. Executive summary*. Rockville, MD: Author.

van Praag, H. M. (1998). The diagnosis of depression in disorder. *Australian and New Zealand Journal of Psychiatry*, *32*, 767–772.

Wakefield, J. C. (1997). Diagnosing DSM-IV: Part I. DSM-IV and the concept of disorder. *Behaviour Research and Therapy*, *35*, 633–649.

Wakefield, J. C., Schmitz, M. F., First, M. B., & Horwitz, A. V. (2007). Extending the bereavement exclusion for major depression to other losses: Evidence from the National Comorbidity Survey. *Archives of General Psychiatry*, *64*, 433–440.

World Health Organization (WHO). (2001). *The world health report 2001: Mental health: New understanding, new hope*. Geneva: Author.

Zimmerman, M., & Mattia, J. I. (1999). Psychiatric diagnosis in clinical practice: Is comorbidity being missed? *Comprehensive Psychiatry*, *40*, 182–191.

Zisook, S., Shear, K., & Kendler, K. S. (2007). Validity of the bereavement exclusion criterion for the diagnosis of major depressive episode. *World Psychiatry*, *6*, 102–107.

Zisook, S., Shuchter, S. R., Pedrelli, P., Sable, J., & Deaciuc, S. C. (2001). Bupropion sustained release for bereavement: Results of an open trial. *Journal of Clinical Psychiatry*, *62*, 227–230.

CHAPTER 14

Taking Disorder Seriously

A Critique of Psychiatric Criteria for Mental Disorders from the Harmful-Dysfunction Perspective

JEROME C. WAKEFIELD

Psychiatry, clinical psychology, clinical social work, and the other mental health professions—for convenience, I collectively refer to these professions here as "psychiatry"—claim to address mental problems that are disorders in the medical sense. This is the rationale for placing these professions within the broader medical professions and providing them with the unique support that the medical professions receive, such as reimbursement for treatment by medical insurance and research funding targeted at health research. Not only is the concept of mental disorder at the foundation of psychiatry as a medical discipline; it is also at the heart of scholarly and public disputes about which mental conditions should be classified as pathological and which as normal suffering or problems of living, and it has ramifications not just for psychiatric diagnosis but also for research, policy, and prevention/screening efforts.

To say that mental disorder is the *primary* or *essential* target of psychiatry is not to deny that psychiatry is mandated to intervene in various other domains of problematic mental conditions. Some of these other conditions are indicated in DSM as nondisordered conditions that are commonly the target of psychiatric intervention (DSM's "V Codes"). These include, for example, problems in selecting a career, relationship problems, normal grief, and other such problems in living. The same distinction exists in physical medicine; for example, fertility, pregnancy, and childbirth pain are all normal conditions that are often the target of medical intervention. Both physical and mental medicine are mandated to extend the application of their knowledge and skills beyond prevention and treatment of disorder to include such tasks as (1) reduction of mental suffering and social role impairment from normal mental states (e.g., grief); (2) control and treatment of normal but socially undesirable trait variations (e.g., lack of assertiveness, higher-than-average fear of public speaking, less-than-average social skill) in individuals whose traits do not fit our social templates, when such normal variation becomes so disadvantageous that it blocks access to our culture's distinctive opportunities and thus is an issue of "psychological justice" (Wakefield, 1988a, 1988b); and (3) enhancement of individuals' normal mental function in "cosmetically" desirable ways

that serve the personal happiness of the individual (e.g., relationship skills, resilience training, leadership training).

I ignore these other domains of intervention here because they are not part of the primary rationale for the existence of the mental health professions as medical professions. As the appellation "mental health professions" would suggest, whatever else one might ask of psychiatry, its essential and defining concern is mental disorder.

Note also that the equation that for convenience I assume here between "mental health" and "absence of mental disorder" has been questioned by "positive psychiatrists" (e.g., Vaillant, 2003), as well as by an expansive World Health Organization (WHO) definition of health that includes social and economic well-being. The "positive psychiatrists" argue that health includes a domain of superlative functioning that goes beyond lack of disorder and includes possession of high levels of (for example) mental fitness and resilience, virtues like courage, and positive feelings such as happiness. However, for present purposes, I work within the framework of the traditional notion that health is a lack of disorder. The question of whether there exists an additional domain of positive mental health requires its own extended answer, to be provided elsewhere (for an initial foray, but in Italian, see Wakefield, 2005b). Even if there is such a domain, that additional target for the mental health professions would not greatly influence my argument about the concept of disorder. And, admittedly, I am prima facie skeptical of expansive claims about the domain of positive mental health, which I suspect are being used to smuggle into the medical category what mostly consist of nonmedical and culturally loaded value judgments about what constitutes a good life.

Given the centrality of the concept of disorder to psychiatry, it is important to ask this question: When are mental conditions justifiably considered mental disorders rather than other kinds of negative mental conditions, such as problems in living, bad relationships, socially disadvantageous traits, or traits that simply make one unhappy? To answer this question requires an answer to the prior question: What is the meaning of the term "mental disorder"? The credibility and even the coherence of psychiatry as a medical discipline depend on there being a persuasive answer to this question.

I approach this question via a conceptual analysis that asks: What do we generally mean when we say that a problematic mental condition—such as adolescent antisocial behavior, a child's defiant behavior toward a parent, intense sadness, intense worry, intense shyness, failure to learn to read, or heavy use of illicit drugs—is not merely a form of normal (albeit undesirable and painful) human functioning, but indicative of psychiatric disorder? Given the surprising degree of agreement about which such conditions are mental disorders, it can be presumed that such judgments are guided by, and must be explained by, a shared conceptual structure represented in the minds of those making the judgments.

Among existing attempts to analyze the concept "mental disorder," a basic division is between value-based and scientific approaches. As Kendell (1986) put it, "The most fundamental issue, and also the most contentious one, is whether disease and illness are normative concepts based on value judgments, or whether they are value-free scientific terms; in other words, whether they are biomedical terms or sociopolitical ones" (p. 25). I have proposed a hybrid account, the "harmful-dysfunction" (HD) analysis of the concept of mental disorder (Wakefield, 1992a, 1992b, 1993, 1996, 1997, 1999a, 1999b). According to this analysis, a disorder is a "harmful dysfunction"—where "harmful" is a value-based term, referring to conditions judged negative by sociocultural standards, and "dysfunction" is a scientific factual term, referring to failure of biologically designed functioning. In modern science, "dysfunction," I argue, is ultimately anchored in evolutionary biology and refers to the failure of an internal mechanism to perform one of its naturally selected functions.

In this chapter, while commenting on some other approaches, I focus on exploring the considerable explanatory power of the HD analysis for understanding the distinction between mental disorder and other problematic mental conditions. I also illustrate the implications of the analysis for assessing the validity of DSM diagnostic criteria.

A few other initial caveats should be stated before I examine some past and present

attempts to define mental disorder. First, I am concerned here with understanding what makes a mental condition a mental *disorder* in the medical sense. I sidestep the perplexing (but, I think, not as conceptually interesting) question: What makes a specific medical disorder a *mental* disorder rather than a physical disorder? (But see Wakefield, 2007.)

There are many further questions about the conceptual foundations of nosology and diagnosis, other than the distinction between disorder and nondisorder, that I do not address here. I believe that most such questions—for example, how to define a specific form of disorder; how to distinguish one disorder from another; the proper role in a classification system of superordinate categories (e.g., "anxiety disorders," "externalizing disorders"); and the dimensional versus categorical structure of disorder indicators and etiological variables—cannot be productively addressed until the more fundamental distinction between disorder and nondisorder is clarified.

I focus on the term "disorder" to identify psychiatry's primary domain as a medical discipline because it covers all forms of pathology, including traumatic injuries and diseases. Some writers emphasize other terms, such as "illness" or "disease" to specify the overall set of pathological conditions relevant to medicine, and "mental illness" or "mental disease" to specify the overall set of pathological conditions that are the special concern of psychiatry. But these other terms have connotations that are too specific as generic terms for medical conditions. For example, are broken bones and snake phobias "illnesses," and are physical poisonings and posttraumatic stress disorder (PTSD) "diseases"? They are surely all disorders.

Contrary to an impression I have occasionally encountered, the use of the generic medical term "disorder" to refer to the entire set of mental pathological conditions that are the primary target of psychiatry did not start with DSM at all, but rather has a long history. To take some random examples, "disorder" was already the term of choice for mental pathology in various entries of Samuel Johnson's *Dictionary*, published in 1755. In the second issue, in October 1844, of the *American Journal of Insanity* (later to morph into the *American Journal of Psychiatry*), the editor, A. Brigham, published an essay on "The Definition of Insanity" that began: "By Insanity is generally understood some disorder of the faculties of the mind" (p. 97). The bibliography included a book by Dr. Henry Johnson, titled *On the Arrangement and Nomenclature of Mental Disorders*, published just the year before (1843). Of course, the use of the term "disorder" leaves open precisely what kind of "order" is supposed to be failing in medical pathology. "Disorder" can be used generically for failure of many types of order, as in "civic disorder." I argue below that in medicine, it is the order derived from the biologically designed functioning of the mind and body that is claimed to fail in attributions of mental and physical disorder. The only other candidate seems to be the order imposed by social values (Spitzer, 1999), and that approach, I argue, does not offer a coherent account of our diagnostic judgments and intuitions.

A mental disorder may be considered a disorder of mental mechanisms and thus conceptually analogous to disorders of other kinds of mechanisms. Consequently, the problem is to define "disorder" in the general sense used in medicine and then apply it to the domain of mental mechanisms. The domain of "mental mechanisms" is not defined in some Cartesian metaphysical way, but simply as whatever hypothesized brain mechanisms underlie certain capacities we label "mental," such as thought, emotion, perception, speech, appetitive behavior, and so on. What deeper property, if any, unites these processes under the category "mental"—such as perhaps the involvement of representational structures—is left unaddressed here.

Because the analysis here ultimately concerns the general concept of disorder as applied to both mental and physical conditions, examples from both mental and physical domains are used to test the analysis. I use "internal mechanism" as a general term to refer to physical structures and organs as well as to mental structures and dispositions, such as motivational, cognitive, affective, and perceptual mechanisms.

The fact that mental disorders are medical disorders in a conceptual sense does *not* necessarily mean that mental disorders must be physiological brain disorders. Mental functions can fail because of problems with functioning at the representational ("soft-

ware") level rather than the physiological ("hardware") level.

I do not assume that there is a precise or crisp boundary between disorder and nondisorder. It is assumed that "mental disorder," like most concepts, has areas of indeterminacy, ambiguity, fuzziness, and vagueness, and that a successful analysis should reflect and explain such aspects of our judgments. One must distinguish the problem of drawing boundaries between disorder and nondisorder along continua from the question of whether there is a meaningful distinction between clear cases of disorder and nondisorder. For example, there is no sharp boundary between being a child and being an adult, but there are lots of clear cases of being a child and of being an adult. No doubt, boundary setting sometimes in part involves values, and is very sensitive to current social views and knowledge. So, for example, the place where the boundary between childhood and adulthood is drawn in different domains (voting, religious services, drivers' licenses, drinking age, potential military service, etc.) does not necessarily represent a natural boundary, but may reflect, within the fuzziness of a continuous dimension, a choice based on broader goals and values. But all this boundary setting is built upon the foundation of a real distinction between clear cases of children and adults. The problem with current diagnostic criteria, I argue, is not with boundary setting but with clear cases of nondisorders being misclassified as disorders.

Lastly, it should be emphasized that the question of whether a condition is a disorder is not the same as the question of whether it should be treated or would benefit from being treated (Spitzer, 1998). Some disorders should not be treated, and some nondisorders should be treated. Perhaps, for example, intense normal grief is best ameliorated at times, just as is childbirth pain. But there are costs to misdiagnosing such normal conditions as disorders: If an individual is believed to be disordered, this belief shapes perceived prognosis and treatment choice, and does not offer the individual fully informed consent in light of the true nature of the condition and the full range of reasonable options for addressing it. Diagnosis of disorder can bias treatment choice toward medication and toward attempts to "fix" the individual rather than change the environment. It also tends to eliminate the option of watchful waiting as an alternative, relative to a diagnosis of an intense normal reaction to environmental circumstances. I focus here on the concept of disorder, and this question is relevant to, but not identical to, such practical questions as whether or how a condition should be treated.

The False-Positives Problem in DSM

From the perspective of DSM-V, one of the main motives for clarifying the concept of mental disorder is to help improve the ability to draw the distinction between mental disorders and nondisordered problems of living. The distinction needs clarification because the label "mental disorder," it has been widely argued, is often incorrectly applied to many other kinds of undesirable but nondisordered conditions.

The issue of whether the conceptual boundary of disorder has been overextended to include nondisordered problems of living is not new, but it has evolved into a new form. In the 1960s and 1970s, there were vehement criticisms of psychiatry from both professional and nonprofessional sources who argued that there is no such thing as "mental disorder" at all in the literal medical sense of disorder. Psychiatric diagnosis was claimed to be just a matter of medically labeling nondisordered but socially disvalued conditions for purposes of social control. It was argued that psychiatrists could not reliably distinguish disorder from nondisorder or one purported disorder from another, thus proving the invalidity of their diagnostic concepts.

There are many reasons why the antipsychiatric movement is no longer a potent force, but one reason is that with the publication of DSM-III (American Psychiatric Association, 1980), many of the antipsychiatrists' criticisms were squarely and systematically addressed by the psychiatric community. DSM-III provided a definition of mental disorder that attempted to distinguish mental disorders in the medical sense from social deviance and other kinds of personal and social problems. Moreover, common nondisordered conditions that may warrant psychiatric attention were distinguished from disorders

and listed separately in a section called "V Codes for Conditions Not Attributable to a Mental Disorder That Are a Focus of Attention or Treatment." Most importantly, DSM-III offered operationalized theory-neutral definitions of each disorder that improved reliability and contributed to valid differentiation of disorders from nondisorders and of one disorder from another. These innovations—along with other developments, such as the growing evidence of a biological basis and effective pharmacological treatment for some disorders—have pretty much put the antipsychiatric critiques to rest. The claim that the concept of mental disorder is incoherent or that mental disorders do not exist is rarely heard these days except in postmodernist or radical behaviorist treatises, and it is certainly not a major concern in public discourse about psychiatry.

Psychiatry now faces a new set of challenges regarding its basic concept of mental disorder, and I would argue that a comparably systematic assault on these challenges should be undertaken in the process of constructing DSM-V. The new challenges again come both from within the mental health professions and from the lay public. Their focus is not on whether mental disorders exist at all, but rather on whether mental health professionals, when using DSM criteria, are overdiagnosing disorders so as to invalidly pathologize many other kinds of human problems. This new challenge contains an echo of the old antipsychiatric concerns about social control and mislabeling of nondisordered conditions as disorders. However, it is much more subtle and targeted, and it is not inherently antagonistic to the broader goals and conceptual approach of psychiatry. The new challenge consists of a diverse set of objections to the labeling of specific conditions as disorders on a category-by-category basis. The claim is not so much that whole categories are bogus, as that overly inclusive criteria mix normal conditions with true disorders in heterogeneous disorder categories that confuse the nature and prognosis of conditions, give rise to faulty epidemiological prevalence estimates and treatment outcome research, and lead to misdiagnosis that undermines informed treatment decisions.

I refer to the problem of psychiatric criteria that potentially classify nondisorders as disorders as the "false-positives" problem. To the degree that diagnostic criteria successfully identify all and only disordered individuals as disordered, the criteria are referred to as "conceptually valid" criteria (Wakefield, 1992a, 1992b).

Conceptual validity has proven difficult to attain with diagnostic criteria that are framed in terms of symptoms, such as DSM's typical criteria. The reason is simply that the symptoms of many mental disorders can occur as normal responses to certain kinds of environments. For example, deep sadness can indicate major depressive disorder, or it can indicate a normal reaction to loss. Intense anxiety can be a symptom of generalized anxiety disorder, or it can be a normal response to an unusually stressful set of circumstances. Adolescent antisocial behavior can represent a dysfunction, such as an inability to empathize with others' needs, an inability to function according to social rules, or an inability to inhibit impulses, and thus can indicate conduct disorder; or such behavior can represent the consequences of a rational decision to join a gang and go along with gang antisocial activities as a way to protect oneself in a dangerous neighborhood. Although excessive alcohol intake is a manifestation of dependence, it can also represent a transient youthful attempt to be exuberantly excessive that involves neither addiction nor abuse. (Some of these examples are developed in greater detail later in this chapter.) In these and many other instances, criteria that rely exclusively on symptoms to identify disorders are in danger of also encompassing potentially large numbers of normal conditions with the same "symptoms" as the disorders.

It should be noted that even those disordered conditions that do not fall under standard diagnostic criteria for a given disorder may still be diagnosed as a disorder under DSM's "wastebasket" categories of diagnosis "not otherwise specified" (NOS; e.g., mood disorder NOS). For example, even subthreshold conditions that do not satisfy diagnostic criteria can nonetheless be classified as disorders via the corresponding NOS category. In contrast, there is no mechanism within DSM allowing the classification of a condition that does satisfy the diagnostic criteria, but that the clinician judges is not a disorder, to be classified as a normal re-

action rather than a disorder. The primary response to false positives should be to adjust diagnostic criteria to reflect the distinction between disorder and nondisorder more validly (Spitzer & Wakefield, 1999). The critique of psychiatric diagnostic criteria based on the HD analysis attempts to accomplish the first step toward such revisions.

Why Psychiatry Can't Escape the Analysis of the Concept of Mental Disorder

The DSM and ICD diagnostic criteria are currently the primary arbiters of what is disordered versus nondisordered in most clinical practice and research. But they are clearly not conceptually final arbiters. The criteria are regularly revised to make them more valid in indicating disorder and to eliminate false positives; such revisions implicitly acknowledge that "errors" in the criteria are possible. Moreover, both the popular press and critics within the mental health professions challenge the validity of the criteria in picking out mental disorder, and these disputes do not seem entirely arbitrary, but rather often seem to appeal to an underlying shared notion of disorder. Indeed, professionals often classify conditions using the NOS category, which requires a sense of what is and is not a disorder independent of specific diagnostic criteria.

Granting the common observation that there is no "gold-standard" laboratory test or physiological indicator for mental disorders and that current criteria are fallible, we might still ask: Why must we grapple with the elusive concept of disorder itself when there are so many empirical techniques for identifying disorders? The reality is that all of the tests commonly used to distinguish disorder from nondisorder rest on implicit assumptions about the concept of disorder; otherwise, it is not clear whether the test is distinguishing disorder from nondisorder, one disorder from another disorder, or one nondisordered condition from another. Common tests of validity—such as statistical deviance, family history/genetic loading, predictive validity, Kendell's (1975; Kendell & Brockington, 1980) discontinuity of distribution, factor-analytic validity, construct validity, syndromal co-occurrence of symptoms, response to medication, the Robins and Guze (1970) criteria, Meehl's (1995; Meehl & Yonce, 1994, 1996) taxometric analysis, and all other such guides—can identify a valid construct and separate one such construct from another. But whether the distinguished constructs are disorders or nondisorders goes beyond the test's capabilities. Every such test is equally satisfied by myriad normal as well as disordered conditions. Even the currently popular (in the United States) use of role impairment does not inherently distinguish disorder from nondisorder (and for this reason is generally avoided by ICD) because there are many normal conditions, ranging from sleep and fatigue to grief and terror, that not only impair routine role functioning but are biologically designed to do so. It only *seems* as though these various kinds of empirical criteria provide a stand-alone standard for disorder because they are used within a context in which disorders (in some background conceptual sense) are already implicitly and independently inferred to exist, and the aim is simply to distinguish among disorders. This essential background assumption itself depends on the concept of disorder being deployed prior to and independently of the specific empirical test. Thus there is no substitute for the concept of mental disorder as the ultimate standard. None of our empirical approaches work without a basis in a conceptual analysis of disorder.

A further reason why we must rely on the concept of disorder is the lack of definitive etiological understanding of mental disorder and the consequent theoretical fragmentation of psychiatry. This fragmentation provided the impetus for the decision to provide theory-neutral criteria in DSM and ICD for diagnosing disorders. Etiological theory (e.g., the return of the repressed, irrational ideas, serotonin deficit) would generally provide ways to distinguish disorder from nondisorder in a more developed science. The need to rely for now on theory-neutral criteria means that the concept of disorder itself, which is to some extent shared by various theories, offers the best way of judging whether a theory-neutral diagnostic criteria set picks out disorders rather than normal conditions (i.e., is conceptually valid [Wakefield, 1992a]). Theory-neutral criteria work to the extent that they adhere to an implicit

understanding of disorder versus nondisorder that is shared across most theoretical perspectives and constitutes a provisional basis for shared identification of disorders for research purposes.

Disorder as Social Evaluation and Sanctioned Seeking of Help

I start by considering two pre-DSM-III attempts to define disorder. The first of these was the behaviorist account. Psychologists were heavily under the sway of behaviorism at the time of the DSM-III revolution, and consequently had little to offer psychiatrists by way of a conceptually sound definition of mental disorder. They saw all behavior as learned via the same normal principles of stimulus and response, and thus they could delineate no theoretically deep difference between the conditions labeled mental disorders and those labeled normal. Instead, they suggested that the difference lay in social evaluations that led to treatment—in effect, asserting that disorder is whatever we decide disorder should be. Starting with Eysenck's (1960) classic analysis and critique of medicalization, behaviorists argued against any deep theoretical difference between disorder and nondisorder. Oddly enough, this placed behaviorists logically in the same camp as antipsychiatrists who argued that diagnosis was essentially about social evaluation and social control.

Here are some excerpts regarding the concept of disorder from the seminal behaviorist account by Ullman and Krasner (1975):

> The central idea of this book is that the behaviors traditionally called abnormal are no different, either quantitatively or qualitatively, in their development and maintenance from other behaviors. . . . In general conversation the word "abnormal" is used to signify that something is unexpected, irregular, and different from the normal or predictable state of affairs. (p. 2) Abnormality was defined as behavior violating interpersonal expectations in a manner that sanctions intervention of mental health practitioners. (p. 9) The principal argument of this book is that abnormal behavior is no different from normal behavior in its development, its maintenance, or the manner in which it may be changed. The difference between normal and abnormal behavior is not intrinsic; rather it lies in a societal reaction. (p. 32) "Sick" and "healthy" labels for behavior represent *social evaluations*. (p. 33; original emphasis)

This value-based account of mental disorder places social evaluation—and consequently sanctions for treatment—at the heart of the concept. This definition seems tailor-made to allow mental health professionals to treat and receive reimbursement for all negatively evaluated conditions for which people may want help.

Ullman and Krasner (1975) offered a quite cogent criterion for a successful definition of disorder—that it be a necessary and sufficient criterion for explaining what people do in fact judge to be disordered: "A definition of abnormality should have certain characteristics. . . . The definition of "abnormal" should include all the people who are indeed abnormal and none of the people who are not. To the extent that abnormal people are not so designated and normal people are, the definition leads to error" (p. 11). Yet they never seriously tested their definition against this simple criterion, and the definition they did offer fails their test miserably. For one thing, myriad behaviors are socially evaluated as negative (from lack of courtesy to incompatible marriages), and people may even think it useful to seek help for these, but people do not label them as mental disorders or even as abnormal behaviors (in the relevant, functional sense of abnormality—statistical abnormality in and of itself is clearly not a pertinent definition of mental disorder; see Wakefield, 1992a). Regarding service use, professional services for a condition may not be available in a given locale or time period, and for a variety of reasons people may not be inclined to utilize such services for certain disordered conditions when they do exist; yet the condition can be a disorder nonetheless.

Disorder as Symptom–Course Syndrome

A common view throughout psychiatry is that disorders are, at least initially pending identification of their etiology, definable by syndromes. A "syndrome" in this sense is simply a condition consisting of some co-

occurring symptoms that may also have a typical course. Psychiatrists have talked a lot about syndromal definitions of mental disorder, by which they mean the identification of a disorder by its symptoms and course. Some specific disorders may have a syndromal structure that enables one to recognize that disorder and to distinguish it from other disorders. Unfortunately, the reasonable idea of using syndromal criteria to pick out a specific disorder once a disordered condition is recognized to exist has gotten confused with the unreasonable idea of defining the very concept of a condition as a psychiatric disorder—that is, differentiating normality and pathology—in terms of possessing syndromal structure. But this makes no sense; the fact that a condition contains certain phenomena (or "symptoms") that regularly tend to co-occur, and that the condition tends to have a somewhat predictable course, says nothing in and of itself about whether the condition is a disorder.

This view has had enormous influence, To take a random example, in explaining the "dependence syndrome" concept of alcoholism that he helped to define and that has reshaped DSM and ICD definitions of substance use disorders, Edwards (1986) says: "The meaning to be given to the term 'syndrome' deserves some attention. . . . [W]hat is essentially implied is a co-occurrence, with some coherence" (p. 172).

In 1972, a highly influential article was published by a group of psychiatric researchers at Washington University in St. Louis (Feighner et al., 1972). This was a report of newly formulated operational diagnostic criteria for mental disorders. The Feighner et al. publication became the precursor of DSM-III's approach to diagnosis. A couple of years later, three authors of this paper published an influential book on psychiatric diagnosis that elaborated on the Feighner et al. criteria and ushered in the DSM era. To explain how they delineated disorders, the authors (Woodruff, Goodwin, & Guze, 1974) stated:

> When the term "disease" is used, this is what is meant: a disease is a cluster of symptoms and/or signs with a more or less predictable course. Symptoms are what patients tell you; signs are what you see. The cluster may be associated with physical abnormality or may not. The central point is that it results in consultation with a physician who specializes in recognizing, preventing, and, sometimes, curing diseases. (p. x)

Although one finds many statements like this in the literature, from a conceptual perspective it makes no sense to characterize disorder in terms of possession of syndromal structure; the fact that a condition is characterizable as a syndrome is neither sufficient (there are many normal syndromes) nor necessary (there can be single-symptom disorders of unpredictable course) for characterizing it as a disorder. The definition is thus absurd even if we leave aside the seeming implication of the last sentence that by definition a disorder is a condition that results in consultation with a physician: Were there no disorders before there were physicians? If we get rid of physicians, do we get rid of disorders?

Even Ullman and Krasner (1975) saw the main problem with this sort of pure syndromal symptom-cluster-and-course approach to disorder: Namely, it encompasses myriad normal conditions. Ironically, however, the syndromal definition was not all that different from their own approach, and both suffered from the same sorts of deficiencies. They said of the syndromal definition: "By this definition, disease is whatever the physician deals with. . . . [Does] a college student, whose status has a "natural history" (including a cure known as graduation) . . . qualify as having a disease? Do fatigue, irritability, and dislike of psychology texts qualify as symptoms and/or signs? And is the use of the campus counseling center a necessary part of the syndrome?" (p. 13). Despite the chiding, the only change in the definition they suggested was adding maladjustment in cultural context as a necessary part of the concept of disorder (p. 12). Yet, clearly, one can reconstruct the same objections after adding that requirement. For example, if having difficulty in college, as described in Ullman and Krasner's counterexample to the syndromal definition, is unacceptable and maladaptive within one's (let's suppose) academically driven cultural context, does that make such problems a mental disorder?

Although the identification of syndromes is of scientific importance, the notion that one can use the syndrome notion to define

the concept of disorder is a ground-level confusion, albeit one repeated by many associated with DSM. It is true that disorders are often recognized as specific disorders via syndromes and often are syndromally defined prior to etiological knowledge, in both physical and mental medicine. But syndromal diagnosis in psychiatry and in medicine more generally still involves the classification of conditions *as disorders*. Thus there is an implicit step prior to the syndromal definition, in which the particular syndrome is inferred to indicate a disorder. This necessary step in using a syndromal approach to disorder is entirely ignored in psychiatric accounts, leaving the disorder status of the syndrome unexplained.

If the HD analysis is correct about the meaning of disorder, then during the syndromal phase of classification of disorders, there must be an implicit inference that the syndromally defined pattern is due to a dysfunction. Syndromal definitions do gradually get replaced by etiological definitions on the basis of accumulating scientific knowledge about the nature of the involved dysfunctions. But prior to that knowledge, the conditions are already classified as disorders (often for millennia) on the basis of their syndromal manifestations. The reason this can occur is that on the basis of the symptom syndrome and circumstantial evidence, but without actual knowledge of etiology, it is inferred (fallibly, but often correctly) that there is a dysfunction underlying the particular syndrome (see the discussion below of the "epistemological objection" to the HD analysis).

Without such an inference to dysfunction, *any* pattern, normal or abnormal, could constitute a "syndrome." Normal shortness is a problematic "syndrome," as is relative stupidity or foolhardiness or lack of athletic ability, and so on. How do we distinguish the problematic syndromal patterns that are disorders from the problematic syndromal patterns that are not disorders? For that matter, however one defines the threshold for having a syndrome, there are probably counterexamples of disorders that do not manifest themselves as syndromes of co-occurring symptoms and predictable course, so how does one encompass them within the syndromal definition unless the definition is itself vacuous and the criterion circular? The most adequate answer to these questions, I believe, is that we consider a problematic syndromally (or nonsyndromally)-defined condition to be a disorder when we believe that the condition is caused by a dysfunction in the sense identified by the HD analysis. Without this additional implicit requirement, the syndromal approach offers no cogent distinction between disorder and nondisorder.

Strengths and Weaknesses of DSM-IV's Definition of Mental Disorder

To understand the potential sources of DSM-IV's false positives, it is useful to start by examining its own definition of mental disorder, which reads in DSM-IV-TR as follows:

> In DSM-IV, each of the mental disorders is conceptualized as a clinically significant behavioral or psychological syndrome or pattern that occurs in an individual and that is associated with present distress (e.g., a painful symptom) or disability (i.e., impairment in one or more important areas of functioning) or with a significantly increased risk of suffering death, pain, disability, or an important loss of freedom. In addition, this syndrome or pattern must not be merely an expectable and culturally sanctioned response to a particular event, for example, the death of a loved one. Whatever its original cause, it must currently be considered a manifestation of a behavioral, psychological, or biological dysfunction in the individual. Neither deviant behavior (e.g., political, religious, or sexual) nor conflicts that are primarily between the individual and society are mental disorders unless the deviance or conflict is a symptom of a dysfunction in the individual, as described above. (American Psychiatric Association, 2000, p. xxxi)

This definition of mental disorder is derived from an extended analysis of the concept of mental disorder provided by Spitzer and Endicott (1978). The analysis and critique of DSM-IV's definition of mental disorder has been pursued in several articles (Wakefield, 1992a, 1992b, 1993, 1996, 1997), and that material is not repeated here. Rather, on the basis of that work, I summarize a few major strengths and weaknesses of the definition as a prelude to proposing a revised analysis.

Regarding strengths, the definition makes four points. First, disorder is something in the individual; it is not simply a bad relationship or poor role performance. Second, the internal condition, which must be inferred to exist from manifest symptoms, is a dysfunction; that is, something must have gone wrong with the way that the internal mechanism normally functions (Klein, 1978; Spitzer & Endicott, 1978). Many problematic internal states, such as ignorance, lack of skill, lack of talent, sadness due to a loss, and foolhardiness, are not disorders in the medical sense because they are not dysfunctions. As noted, in the case of mental disorders, the dysfunctional mechanism must be a cognitive, motivational, behavioral, emotional, or other psychological mechanism. ("Mechanism" is used here without any mechanistic implications about the nature of the mind, but simply as a term commonly used in the evolutionary literature to refer to any inner process or structure.)

Third, a dysfunction of some internal mechanism is not enough by itself to imply disorder. Many things that go wrong with various mental and physical mechanisms do not deserve to be called disorders because they do not have sufficiently negative implications for the individual's overall well-being. The difference between dysfunctions that can be classified as disorders and dysfunctions that cannot be classified as disorders thus lies in whether the dysfunction causes significant harm to the person.

Finally, a strength is that the definition cautions that the distress or disability must come about due to a dysfunction and cannot be due only to social deviance, disapproval by others, or conflict with society or with others. This requirement is meant to preclude the misuse of psychiatry for sheer social control purposes, as occurred in Soviet psychiatry.

Regarding the definition's weaknesses, first, the use of the phrase "clinically significant" is circular in a definition of disorder because the decision as to whether or not a syndrome or pattern is clinically significant depends on whether it is considered a disorder. Second, the list of possible harmful effects, which has grown through successive editions of DSM, has become unwieldy. Clauses have been added to take care of specific problem categories, so the list has an ad hoc quality. There is the sense that it is only a matter of time until yet further clauses must be added. The fact is that any significant harm directly caused by a dysfunction will qualify the dysfunction as a disorder.

There are also problems with defining the concept of "disability" as "impaired function." If this includes impaired function of an internal mechanism, it leads to counterexamples because, for example, a specific gene can be impaired and dysfunctional without causing a disorder. There is also a general problem of distinguishing normal variation in ability from pathological disability; for example, inability to excel at sports is not necessarily a disability. The notion of disability seems itself to depend on a prior understanding of function and dysfunction (Wakefield, 1993).

The most serious problem, however—and the one underlying most of the other quibbles above—is that there is no explanation or analysis of the critical concept "dysfunction." Any definition of "disorder" in terms of the closely related concept "dysfunction" is inadequate unless "dysfunction" gets some independent analysis. DSM-IV's definition does require that a disorder cannot be an expectable or socially sanctioned response to events, and this is perhaps the closest it comes to attempting to offer an explication of dysfunction. However, "dysfunction" diverges from both "unexpectable" and "culturally unsanctioned" functioning. Much culturally unsanctioned functioning is not necessarily disordered, ranging from bad manners and petty crime to defiance of social conventions or civil disobedience based on high moral principles (ranging from Vietnam War protesters in the United States to the prototypical case of the inappropriate diagnosis of the Soviet dissidents). Moreover, normal reactions to external stresses (e.g., grief, terror) can be unexpectable (in a statistical sense) and harmful, and nondysfunctional internal conditions (e.g., illiteracy, greediness, or slovenliness) can be unexpectable and harmful, yet not disorders. Conversely, some conditions that are dysfunctions can be quite expectable in context, such as PTSD after a severe trauma. Indeed, most of the conditions included in the V Code chapter of DSM-IV (e.g., family or occupational conflict) are unexpectable and harmful, but are not disorders. Thus

the definition does not adequately operationalize "dysfunction."

Although the DSM definition of disorder has some limitations, the false positives to be found in the DSM are not due primarily to a faulty definition of disorder. This is because the strengths of the definition, and in particular the reference to dysfunction, would be enough (if followed up rigorously and properly elaborated) to eliminate many false positives. Rather, the definition's key requirement of dysfunction remains unelaborated—and, when elaborated by the HD analysis, it turns out that criteria sets do not meet the criteria for disorder set by the definition. This disparity between the definition and the criteria sets is the source of most of the false positives (Wakefield, 1997).

Clinical Significance

Beyond the definition of mental disorder, the most notable attempt in DSM-IV to make general progress on the false-positives problem has been the development of a clinical significance criterion (CSC) for use in evaluating mental disorder—basically transferring the clinical significance feature from the definition of mental disorder to the criteria sets for specific disorders. The CSC proposed in DSM-IV required that the symptoms cause either clinically significant distress or impairment in social, occupational, or other important areas of functioning. (Note that such impairment in role functioning as socially defined is not in and of itself equivalent to "dysfunction" in the sense of a failure of a biologically shaped function; many normal states, such as grief and sleep, impair role functioning in this sense.) The CSC aims to set an impairment/distress threshold for diagnosis, so as to eliminate false positives where there is minimal harm to the individual, and in a few instances it does improve validity. However, requiring "clinically significant" distress or role impairment as a criterion for distinguishing disorder from nondisorder is circular because the amount of distress or impairment varies greatly with both normal and disordered negative conditions. Thus, as a criterion for disorder, to say that distress or impairment is "clinically significant" in this context can only mean that the distress or impairment is significant enough to imply the existence of a disorder—a tautologous criterion. The phrase offers no real guidance in deciding whether the level of impairment is or is not sufficient to imply disorder.

Furthermore, the CSC does not deal with a large number of potential false positives—specifically, those where there may be harm but no dysfunction. For example, the normal child in a threatening environment whose aggressive behavior meets the criteria for conduct disorder, and the normal child threatened by a school bully who meets the selective mutism criteria by virtue of not speaking at school because of fear, do both experience distress and significant impairment in functioning as part of their normal reactions and so are not excluded from diagnosis by the CSC. Although there is obviously a thorny issue lurking here about the point at which motivations become so intense and rigid as to be pathological, it seems clear that in some cases such motivations (e.g., the motivation to avoid talking so as to avoid being beaten up by a bully) can be perfectly normal, even though they impair performance and are distressful. Thus clinical significance by itself does not imply disorder.

Another problem with the CSC is the potential for yielding false-negative misdiagnoses when it is indiscriminately added to criteria sets because it requires such specific forms of harm. For example, DSM-IV's requirement that a pattern of substance use must cause clinically significant impairment or distress before dependence can be diagnosed could yield large numbers of false negatives. It is not uncommon to encounter individuals whose health is threatened by drug addiction (and who surely have a disorder), but who are not distressed and who can carry on successful role functioning. The proverbial successful stockbroker with a cocaine addiction may be an instance, as may be many people who use tobacco. The problem in many of these cases is that distress and role impairment are not the only kinds of harms that can be caused by dysfunctions. However, in principle, the latter kinds of problems with the CSC could be addressed simply by requiring "significant harm."

Perhaps the most problematic aspect of the CSC is that it reflects a misunderstanding of the main problem underlying false positives, and thus a misdirection of effort. The CSC

is based on the assumption that the way to ensure that a condition is pathological is to ensure that it causes sufficient distress or impairment in social or role functioning—an assumption at odds with broader diagnostic practice in medicine. Moreover, DSM-based false positives are most often due not to a failure of symptoms to reach a threshold of harmfulness, but to a failure of symptomatic criteria to indicate the presence of an underlying dysfunction. Thus ratcheting up the level of harm such as distress or impairment is not sufficient for distinguishing disorder from nondisorder. There are two good indicators of this failure in DSM itself. The first is that the most obvious potential false positive in the manual—uncomplicated bereavement as distinguished from major depressive disorder—must be dealt with in an additional special exclusion clause and is not eliminated by the CSC in the criteria set because normal grief can be just as distressful and role-impairing as pathological depression, even though it is not caused by a dysfunction. The second indicator is that although the CSC is added to the criteria for conduct disorder (see above), DSM still adds a textual note that adolescents may satisfy the criteria and still not be disordered because their antisocial behavior may not be due to a dysfunction but to a normal reaction to a problematic environment. Clearly, the CSC does not address the dysfunction problem. Both distress and role impairment can result from normal or disordered emotions and behaviors. Wakefield and Spitzer (2002b) suggest that the issue of false positives in diagnosis is better approached by examining the context of the particular symptoms and adjusting the details of the diagnostic criteria.

The HD Analysis of the Concept of Mental Disorder

The HD analysis departs from three observations. First, the concept of "disorder" has been around in physical medicine and applied to some mental conditions for millennia and is broadly understood in a shared way by laypeople and professionals. Second, a central goal of an analysis of "mental disorder" is to clarify and reveal the degree of legitimacy in psychiatry's claims to be a truly medical discipline rather than, as antipsychiatrists and others have claimed, a social control institution masquerading as a medical discipline. Third, there are strong widely held intuitions that diagnosis can be misapplied and even abused when applied for social control purposes to mental conditions socially considered negative (as in the case of the Soviet dissidents), and thus it appears that there is more to the concept of mental disorder than just social values.

The approach to defining "mental disorder" that is suggested by the first observation is a conceptual analysis of the existing meaning of "disorder" as it is generally understood in medicine and society in general, with a focus on whether and how this concept applies to the mental domain. The challenge is to forge an analysis that is consistent with this shared meaning. The claim of psychiatry to be a medical discipline depends on there being genuine mental disorders in the same sense of "disorder" that is used in physical medicine, so the analysis must in fact rise to the challenge of providing an analysis of the generic concept of disorder in order to establish that mental conditions can fall under it. Any proposal to define "mental disorder" in a way unique to psychiatry that does not fall under the broader medical concept of disorder would fail to address the issue of the medical nature of psychiatry. Finally, the possibility that even an entire culture may be mistaken about disorder, as well as the fact that there are many negative conditions not considered medical disorders, suggests that the concept has at least some factual or objective component that is more than simply a value judgment. The challenge here is to explain the nature of this factual component. The HD analysis is aimed at addressing these challenges.

The Value Component of "Disorder"

As traditional value-based accounts suggest, a condition is a mental disorder only if it is harmful according to social values and thus at least potentially warrants medical attention. Medicine in general, and psychiatry in particular, are irrevocably value-based professions. "Harm" is construed broadly here to include all negative conditions.

Both lay and professional classificatory behaviors demonstrate that the concept of

mental disorder contains a value component. For example, inability to learn to read due to a dysfunction in the corpus callosum (I am assuming that this theory of some forms of dyslexia is correct) is harmful in literate societies but not harmful in preliterate societies, where reading is not a skill that is taught or valued, and thus this dysfunction is not a disorder in those societies. Most people have what physicians call "benign anomalies"—that is, minor malformations that are the result of genetic or developmental errors but that cause no significant problem—and such anomalies are not considered disorders. For example, benign angiomas are small blood vessels whose growth has gone awry, leading them to connect to the skin; however, because they are not harmful, they are not considered disorders. The requirement that there be harm also accounts for why simple albinism, heart position reversal, and fused toes are not generally considered disorders, even though each results from an abnormal breakdown in the way some mechanism is designed to function. Purely scientific accounts of "disorder," even those based on evolutionary function as in the analysis below (e.g., Boorse, 1975, 1976), fail to address this value component.

In the DSM and ICD diagnostic criteria, the symptoms and clinical significance requirement generally ensure that the condition causes harm and is negatively valued. The dispute remains about whether "mental disorder" is purely evaluative or contains a significant factual component that can discriminate a potential domain of negative conditions that are disorders from those that are nondisorders. Many negative conditions are not disorders, and many of them contain symptoms and are clinically significant in that they cause distress or role impairment (e.g., grief). The distinction between disorders and nondisorders thus seems to depend on some further criterion.

The Factual Component of "Disorder" as Failure of Naturally Selected Functions

Contrary to those who maintain that a mental disorder is simply a socially disapproved mental condition (e.g., Houts, 2001; Sedgwick, 1982), "mental disorder" as commonly used is just one category of the many negative mental conditions that can afflict a person. We need an additional factual component to distinguish disorders from the many other negative mental conditions not considered disorders, such as ignorance, lack of skill, lack of talent, low intelligence, illiteracy, criminality, bad manners, foolishness, and moral weakness.

Indeed, both professionals and laypersons distinguish between quite similar negative conditions as disorders versus nondisorders. For example, illiteracy is not in itself considered a disorder, even though it is disvalued and harmful in Western society, but a similar condition that is believed to be due to lack of ability to learn to read because of some internal neurological flaw or psychological inhibition is considered a disorder. Male inclinations to aggressiveness and to sexual infidelity are considered negative but are not generally considered disorders because they are seen as the result of natural functioning, although similar compulsive motivational conditions are seen as disorders. Grief is seen as normal, whereas similarly intense sadness not triggered by real loss is seen as disordered. A purely value-based account of "disorder" does not explain such distinctions among negative conditions.

Moreover, we often adjust our views of disorder according to cross-cultural evidence that may go against our values. For example, U.S. culture does not value polygamy, but Americans judge that it is not a failure of natural functioning and thus not a disorder, partly on the basis of cross-cultural data.

The challenge, then, is to elucidate the factual component. Based on common usage in the literature, I call this factual component a "dysfunction." What, then, is a dysfunction? An obvious place to begin is with the supposition that a dysfunction implies an unfulfilled function—that is, a failure of some mechanism in the organism to perform its function. However, not all uses of "function" and "dysfunction" are relevant. The medically relevant sense of "dysfunction" is clearly *not* the colloquial sense in which the term refers to an individual's failure to perform well in a social role or in a given environment, as in assertions like "I'm in a dysfunctional relationship" or "Discomfort with hierarchical power structures is dysfunctional in today's corporate environment." These kinds of problems need not be

individual disorders. A disorder is different from a failure to function in a socially or personally preferred manner, precisely because a dysfunction exists only when something has gone wrong with functioning, so that a mechanism cannot perform as it is naturally (i.e., independently of human intentions) supposed to perform.

Presumably, then, the functions that are relevant are "natural" or "biological" functions. Such functions are frequently attributed to inferred mental mechanisms that may remain to be identified, and failures are labeled dysfunctions. For example, a natural function of the perceptual apparatus is to convey roughly accurate information about the immediate environment, so gross hallucinations indicate dysfunction. Some cognitive mechanisms have the function of providing a person with the capacity for a degree of rationality as expressed in deductive, inductive, and means–end reasoning, so it is a dysfunction when the capacity for such reasoning breaks down, as in severe psychotic states.

The function of a mechanism is important because of its distinctive form of explanatory power; the existence and structure of the mechanism is explained by reference to the mechanism's effects. For example, the heart's effect of pumping the blood is also part of the heart's explanation, in that one can legitimately answer a question like "Why do we have hearts?" or "Why do hearts exist?" with "Because hearts pump the blood." The effect of pumping the blood also enters into explanations of the detailed structure and activity of the heart. Talk of "design" and "purpose" in the case of naturally occurring mechanisms is just a metaphorical way of referring to this unique explanatory property that the effects of a mechanism explain the mechanism. So "natural function" can be analyzed as follows: A natural function of an organ or other mechanism is an effect of the organ or mechanism that enters into an explanation of the existence, structure, or activity of the organ or mechanism. A "dysfunction" exists when an internal mechanism is unable to perform one of its natural functions. (This is only a first approximation to a full analysis; there are additional issues in the analysis of "function" that cannot be dealt with here. See Wakefield, 2000a, 2000b, 2005a.)

The analysis above applies equally well to the natural functions of mental mechanisms. Like artifacts and organs, mental mechanisms, such as cognitive, linguistic, perceptual, affective, and motivational mechanisms, have such strikingly beneficial effects and depend on such complex and harmonious interactions that the effects cannot be entirely accidental. Thus functional explanations of mental mechanisms are sometimes justified by what we know about how people manage to survive and reproduce. For example, a function of linguistic mechanisms is to provide a capacity for communication; a function of the fear response is to avoid danger; and a function of tiredness is to bring about rest and sleep. These functional explanations yield ascriptions of dysfunctions when respective mechanisms fail to perform their functions, as in aphasia, phobia, and insomnia.

"Dysfunction" is thus a purely factual scientific concept. However, discovering what in fact is natural or dysfunctional (and thus what is disordered) may be difficult and may be subject to scientific controversy—especially with respect to mental mechanisms, about which we are still largely ignorant. This ignorance is part of the reason for the high degree of confusion and controversy concerning which conditions are really mental disorders. However, functional explanations can be plausible and useful even when little is known about the actual nature of a mechanism or even about the nature of a function. For example, we know little about the mechanisms underlying sleep, and little about the functions of sleep, but circumstantial evidence persuades us that sleep is a normal, biologically designed phenomenon and not (despite the fact that it incapacitates us for roughly one-third of our lives) a disorder; the circumstantial evidence enables us to distinguish some normal versus disordered conditions related to sleep, despite our ignorance.

Obviously, one can go wrong in such explanatory attempts; what seems nonaccidental may turn out to be accidental. Moreover, cultural preconceptions may easily influence one's judgment about what is biologically natural. But often one is right, and one is making a factual claim that can be defeated by evidence. Functional explanatory hypotheses communicate complex knowledge that

may not be so easily and efficiently communicated in any other way.

Today, evolutionary theory provides a better explanation of how a mechanism's effects can explain the mechanism's presence and structure. In brief, those mechanisms with effects on the organism that contributed to the organism's reproductive success over enough generations thereby increased in frequency, and hence were "naturally selected" and exist in today's organisms. Thus an explanation of a mechanism in terms of its natural function may be considered a roundabout way of referring to a causal explanation in terms of natural selection. Since natural selection is the only known means by which an effect can explain a naturally occurring mechanism that provides it, evolutionary explanations presumably underlie all correct ascriptions of natural functions. Consequently, an evolutionary approach to mental functioning (Wakefield, 1999b, 2005a) is central to an understanding of psychopathology.

One might object that what goes wrong in disorders is sometimes a social function that has nothing to do with natural, universal categories. For example, reading disorders seem to be failures of a social function because there is nothing natural or designed about reading. However, illiteracy involves the very same kind of harm as reading disorder, but it is not considered a disorder. Inability to read is only considered indicative of disorder when circumstances suggest that the reason for the inability lies in a failure of some brain or psychological mechanism to perform its natural function. There are many failures of individuals to fulfill social functions, and they are not considered disorders unless they are attributed to a failed natural function.

If one looks down the list of disorders in DSM, it is apparent that by and large it is a list of the various ways that something can go wrong with what appear to be designed features of the mind. Very roughly, psychotic disorders involve failures of thought processes to work as designed; anxiety disorders involve failures of anxiety- and fear-generating mechanisms to work as designed; depressive disorders involve failures of mechanisms regulating sadness and responses to loss; disruptive behavior disorders of children involve failures of socialization processes and processes underlying conscience and social cooperation; sleep disorders involve failures of sleep processes to function properly; sexual dysfunctions involve failures of various mechanism involved in sexual motivation and response; eating disorders involve failures of appetitive mechanisms; and so on. There is a certain amount of nonsense in DSM, and criteria are often overly inclusive. However, the vast majority of categories are inspired by conditions that even a layperson would correctly recognize as failures of designed functioning.

It is clear that we need not know either the natural function of a response (or set of responses) or the specific nature of the mechanisms underlying the response to judge from circumstantial evidence that the response is likely to be a designed feature of the mind and its failure a dysfunction. Take, for example, sleep. We scientifically understand neither the precise functions of sleep nor the mechanisms that trigger and regulate sleep; yet almost everyone recognizes sleep as a natural response that must be the effect of some set of mechanisms that were partly selected because they yield sleep. Of course, this could turn out to be incorrect, however unlikely it may seem. Moreover, although there is certainly a large fuzzy area, we do recognize that a certain degree of deviation in sleep pattern toward either insomnia or hypersomnia, if not for obvious temporary normal reasons (e.g., noise, exhaustion), probably indicates a disorder.

When we distinguish normal grief from pathological depression, or normal delinquent behavior from conduct disorder, or normal criminality from antisocial personality disorder, or normal unhappiness from adjustment disorder, or illiteracy from reading disorder, we are implicitly using the "failure-of-designed-function" criterion. All of these conditions—normal and abnormal—are disvalued and harmful conditions, and the effects of the normal and pathological conditions can be quite similar behaviorally; yet some are considered pathological and some not. The natural-function criterion explains these distinctions.

It bears emphasis that even biological conditions that are harmful in the current environment are not considered disorders if they are considered designed features. For example, the taste preference for fat is not

considered a disorder, even though in today's food-rich environment it may kill you, because it is considered a designed feature that helped our ancestors to obtain needed calories in a previous food-scarce environment. Higher average male aggressiveness is not considered a mass disorder of men even though in today's society it is arguably harmful because it is considered the way men are designed (of course, there are aggressiveness disorders; here as elsewhere, individuals may have disordered responses of designed features).

In sum, a mental disorder is a harmful mental dysfunction. If the HD analysis is correct, then a society's categories of mental disorder offer two pieces of information. First, they indicate a value judgment that the society considers the condition negative or harmful. Second, they make the factual claim that the harm is due to a failure of the mind to work as designed. This claim may be correct or incorrect, but in any event it reveals what the society thinks about the natural or designed working of the human mind.

The Epistemological Objection

One of the most common objections to the HD analysis is that we just don't know the evolutionary history of the features of the human mind. The argument is that because the HD analysis holds that whether one has a disorder depends on facts about internal mechanisms and their evolutionary history, and we are largely ignorant of these facts, the analysis therefore implies that it is impossible to know at this time whether conditions are disorders or nondisorders. This might be called the "epistemological" objection because it concerns limits in our ability to know about and recognize disorders, rather than the logic of the analysis of disorder itself.

The epistemological objection is based on the assumption that to know that there is a dysfunction, one must know the dysfunctional mechanisms and their evolutionary history. This assumption is false. To know that a dysfunction exists, one need only have sufficient indirect evidence—for example, surface evidence that indicates or correlates with the existence of internal dysfunction—to infer that some mechanism is failing to perform as designed. This is all that the definition of "dysfunction" I have given earlier actually implies.

What does follow from my account, then, is that to attribute disorder, *either* one has to know about the mechanisms and evolution, *or* one has to have indirect evidence allowing one to judge that there is a dysfunction without knowing the internal or evolutionary details. At present, the latter, indirect method is used in most cases to judge when there is a disorder. In particular, for most DSM-IV categories, the indirect method is enough to enable us to make plausible judgments about whether problematic conditions stem from dysfunctions and are thus disorders. We do not have to know the details of evolution or of internal mechanisms to know, for example, that typical cases of thought disorder, drug dependence, mood disorders, sexual dysfunction, anxiety disorders, and so on, are failures of some mechanisms to perform their designed functions; it is obvious from surface features. This is what makes a symptom-based diagnostic system possible, to the extent that it is possible (see Wakefield, 1996, for a discussion of the limits of DSM-IV's symptom-based criteria in picking out dysfunctions, and DSM-IV's resulting overinclusiveness). My claim is that in routinely distinguishing normal suffering from mental disorder, people are implicitly making the sort of distinction specified in the HD analysis. If the distinction is ignored, then conceptual confusion is likely to follow.

Let me use an analogy from artifact functions to illustrate the logic of such indirect evidence. When my automobile does not work, there can be various explanations. The problem could be due to a variety of circumstances that do not indicate that there is anything wrong with the car itself; for example, the gas tank could be empty, the ignition might not turn the engine on because the car's transmission is not in "park," there could be objects blocking the wheels, and so on. However, if such circumstantial causes are eliminated, then I can often reasonably infer that the problem is due to the fact that some internal mechanism is malfunctioning—that is, is not doing what it was designed to do.

Suppose that I am in a state of great ignorance about the mechanisms that make up my car and how they are designed, and have

no idea what mechanism is malfunctioning. The epistemological objection suggests that I would not be justified in inferring an automobile malfunction, and that such judgments must be deferred until I learn about the parts of my automobile and their design. But we know that in the case of the automobile, something is wrong with this objection. I know almost nothing about the design of automobiles, but I am perfectly capable of recognizing many cases of automotive malfunction, and regularly discriminate such cases from proper automotive functioning. How do I perform this feat, which the objection claims to be impossible?

Although I do not understand a car's internal parts and have no idea of the history or details of their design or their immediate functions, some functions of those parts are obvious because of the clearly nonaccidental benefits that accrue from them. For example, it cannot be accidental that cars take people from place to place with great ease and efficiency; it cannot be accidental that lights allow people to drive cars safely at night; it cannot be accidental that the gas gauge generally indicates the amount of gas in the car; and so on. For complex reasons, these sorts of beneficial, designed phenomena are often recognizable, and their failures can often be recognized as malfunctions, even if one does not know how the underlying mechanisms work or even what they are. Thus I know that something is wrong with the car's internal mechanisms when the car will not move after I have filled it with gas, turned on the engine, put it into gear, and eliminated other circumstantial causes as noted above; when the lights do not go on after I turn the light switch to "on"; or when the gas gauge stays on "empty" no matter how much gas I put in the tank. Even if I do not know about pistons or their immediate designed functions in engine performance, I do know that when my car is backfiring and unable to go over 35 miles per hour, something is wrong with some mechanism. Once I conclude from the observation of nonaccidental surface features that unknown internal mechanisms must be designed to accomplish certain surface functions, I can often infer the existence of internal malfunctions from observations of the failures of those surface functions. These inferences can certainly go wrong and lead to false conclusions. But they often lead to correct conclusions, and they are the same sort of inferences that were at the foundation of physical medicine until a few hundred years ago, when knowledge of internal mechanisms and disorder etiologies exploded. Until then, the judgment that, for example, blindness or paralysis were disorders involved the same kinds of inferences without any knowledge of internal mechanisms.

The HD analysis implies that exploration of mental mechanisms and their functions and dysfunctions is essential if we are to understand mental disorders fully and treat them effectively in the long run. The HD analysis thus provides a useful framework for understanding psychiatric research; we want to identify the specific dysfunctions in the specific internal mechanisms that correspond to each mental disorder. But while we are waiting for such knowledge, the HD analysis explains why we can often make the valid judgments we do, and it illuminates the indirect evidence on which such judgments are based.

Implications of the HD Analysis for Validity of Diagnostic Criteria

One of the disadvantages of pure social-constructivist and antipsychiatric views of mental disorder is that, in rejecting the very notion of mental disorder as a coherent medical-scientific concept, they offer us no place to stand from which to critique current diagnostic criteria and to improve their validity. In contrast, the HD analysis allows us to identify criteria that are too broad and that incorrectly include normal reactions under the "disorder" category. Here are some brief examples of problematic criteria.[1]

Major Depressive Disorder

The DSM-IV criteria for major depressive disorder contain an exclusion for uncomplicated bereavement (up to 2 months of symptoms after loss of a loved one are allowed as normal), but no exclusions for equally normal reactions to other major losses, such as a terminal medical diagnosis in oneself or a loved one, separation from one's spouse, the end of an intense love affair, or loss of one's job and retirement fund. Reactions to such

losses may satisfy DSM-IV diagnostic criteria but are not necessarily disorders. If one's reaction to such a loss includes, for example, just 2 weeks of depressed mood, diminished pleasure in usual activities, insomnia, fatigue, and diminished ability to concentrate on work tasks, then one's reaction satisfies DSM-IV criteria for major depressive disorder, even though such a reaction need not imply pathology any more than it does in bereavement. Clearly, the essential requirement that there be a dysfunction in a depressive disorder—perhaps one in which loss response mechanisms are not responding proportionately to loss as designed—is not adequately captured by DSM-IV criteria (Horwitz & Wakefield, 2007; Wakefield, Schmitz, First, & Horwitz, 2007).

Note that the argument here is *not* that "reactive" depressions are not disorders; the "disorder–nondisorder" distinction should not be confused with the traditional "reactive–endogenous" distinction. Some reactive depressions represent proportionate, designed responses to environmental events that do not involve any internal dysfunction, and are not disorders. But other reactions to loss can be of such disproportionate intensity or duration, or can involve such extreme symptoms (e.g., suicidal behavior or severe psychomotor retardation) out of the list of possible symptoms allowable by DSM-IV, as to imply the probability of a breakdown in the designed, adaptive functioning of loss response mechanisms. Thus many reactive depressions that fall under DSM-IV criteria are indeed disorders. This point is recognized by DSM-IV in the concept of "complicated" or disordered bereavement, and the same logic holds for other "complicated" loss reactions. In such cases, the triggering environmental event interacting with other characteristics of the individual or environment causes enduring harmful dysfunction in an internal mechanism, yielding a disorder. So the argument here is not that reactive depression is not a disorder, but rather that among reactive depressions to a variety of losses, some are disorders and some are not, and DSM-IV criteria do not adequately distinguish the disordered ("complicated") reactions from the normal ("uncomplicated") ones.

Because of these flaws, the epidemiological data on prevalence of depression can be misleading, yielding potentially inflated estimates of the social and economic costs of depression. Based on international epidemiological studies using symptom-based criteria, the WHO has publicized the apparently immense costs of depression. However, the claimed enormity of this burden relative to other serious diseases, and the consequent influence on priorities, may result in part from the failure to distinguish depressive disorders from intense normal sadness. The epidemiological studies encompass everyone who meets symptom criteria—a group that, due to the possible confounding of normal sadness with disorder, may be heterogeneous to a greater degree than clinical patients would indicate, yielding an invalid overall estimation of disease burden. Unraveling these confusions could lead to a more optimal distribution of the WHO's health resources.

Conduct Disorder

The DSM-IV diagnostic criteria for conduct disorder allow adolescents who are responding with antisocial behavior to peer pressure, threatening environment, or abuses at home to be diagnosed with this disorder. For example, if a girl, attempting to avoid escalating sexual abuse by her stepfather, lies to her parents about her whereabouts and often stays out late at night despite their prohibitions—and then, tired during the day, often skips school, and her academic functioning is consequently impaired—she can be diagnosed as having conduct disorder. Rebellious kids or kids who fall in with the wrong crowd, skip school, and repetitively engage in shoplifting and vandalism also qualify for diagnosis. Regrettably, Tom Sawyer and Huck Finn fare no better diagnostically under DSM-IV criteria (Richters & Cicchetti, 1993). Such conditions are not necessarily disorders, as laypersons and professionals agree (Pottick, Kirk, Hsieh, & Tian, 2007; Wakefield, Kirk, Pottick, Tian, & Hsieh, 2006; Wakefield, Pottick, & Kirk, 2002).

However, in an acknowledgment of such problems, this statement is included in the "Specific Culture, Age, and Gender Features" section of the DSM-IV-TR text for conduct disorder: "Consistent with the DSM-IV definition of mental disorder, the Conduct Disorder diagnosis should be applied only when the behavior in question is symptomatic of an

underlying dysfunction within the individual and not simply a reaction to the immediate social context" (American Psychiatric Association, 2000, p. 96). If these ideas had been incorporated into the diagnostic criteria, many false positives could have been eliminated. Unfortunately, in epidemiological and research contexts, such textual nuances are likely to be ignored.

The problem evident with the diagnostic criteria for conduct disorder is that because they rely on specific socially disapproved behaviors and eliminate traditional psychopathy indicators (such as lack of empathy and failure to develop moral conscience), the criteria do not adequately distinguish criminality and normal delinquency from genuine mental disorder. The same problem afflicts the criteria for antisocial personality disorder (see below).

Separation Anxiety Disorder

Separation anxiety disorder is diagnosed in children on the basis of symptoms indicating age-inappropriate, excessive anxiety concerning separation from home or from those to whom a child is attached, lasting at least 4 weeks. The symptoms (e.g., excessive distress when separation occurs, worry that some event will lead to separation, refusal to go to school because of fear of separation, reluctance to be alone or without a major attachment figure) are just the sorts of things children experience when they have a normal, intense separation anxiety response. The criteria do not distinguish between a true disorder, in which separation responses are triggered inappropriately, and normal responses to perceived threats to a child's primary bond (due to an unreliable caregiver or other serious disruptions). For example, in a study of children of military personnel at three bases that happened to occur at the time of Operation Desert Storm—when many parents of the children were in fact leaving for the Middle East, and when children knew other children with parents who had been killed or injured—the level of separation anxiety was high enough among many of the children for them to qualify as having separation anxiety disorder according to DSM standards. In fact, however, they were responding with a normal-range separation response to an unusual environment in which they had realistic concerns that their parents would not come back (A. M. Brannan, personal communication, 1998).

Paraphilias (and "Sexual Predator" Laws)

In some states in the United States, a repeat sexual offender who is considered to have a mental disorder in the form of a sexual paraphilia that places his actions out of his control, and who may therefore be a threat to children or adults in the community through repetition of his criminal acts, may be classified as a "sexual predator" and detained through civil commitment after a hearing, even after the individual has fully served a prison term for a sexual crime. In such hearings, the diagnosis of mental disorder is crucial to continued detention of the individual. Often judgments about the presence of a paraphilia are made even when the proposed category is not specifically included in DSM; the DSM's residual category of sexual disorders NOS is used in such cases. However, conditions that are socially disapproved and that lead to illegal actions are not the same as mental disorders, yet may be cited in such hearings. For example, a man who has sexual relations with several underage females may be acting illegally and immorally, but it is unlikely that in virtue of those sexual acts alone he has a mental disorder. Attraction to teenage and even preteen females by males seems common across cultures; has evolutionary roots, in all likelihood; and indeed emerges in such facts as that during certain periods in some countries, a large proportion of prostitutes were (and sometimes still are) preteens.

Social Phobia

Whereas social phobia is a real disorder in which people can sometimes be incapable of engaging in the most routine social interactions, the current diagnostic criteria allow diagnosis when someone is, say, intensely anxious about public speaking in front of strangers. But it remains unclear whether such fear is really a failure of normal functioning or rather an expression of normal-range danger signals that were adaptive in the past, when failure in such situations could lead to ejection from the group and a

consequent threat to survival. This diagnosis seems potentially an expression of North American society's high need for people who can engage in occupations that require communicating to large groups (Wakefield, Horwitz, & Schmitz, 2005a, 2005b).

Adjustment Disorder

Adjustment disorder is defined in terms of a reaction to an identifiable stressor that either (1) causes marked distress that is in excess of what would be expected from exposure to the stressor; or (2) significantly impairs academic, occupational, or social functioning. The first clause allows the top third, say, of the normal distribution of reactivity to stress to be diagnosed as disordered, and it does not take into account the contextual factors that may provide good reasons for one person to react more intensely than others. The second criterion classifies as disordered any normal reaction to adversity that temporarily impairs functioning (e.g., one does not want to socialize, or one does not feel up to going to work). Here, too, the criteria contain an exclusion for bereavement but not for other equally normal reactions to misfortune.

Antisocial Personality Disorder

The criteria for antisocial personality disorder fail to distinguish some instances of sheer criminal behavior from this disorder. Traditionally, this distinction was made by requiring that an antisocial mental disorder must involve a dysfunction in one of the mechanisms that usually inhibit such behavior, such as those providing the capacity for guilt, anxiety, remorse, learning from mistakes, or capacity for loyalty. DSM-III and DSM-III-R (American Psychiatric Association, 1987) criteria failed to make this distinction adequately. Allen Frances, the Chairperson of the Task Force on DSM-IV, said the following in 1980 about DSM-III criteria for antisocial personality disorder:

> The DSM-III diagnostic criteria specifying antisocial personality are indeed clear and reliable . . . but they may have missed the most important clinical point. Using criteria comparable to those in DSM-III, approximately 80% of all criminals are diagnosed as antisocial. . . . It would seem to be more useful to have criteria that distinguish those criminals who are capable of loyalty, anxiety, and guilt from those who are not. . . . There was also considerable concern that the DSM-III criteria would be too easily and universally attained by individuals growing up in rough and deprived areas. (Frances, 1980, p. 1053)

The DSM-III criteria about which Frances wrote were as follows. In addition to having shown evidence of conduct disorder before age 15, an adult must meet four or more of the following criteria: inconsistent work (e.g., 6 months of unemployment in 5 years); irresponsible parenting; breaking the law (e.g., selling drugs, repeated arrests); lack of an enduring sexual relationship (e.g., two divorces or separations, 10 or more sexual partners in a year); irritability and aggressiveness; failure to honor financial obligations; impulsivity (e.g., moving without a prearranged job); deceitfulness; and recklessness (e.g., driving while intoxicated). Frances was surely right that these are invalid criteria for picking out those who have antisocial personality disorder from nondisordered criminals and others, for a host of face validity reasons.

DSM-IV criteria for antisocial personality disorder are essentially the same as in DSM-III, except for the following changes: DSM-IV combines the work and finance criteria into one "either–or" criterion; removes the criteria related to parenting and an enduring sexual relationship; has an additional "lack of remorse" criterion (added in DSM-III-R); and requires three instead of four of the resulting seven criteria for diagnosis. These changes fail to address the main problem with the criteria—namely, that they do not distinguish between career criminals and persons with a mental disorder. The criminal certainly meets the illegal activity criterion and may well meet the work/finance criterion (criminal activity is not "work" as intended in this criterion); the deceit criterion (criminal careers often involve substantial deceit); and one or more of the impulsivity, irritability/aggressiveness, or recklessness criteria (by the very nature of criminal activity). The "remorse" criterion was added as a concession to the traditional approach to validity, but because only three criteria are necessary for diagnosis, the inclusion of a seventh "remorse" criterion does nothing to prevent false positives on the basis of three

out of the other six criteria. It thus appears that DSM-IV again "missed the most important clinical point." These flaws in the criteria for antisocial personality disorder are particularly of concern, not only because they create the possibility that psychiatrically normal criminals could mount a psychiatric defense—but, more importantly, because laws recently enacted in Great Britain allow detention beyond the jail term meted out in criminal proceedings of those offenders with antisocial personality disorder. Such laws make the overinclusiveness of the criteria potentially a tool of social control under a medical label.

Personality Disorder

Nor has DSM-IV fixed a more fundamental problem with its general definition of personality disorder, which guides the application of the frequently used personality disorder NOS category and provides the rationale for the more specific categories (Wakefield, 1989, 2006, 2008). The definition requires a pattern of inner experience and behavior that is (1) inflexible and pervasive across situations; (2) stable and of long duration and early onset (i.e., started at least as early as adolescence or early adulthood); (3) deviates markedly from the expectations of the person's culture; and, (4) causes significant distress or impairment. The first two of these criteria just describe what it is for a pattern of behavior to be considered a personality trait, whether normal or pathological. So a personality disorder is essentially defined as a personality trait that causes distress or impairment and is not what is expected in one's culture. The problem with this definition is that it covers a vast range of normal personality variations. For example, in our culture, a person of below-average intelligence may experience distress and will deviate from normative expectations; people who are foolish, selfish, nonconformist, or irreverent, and people who are creative and who experience distress or impairment from their creative birth pangs and deviate from social expectations in the pursuit of their muse, may all qualify as having personality disorders according to these criteria. Even the Soviet dissidents mentioned earlier in this chapter would probably have qualified for such a diagnosis because they inflexibly protested tyranny and thus deviated from what their society expected, experiencing distress and social impairment as a result. Closer to home, it has been noted that Martin Luther King—who, in heroically fighting for racial justice, persisted in violating the expectations of his local Southern culture and as a result sustained great distress and even death—would have to be considered to have a personality disorder, according to DSM-IV (Kalat, 1996, p. 597).

Disorder of Written Expression

An op-ed piece on DSM-IV in the *New York Times* by Stuart Kirk and Herb Kutchins (1994), which the *Times* titled "Is Bad Writing a Mental Disorder?", used disorder of written expression from the category of learning disorders as an example of the "nonsense" that is in the manual. Because DSM-IV criteria for this and other learning disorders require only that a child's achievement level be substantially below average, it is true that the criteria do not distinguish very bad penmanship from disorder. Clearly, a distinction is needed here: Bad penmanship is not a disorder in itself, but bad penmanship caused by a dysfunction in one of the mechanisms that enable children to learn to write is a disorder. (Kirk and Kutchins acknowledged that this distinction could be made.) This sort of distinction has been common in the learning disorders community for decades, and it requires that before diagnosing a child with a learning disorder, one must attempt to eliminate possible "normal" causes of the lack of achievement, such as family distractions, lack of motivation, or inability to understand the language of instruction. Because DSM-IV criteria fail to make this distinction, there is no adequate answer to Kirk and Kutchins's embarrassing question. In actuality, the problem is not with the category of disorder of written expression but with the invalid DSM-IV criteria. The danger is that when DSM-IV puts forward criteria that are open to such ridicule, the legitimate distinction can easily get lost. In the case of the learning disorders, this distinction is critical not only for diagnosis, but for the integrity and public support of special education programs.

The DSM-IV disorders usually first diagnosed in infancy, childhood, or adolescence

present several further examples of failure to address known problems. These disorders have been at the center of some of the most embarrassing public allegations about diagnostic invalidity—including charges that some normal children who do not keep up in school are classified as having learning disorders and inappropriately receive costly special education resources; that some normal but rambunctious children are diagnosed as having attention-deficit/hyperactivity disorder and are drugged into submission; and that some normal children who are difficult for their parents to handle are hospitalized under the diagnosis of oppositional defiant disorder. Given the special vulnerabilities of children, it is here, if anywhere, that the diagnostic expertise of the mental health professions should display itself by providing ways to distinguish such false positives from true disorders.

Substance Abuse

DSM-IV substance abuse roughly requires any one of four criteria: poor role performance at work or at home due to substance use; substance use in hazardous circumstances, such as driving under the influence of alcohol; recurrent substance-related legal problems; or social or interpersonal problems due to substance use, such as arguments with family members about this use. These criteria are not only face-invalid as indicators of disorder; they are also inconsistent with DSM-IV's own definition of mental disorder, which asserts that "symptoms" must not be due to conflict with society. Arrests for illegal activity and disapproval of family members are exactly the kinds of social conflicts that are insufficient for diagnosis of disorder according to DSM-IV's definition. It is remarkable that DSM-IV allows arguments with one's spouse about alcohol or drug use to be sufficient by itself for being diagnosed with substance abuse. In other words, if you drink or smoke marijuana, your spouse can now give you a mental disorder simply by arguing with you about it, and can cure you by becoming more tolerant! Given that parents are likely to argue with their children about even minor experiments with alcohol or drugs, this criterion is dangerously overinclusive indeed. Being arrested more than once for driving while under the influence of alcohol or for possession of marijuana is also sufficient for diagnosis, making one's diagnostic status depend on the diligence of the local police force and the vagaries of local drug laws. As to the "hazardousness" criterion, it is clear that very large numbers of people drive under the influence of alcohol for all kinds of foolish reasons, and that they need not have a mental disorder to do so.

Substance Dependence

The DSM-IV diagnosis of substance dependence is based on three or more symptoms from among those indicating either physiological dependence (tolerance or withdrawal) or psychological dependence (takes more than was intended; desires to cut down; great deal of time spent on getting, taking, and recovering from substance use; other activities reduced because of time spent on substance use; and use of substance is continued despite health risks). The problem is that DSM-IV's operationalization of psychological dependence consists of criteria that essentially operationalize the broader notion of "intense desire" or "strong preference," not the narrower class of pathological psychological addictions. Yet intense desires or strong preferences in themselves, whether for substances or for anything else, do not imply disorder. Consequently, it would appear that many persons who avidly use substances but who are not truly dependent on them would be misdiagnosed as disordered by these criteria. This flaw in the criteria could fuel the suspicion that the inclusiveness of the criteria reflect social disapproval and social goals of controlling substance use, rather than the logic of disorder.

This problem yielded public embarrassment for DSM-IV when the criteria for substance dependence were held up for public ridicule by defenders of the tobacco companies in debates about whether smokers suffer from a true addictive mental disorder. While it is clearly the case that many smokers are literally addicted, the tobacco companies scored easy points by arguing that if the DSM-IV criteria for substance dependence were applied to other domains of voluntary behavior, they would identify large numbers of preferred activities as psychological addictions and disorders. For example, an in-

dividual at risk for heart disease who desires and intends to cut fat intake down to 10% of calories in accordance with dietary guidelines, but makes it only to 20% because of his or her enjoyment of the taste of fat in foods, logically satisfies DSM-IV criteria for psychological substance dependence but would not seem to be disordered. Or a person who goes to bed without flossing his or her teeth more often than intended despite the health consequences, and wishes he or she would floss more often, satisfies the same criteria but is not disordered. Of course, these individuals are not technically diagnosable as having substance dependence—for the purpose of diagnosis, DSM-IV-TR defines "substance" as "a drug of abuse, a medication, or a toxin" (American Psychiatric Association, 2000, p. 191)—but the tobacco companies still had a point. The concept of psychological dependence should have the same logic, whether the object of desire is substance use or some other activity (and, in fact, the addition of gambling and other such behaviors as potential addictions or dependence conditions is under active discussion). Thus the fact that the hypothesized individuals' desires for eating fat and not flossing, respectively, are not considered disordered, whereas individuals who have exactly the same kind of relationship to their cigarette smoking are classified as disordered by DSM-IV, indicates the overinclusive nature of DSM-IV's operationalization of psychological substance dependence. DSM-IV criteria for substance dependence appear to overpathologize drug use by presupposing that all nondisordered desires are wholly ruled by reason; the criteria thus fail to distinguish truly disordered dependence from intense enjoyment, strong preference, and other nondisordered modes of drug use.

Bipolar II Disorder

The criteria for the new (to DSM-IV) category of bipolar II disorder require that at some point in life the individual must have suffered a major depressive episode and a hypomanic episode; a hypomanic episode is essentially a mild, short-lived (at least 4 days), and nonimpairing manic episode. Because of the mildness of the hypomanic symptoms, this category may be subject to many false positives. For example, a period of intense romantic involvement may satisfy the criteria for hypomanic episode if it lasts at least 4 days, and if the lover experiences an elevated, expansive mood during which he or she is three or more of the following: sexually indiscreet, distractible, physically active, talkative, high in self-esteem, lacking sleep, and having thoughts racing. If the lover is spurned and goes into a depressed tailspin for 2 weeks that satisfies the criteria for major depressive episode (or if the lover previously had a spell of sadness due to some serious loss that spuriously qualifies for disorder status under the criteria for major depressive disorder—see above), then he or she will qualify for a diagnosis of bipolar II disorder, even if no disorder is really present. Another common source of false positives for hypomania is the presence of "irritability" for several days due to lengthy marital spats. This failure of validity is potentially an especially serious problem for epidemiologists; even with the much more stringent DSM criteria for manic episode, epidemiological surveys using lay-administered structured interviews have gotten a very large proportion of false positives, partly because of misclassification of normal variations in mood as manic episodes (R. Kessler, personal communication, 1998). None of this is to say that bipolar II disorder is not a legitimate category, but only that DSM-IV criteria do not validly identify it.

Acute Stress Disorder

The recently added category of acute stress disorder seems to pathologize normal-range stress responses. If a terrible event (e.g., threatened death or injury, rape) causes fear, helplessness, or horror (as it typically might), and one has stress response symptoms (e.g., feels in a daze and "out of it," thinks about the event, reacts to reminders of the event) for more than 2 *days*, then one is considered to qualify for this diagnosis. Moreover, the criteria are written in such a way that the more extreme dissociative symptoms need only be present while one is actually experiencing the event; they need not continue after the event itself. After the event, one must only be distressed by reminders of the event or keep processing thoughts about the event, try to avoid those reminders, and remain anxious and impaired in function-

ing for 2 days. After such an event, it would seem odd *not* to have such a reaction. There is no doubt that some acute stress responses are so severe and harmful as to be disorders, but the DSM-IV criteria do not distinguish these genuine disorders from intense, normal stress reactions.

These examples, important as they are, are meant to be only illustrative, not exhaustive. Similar problems exist in many other categories throughout the manual.

Conclusion

Careful attention to the concept of mental disorder underlying psychiatry suggests that, contrary to various critics' claims, there is indeed a coherent medical concept of mental disorder in which "disorder" is used precisely as it is in physical medicine. Once this concept is made explicit, it offers a "place to stand" in evaluating whether current symptom-based DSM and ICD diagnostic criteria are accomplishing their goal of identifying psychiatric disorders as opposed to normal problematic mental conditions. This concept appears to be one that is intuitively understood by both the lay public and mental health professionals (Wakefield et al., 2002, 2006). Thus it is possible that those who must argue cases based on expert judgments of mental disorder have opportunities to develop new pathways for examination and cross-examination that can both support and challenge such judgments, independently of what the official diagnostic criteria indicate. Although the HD analysis has been cited in several law review articles, the broader conceptual implications of this analysis for law and psychiatry largely remain to be explored.

Note

1. Some of these examples as well as some passages are revised versions of material that has appeared elsewhere. I thank the American Psychological Association (Wakefield, 1996), the American Psychiatric Press (Wakefield & First, 2003), and Elsevier B.V. (Wakefield, 1997) for permission to use those passages.

References

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., rev.). Washington, DC: Author.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.

Boorse, C. (1975). On the distinction between disease and illness. *Philosophy and Public Affairs*, *5*, 49–68.

Boorse, C. (1976). What a theory of mental health should be. *Journal of the Theory of Social Behavior*, *6*, 61–84.

Brigham, A. (1844). Definition of insanity: Nature of the disease. *American Journal of Insanity*, *1*, 97–116.

Edwards, G. (1986). The alcohol dependence syndrome: A concept as stimulus to enquiry. *British Journal of Addiction*, *81*, 171–183.

Eysenck, H. J. (1960). Classification and the problem of diagnosis. In H. J. Eysenck (Ed.), *Handbook of abnormal psychology* (pp. 1–31). London: Pitman.

Feighner, J. P., Robins, E., Guze, S. B., Woodruff, R. A., Jr., Winokur, G., & Munoz, R. (1972). Diagnostic criteria for use in psychiatric research. *Archives of General Psychiatry*, *26*, 57–63.

Frances, A. (1980). The DSM-III personality disorders section: A commentary. *American Journal of Psychiatry*, *137*, 1050–1054.

Horwitz, A. V., & Wakefield, J. C. (2007). *The loss of sadness: How psychiatry transformed normal sorrow into depressive disorder*. New York: Oxford University Press.

Houts, A. C. (2001). Harmful dysfunction and the search for value neutrality in the definition of mental disorder: Response to Wakefield, Part 2. *Behaviour Research and Therapy*, *39*, 1099–1132.

Johnson, H. (1843). *On the arrangement and nomenclature of mental disorders*. Edinburgh, UK.

Johnson, S. (1755). *Dictionary of the English language*. London: J. F. & C. Rivington.

Kalat, J. W. (1996). *Introduction to psychology* (4th ed.). Pacific Grove, CA: Brooks/Cole.

Kendell, R. E. (1975). *The role of diagnosis in psychiatry*. Oxford, UK: Blackwell.

Kendell, R. E. (1986). What are mental disorders? In A. M. Freedman, R. Brotman, I. Silverman, & D. Hutson (Eds.), *Issues in psychiatric classification: Science, practice and social policy* (pp. 23–45). New York: Human Sciences Press.

Kendell, R. W., & Brockington, I. F. (1980). The identification of disease entities and the relationship between schizophrenic and affective psychoses. *British Journal of Psychiatry*, *137*, 324–331.

Kirk, S. A., & Kutchins, H. (1994, June 20). Is bad writing a mental disorder? *New York Times*, p. A17.

Klein, D. F. (1978). A proposed definition of mental illness. In R. L. Spitzer & D. F. Klein (Eds.), *Critical issues in psychiatric diagnosis* (pp. 41–71). New York: Raven Press.

Meehl, P. E. (1995). Bootstraps taxometrics: Solving the classification problem in psychopathology. *American Psychologist*, *50*, 266–275.

Meehl, P. E., & Yonce, L. J. (1994). Taxometric analysis: I. Detecting taxonicity with two qualitative indicators using means above and below a sliding cut. *Psychological Reports*, *74*, 1059–1274.

Meehl, P. E., & Yonce, L. J. (1996). Taxometric analysis: II. Detecting taxonicity using covariance of two quantitative indicators in successive intervals of a third indicator (maxcov procedure). *Psychological Reports*, *78*, 1091–1227.

Pottick, K. J., Kirk, S. A., Hsieh, D. K., & Tian, X. (2007). Judging mental disorder in youths: Effects of client, clinician, and contextual differences. *Journal of Consulting and Clinical Psychology*, *75*, 1–8.

Richters, J. E., & Cicchetti, D. (1993). Mark Twain meets DSM-III-R: Conduct disorder, development, and the concept of harmful dysfunction. *Development and Psychopathology*, *5*, 5–29.

Robins, E., & Guze, S. B. (1970). Establishment of diagnostic validity in psychiatric illness: Its application in schizophrenia. *American Journal of Psychiatry*, *126*, 107–111.

Sedgwick, P. (1982). *Psycho politics*. New York: Harper & Row.

Spitzer, R. L. (1998). Diagnosis and need for treatment are not the same. *Archives of General Psychiatry*, *55*, 120.

Spitzer, R. L. (1999). Harmful dysfunction and the DSM definition of mental disorder. *Journal of Abnormal Psychology*, *108*(3), 430–432.

Spitzer, R. L., & Endicott, J. (1978). Medical and mental disorder: Proposed definition and criteria. In R. L. Spitzer & D. F. Klein (Eds.), *Critical issues in psychiatric diagnosis* (pp. 15–39). New York: Raven Press.

Spitzer, R. L., & Wakefield, J. C. (1999). DSM-IV diagnostic criterion for clinical significance: Does it help solve the false positives problem? *American Journal of Psychiatry*, *156*, 1856–1864.

Ullman, L. P., & Krasner, L. (1975). *A psychological approach to abnormal behavior* (2nd ed.). Englewood Cliffs, NJ: Prentice-Hall.

Vaillant G. (2003). Mental health. *American Journal of Psychiatry*, *160*, 1272–1284.

Wakefield, J. C. (1988a). Psychotherapy, distributive justice, and social work: I. Distributive justice as a conceptual framework for social work. *Social Service Review*, *62*, 187–210.

Wakefield, J. C. (1988b). Psychotherapy, distributive justice, and social work: II. Psychotherapy and the pursuit of justice. *Social Service Review*, *62*, 353–382.

Wakefield, J. C. (1989). Levels of explanation in personality theory. In D. M. Buss & N. Cantor (Eds.), *Personality psychology: Recent trends and emerging directions* (pp. 333–346). New York: Springer-Verlag.

Wakefield, J. C. (1992a). The concept of mental disorder: On the boundary between biological facts and social values. *American Psychologist*, *47*, 373–388.

Wakefield, J. C. (1992b). Disorder as harmful dysfunction: A conceptual critique of DSM-III-R's definition of mental disorder. *Psychological Review*, *99*, 232–247.

Wakefield, J. C. (1993). Limits of operationalization: A critique of Spitzer and Endicott's (1978) proposed operational criteria for mental disorder. *Journal of Abnormal Psychology*, *102*, 160–172.

Wakefield, J. C. (1996). DSM-IV: Are we making diagnostic progress? *Contemporary Psychology*, *41*, 646–652.

Wakefield, J. C. (1997). Diagnosing DSM, Part 1: DSM and the concept of mental disorder. *Behaviour Research and Therapy*, *35*, 633–650.

Wakefield, J. C. (1999a). Disorder as a black box essentialist concept. *Journal of Abnormal Psychology*, *108*, 465–472.

Wakefield, J. C. (1999b). Evolutionary versus prototype analyses of the concept of disorder.

Journal of Abnormal Psychology, *108*, 374–399.

Wakefield, J. C. (2000a). Aristotle as sociobiologist: The "function of a human being" argument, black box essentialism, and the concept of mental disorder. *Philosophy, Psychiatry, and Psychology*, *7*, 17–44.

Wakefield, J. C. (2000b). Spandrels, vestigial organs, and such: Reply to Murphy and Woolfolk's "The harmful dysfunction analysis of mental disorder." *Philosophy, Psychiatry, and Psychology*, *7*, 253–270.

Wakefield, J. C. (2005a). Biological function and dysfunction. In D. M. Buss (Ed.), *Handbook of evolutionary psychology* (pp. 878–902). New York: Oxford University Press.

Wakefield, J. C. (2005b). Vaillant on positive mental health. *Psicoterapia E Scienze Umane*, *39*, 91–96.

Wakefield, J. C. (2006). Personality disorder as harmful dysfunction: DSM's cultural deviance requirement reconsidered. *Journal of Personality Disorders*, *20*, 157–169.

Wakefield, J. C. (2007). What makes a mental disorder *mental? Philosophy, Psychiatry, and Psychology*, *13*, 123–131.

Wakefield, J. C. (2008). The perils of dimensionalization: Distinguishing personality traits from personality disorders. *Psychiatric Clinics of North America*, *31*, 379–393.

Wakefield, J. C., & First, M. (2003). Clarifying the distinction between disorder and non-disorder: Confronting the overdiagnosis ("false positives") problem in DSM-V. In K. A. Phillips, M. B. First, & H. A. Pincus (Eds.), *Advancing DSM: Dilemmas in psychiatric diagnosis* (pp. 23–56). Washington, DC: American Psychiatric Press.

Wakefield, J. C., Horwitz, A. V., & Schmitz, M. (2005a). Are we overpathologizing social anxiety?: Social phobia from a harmful dysfunction perspective. *Canadian Journal of Psychiatry* 50, 317–319.

Wakefield, J. C., Horwitz, A. V., & Schmitz, M. (2005b). Social disadvantage is not mental disorder: Response to Campbell-Sills and Stein. *Canadian Journal of Psychiatry*, *50*, 324–326.

Wakefield, J. C., Kirk, S. A., Pottick, K. J., Tian, X., & Hsieh, D. K. (2006). The lay concept of conduct disorder: Do non-professionals use syndromal symptoms or internal dysfunction to distinguish disorder from delinquency? *Canadian Journal of Psychiatry*, *51*, 210–217.

Wakefield, J. C., Pottick, K. J., & Kirk, S. A. (2002). Should the DSM-IV diagnostic criteria for conduct disorder consider social context? *American Journal of Psychiatry*, *159*, 380–386.

Wakefield, J. C., Schmitz, M. F., First, M. B., & Horwitz, A. V. (2007). Should the bereavement exclusion for major depression be extended to other losses?: Evidence from the National Comorbidity Survey. *Archives of General Psychiatry*, *64*, 433–440.

Woodruff, R. A., Goodwin, D. W., & Guze, S. B. (1974). *Psychiatric diagnosis*. New York: Oxford University Press.

PART III

Methodological Approaches to Categories, Dimensions, and Prototypes

CHAPTER 15

On the Substantive Grounding and Clinical Utility of Categories versus Dimensions

WILLIAM M. GROVE
SCOTT I. VRIEZE

Investigation of the structure of psychopathology and personality pathology has, at different times and with different persons, been constructive and/or dogmatic. Sometimes the particular conversation is substantially misguided, and could be obviated by a more sophisticated understanding of scientific methodology, statistical inference, and philosophy of science. We propose to contribute constructively to this conversation in three ways: (1) to frame the categorical–dimensional question for the working scientist, as opposed to the statistician; (2) to criticize current statistical/methodological approaches to classification, as well as to identify common erroneous inferences made from them; and (3) to offer our opinion of how future research should proceed, and what inferences from currently used methods are warranted.

At the outset, we must confess a philosophical alignment with Popper (1937/2002), Whewell (1858), and Salmon (1984). In particular, we advocate an approach to theory testing with roots in Popper, based on experimental tests in which one of the scientist's primary purposes in theory building is to put the theory at the maximum possible risk of falsification. For example, a theory that makes a point prediction of a numerical value of an observation, together with the type of function (linear, exponential, logarithmic, etc.) relating x and y, is a "risky" prediction. This stands in contrast to the usual approach in psychology, which consists of constructing a null statistical hypothesis of no actual scientific interest, and having the real substantive scientific theory predict only that the null hypothesis is false (so that just one interval or point null hypothesis is not predicted). This very weak prediction is then tested by observations. The emphasis in psychology is generally quite strongly on attempted easy confirmations rather than on falsifications (see Meehl, 1978, for a more general discussion of Popperian falsificationism in psychology).

Furthermore, we value gathering evidence in favor of a scientific theory in which disparate lines of supporting data converge to support the theory. This is relying on the consilience principle of Whewell (1858) or the "damned strange coincidence" principle of Salmon (1984). One tries to derive as many epistemically independent consequences from the theory as possible, and seeks to learn how many can be corroborated versus falsified by empirical observations.

A perhaps overused example of this concept is apparent in the investigation of molecules and the computation of Avogadro's number. There are more than a dozen starkly different ways to count the number of molecules in a mole, all of which arrive at approximately the same number (6.022×10^{23}). The fact that the same number is achieved so consistently, and with such precision, is what leads many skeptics to admit the existence of molecules.

All theory testing has to be viewed in light of the augmented Quine–Duhem thesis (Gillies, 1993):

$$\text{Test: } T \cdot \{C_P \cdot T_A \cdot T_I \cdot C_B\} \vdash (O_1 \supset O_2) \tag{15.1}$$

where T is the theory under test, C_P is the ceteris paribus clause, T_A are the auxiliary theories relied on in the theory test, T_I are the theories regarding the measuring instruments relied on, and C_B are the material (boundary) conditions for the theory test. O_1 is the set of conditions, either manipulated or observed, required conditionally to predict the occurrence of O_2, the observation of the intended experimental result. $\vdash$ denotes deduction. Note that no theory is ever tested in isolation. The theory-testing scientist always relies on auxiliary theories about instrumentation (T_I; e.g., the Minnesota Multiphasic Personality Inventory–2 [MMPI-2]) and matters outside the scope of the theory at hand (T_A); ensures that boundary conditions (C_B) on the functional forms relating O_1 and O_2 were met; and ensures that all other possible influencing factors were controlled for (C_P; e.g., by random assignment). A clean falsification of the theory is impossible to achieve in a single test. This fact has led some to study the behavior of research programs in general, instead of single "risky" tests (Lakatos, 1980).

In the "hard" sciences, T_A and T_I are often not very controversial, and/or there are methods for controlling or measuring and factoring out the troublesome effects of variations in C_P and C_B. However, in the soft sciences like psychology (especially including clinical and personality psychology), it is quite regularly the case that all the elements on the left-hand side of equation 15.1 are problematic, ill understood, and hard to control or even measure for post hoc adjustment, so that the contribution of each is not only typically unknown, but likely not to be less than T itself (Meehl, 1990).

In what follows, we also draw significantly from Meehl's (1992, 2002, 2004) work on cliometric metatheory, including a list of 11 features scientists use in evaluating theories (see Meehl, 2002, for discussion and justification of these criteria). Table 15.1 gives these 11 features. Most of these will be clear in meaning from the table contents; however, a couple will benefit from further explanation. Feature 6 refers to the fact that a theory may allow the scientist to derive the negative of facts that, if observed, would discorroborate the theory, but when the experiment is done the prediction fails to come true, and so the theory is confirmed. Feature 8 refers to the fact that when a theory is concocted, the author may have access to facts *a*, *b*, *c*, and *d*. He or she concocts a theory to explain these,

TABLE 15.1. Eleven Features Scientists Use to Evaluate Theories

Number	Short name	Explanation
1	Parsimony_1	Simplest Curve
2	Parsimony_1	Economy of postulates
3	Parsimony_1	Economy of theoretical concepts
4	Parsimony_1	Occam's razor (Don't invent a theory to explain a new fact explainable by an ensconced theory)
5		Number of corroborating factors derived
6		Number of discorroborating facts derived
7		Qualitative diversity of facts derived
8		Novelty of facts derived
9		Numerical precision of derived facts
10	Reducibility, passive	The theory as reduced (quasi-Comtean)
11	Reducibility, active	The theory as reducer (quasi-Comtean; Meehl, 2002, p. 346)

and so these facts are useless as tests of the theory, but if the theory also derives *e* and *f*, these novel facts are informative as to the validity of the theory. Feature 10 refers to the fact that if a theory comes to be seen as reducing to a special case of another theory that is well corroborated, the corroboration of the special case goes up. Feature 11 refers to the corresponding fact that a well-corroborated reducing theory spreads some of its validity to the theory of which it is a special case.

Of these 11 features, model selection criteria such as the Akaike information criterion (AIC; Akaike, 1974) and the Bayesian information criterion (BIC, also known as the Schwarz information criterion; Schwarz, 1978) take into account just two to three of these: feature 1 in the sense of fewest free parameters, and features 5 and/or 6 in the derivation of (possibly dis-)corroborating facts. These model selection criteria are thus at best quite limited as theory selection indices, and at worse may prove extremely misleading as overall indices of theory merit. This will be important below.

We apply the considerations above to our discussion of the substantive grounding of taxa versus dimensions in the science of psychopathological theory. A categorical conceptualization of psychopathology has long been ensconced in clinical psychology, and has been increasingly dominant since DSM-III (American Psychiatric Association, 1980). Although the authors of DSM eschew alignments with any particular theory in psychology, and present the manual as "atheoretical" in its preface, it is obvious to any thinking person that DSM was authored (even by committee) with particular theoretical positions in mind, whether this was ever directly acknowledged by higher-ranking task force members or not. The dominant classificatory edifice was and is that of many discrete categories, as opposed to a more dimensional conceptualization. Many might argue that many DSM categories are matters of administrative convenience, and that of course there is no "bright line" between depression and nondepression, or social phobia and nonclinical social anxiety. However, this line of reasoning (usually from personal experience with sad and/or anxious individuals) is hardly experimental, although it is definitely empirical. A recent line of research has proposed that dimensions lie deeper, and that many seemingly disparate disorders can be reconciled by appeal to a handful of latent dimensions (some theories propose two major dimensions and a handful of subdimensions). We examine one such theory in this chapter, primarily because it is a popular theory spearheaded by Robert F. Krueger, a leading figure in the classification of psychopathology; we figure we may as well start at the top.

Krueger's Research on Dimensional versus Categorical Structure

Krueger and his students and colleagues have produced a really impressive body of theory and empirical research aimed at establishing a dimensional basis for myriad psychopathologies. Their work appears promising in replacing most but not necessarily all DSM diagnoses (see below) with rather few dimensions that link normal (e.g., the Big Five; Norman, 1963) and abnormal (Axis II) personality variation with Axis I psychopathology.

We choose to review Krueger's work in some detail, not because we wish to argue that it is radically defective in comparison with other work in the literature, or that it fares poorly when compared to other work based on quite different methods that we review later. In fact, the amount and variety of work Krueger and colleagues have produced is a wonder. Generally, the methodological sophistication of their papers was initially good, and it has grown over time to reflect the state of the art, within the mainstream vein of statistical inference to which they adhere. We have critical comments to make, but these are mostly in the vein of pointing out some unchallenged basic assumptions.

The work of Krueger and colleagues relies on five main arguments for preferring dimensions over categories:

1. Demonstration that the comorbidity of DSM disorders (Axis I or II or both) is consistent with a simple factor structure or a few unidimensional item response theory (IRT) structures of disorder diagnoses or disorder signs and symptoms (comorbidity argument).
2. Continuity between normal and abnormal personality (Axis II; phenotypic continuity argument).

3. Linkage of normal and abnormal personality to concomitant or later-appearing Axis I psychopathology (longitudinal continuity argument).
4. A common etiology (e.g., common genes or environments) causing personality features and/or Axis I psychopathologies (common-etiology argument).
5. Direct tests of dimensional statistical models against competing categorical models (model selection argument).

The Krueger group has used four main statistical methods in its empirical work: (1) factor analysis; (2) IRT analysis; (3) IRT analysis in contrast with latent-class analysis (LCA) by means of model selection statistics, to be explained below; and finally (4) behavior genetic modeling that assigns numerical values to various genetic and shared versus nonshared environmental causal influences over time, on multivariate phenotypes (e.g., personality as well as Axis I psychopathology). Each method can be an informative type of data analysis, and we have no "bone to pick" with any of these methods per se. However, when they are used to try to settle taxonomic hypotheses, the situation can become very complex, and we point out some of these complexities here regarding the first three methods.

Factor Analysis

Krueger (1999) used factor analysis of tetrachoric correlations between 10 "common" DSM-III-R disorders, including major depression but not any of its subtypes. Schizophrenia and all other psychoses, bipolar disorders, and all organic mental disorders were also excluded. We note that this throws out the great majority of all disorders seen on inpatient psychiatric wards: melancholic and psychotic depression; schizophrenia, schizophreniform illnesses, and schizoaffective disorders (not to mention the rarer delusional disorder states); and all cases of delirium, dementia, and amnestic syndrome. The elimination of the organic disorders can be understood if the typology is to cover only "functional" states, but not the others.

In any case, the ability demonstrated by Krueger (1999) to get good fit for a three-factor model for the 10 disorders studied is not evidence against a categorical model per se; it is merely evidence against a *10*-category model. If only signs and symptoms for each disorder are included and are reduced to positive diagnoses (or the lack thereof), then if the entities are categorical, well distinguished on the diagnostic criteria from one another, and independent, there should obviously be as many factors as disorders. If signs and symptoms both typical and atypical of each disorder were included, and factor analysis were conducted at the sign/symptom level, then as many *bipolar* factors as entities should be found. In short, factor analysis depends only on the second moments of the data, and if there exists covariation between measures (signs, symptoms, behavioral abnormalities) with more than one measure to reflect each type of abnormality, then (absent sampling error) common factors will emerge. This point has been made by many; Grove (2007) explored its mathematics in detail for two latent groups and one to k resultant latent dimensions, and Bartholomew (1987) devoted an entire excellent volume to the formal duality between latent factors and latent profiles (latent mean vector differences between categories). Hence the fit of $\Sigma = \Lambda\Phi\Lambda' + \Psi^2$, the common-factor model for a covariance matrix Σ, will fit even if Σ is actually generated according to $\Sigma = PQ\ (\mu_t - \mu_c)(\mu_t - \mu_c)'$ (i.e., an admixture of two latent categories with within-class zero covariance between all the manifest variables).

Let us return to the topic of comorbidity. One way comorbidity can occur without the presence only of dimensions is if one disorder is a genuine risk factor for another. For example, the genetic disease a1 antitrypsin deficiency (protease inhibitor Z [PiZ] homozygote) is a risk factor for emphysema (Elliott, Bilton, & Lomas, 1998), pneumonia, pleurisy, hepatitis, cirrhosis of the liver (Cox, 1989), and (especially in persons who smoke) death by age 60 (86% of homozygotes vs. 16% of the general population; Brantly et al., 1988). It would be foolish to expect, given the complex interworkings of the human body, that various disorders would ordinarily occur at joint frequencies equal to the product of their marginal frequencies in the population as a whole (i.e., independently). In sooth, a factor analysis of a1 genotype, lung disorders, and liver disor-

ders would show a strong first factor if the population on whom the factor analysis was conducted contained a reasonable percentage of PiZ homozygotes, as it would if subjects were sampled in a respiratory clinic (the PiZ allele is fairly common, estimated to occur at 2% in the United States, but would of course be much higher among those selected for having known lung problems, where the rate runs up to 12%).

Here is another example: Frequent upper respiratory infections, childhood asthma, and *pectus excavatum* (sunken sternum, caused by breathing with the accessory muscles during asthma attacks while the diaphragm cannot expand the rib cage, owing to severe bronchoconstriction) all go together. These three signs are "comorbid" because asthma is a risk factor for the others; factor analysis cannot show how the causal arrow points from asthma to the other two because it does not have that kind of capability. The bottom line is

that comorbidity occurs in physical medicine situations, and one does not generally know how to explain it *until* the specific etiologies of all relevant disorders are at least reasonably well understood. The fact and extent of comorbidity in psychopathology thus constitute a poor argument, even prima facie, in psychopathology for or against categories or dimensions.

Under the circumstances just outlined, comorbidity is not in and of itself an embarrassment to a classification system, and any statistical analysis must account for it as a fact of nature. The presence of large amounts of comorbidity for certain clusters of disorders, in certain patient populations, or for the nomenclature as a whole may be simply the mark of a complex causal structure—not evidence that a class structure does not describe the domain. Equally, of course, it *may* arise from a dimensional causal setup; we would simply argue that comorbidity is not evidence for either categories or dimensions. Reporting a fact, like comorbidity, that can be accounted for by either taxonic or dimensional theories is reporting a corroborating fact for both theories, rather than a corroborating fact for one theory and a discorroborating fact for the other one. This *greatly* lessens the value of the factor analysis findings as an evidentiary stake around the tent pole of dimensions, unless it can be demonstrated, for example, that the only versions of the taxon hypothesis are those that have unreasonable parameter values.

Krueger and colleagues (2002) conducted a behavior genetic analysis of personality traits (disinhibited personality style), the externalizing psychopathology dimension, number of antisocial behaviors, and psychoactive substance use behaviors as measured by the Multidimensional Personality Questionnaire (Tellegen & Waller, 2008). Their selected model contained additive genetic and nonshared environmental inputs for the externalizing dimension. This then had sizable inputs (.47 to .78) into alcohol dependence, drug dependence, adult antisocial behaviors, conduct disorder, and constraint—reversed. Each of these had its own nontrivial additive genetic, common environment, and nonshared environment inputs. The shared input from the externalizing dimension accounts for the comorbidity, and the sizable (b = .90) genetic coefficient on this effect suggests that the same family could include one member with alcohol or drug dependence and another with conduct disorder or adult antisocial behaviors, all with a common underlayer of externalizing. These findings help to undercut the possibility that taxa are present, and that the manifest categories are showing comorbidity up to $\Sigma^{k}_{i} = 1(^{k}_{i})$ ways that just happen to mimic the action of an externalizing spectrum.

IRT Analyses

Krueger and colleagues' more recent work has employed a different model—namely, one in which the probability of dichotomous item endorsement is modeled as a function of a single latent dimension. Most commonly, the model is the logistic IRT model:

$$\Pr\{X_g = 1|\theta\} = \frac{\exp[Da_g(\theta - b_g)]}{1 + \exp[Da_g(\theta - b_g)]}$$

Very difficult items have high b_g (the point on θ where 50% of examinees pass or endorse the item), while highly discriminating items have high a_g. A highly discriminating item is one that has high slope where b_g = 1/2; it is good at telling whether the examinee has $\theta_i > b_g$ or not. D is a constant used to

make the shape of the curve match a normal ogive and equals 1.7.

Krueger, Markon, Patrick, Benning, and Kramer (2007) used just such a model to tie together the externalizing spectrum including personality traits, antisocial behavior, and psychoactive substance use. Reasonable fit was obtained (criterion 5 in Table 15.1). Unfortunately, nothing novel or varied (Meehl's criterion 6 or 8) was predicted, nor was any risky discorroboration attempted. As a consequence, the result was only impressive to the extent that IRT models failed to fit the data. In fact, as long as data sets have been preselected to be unidimensional (all that is required is to make a tetrachoric correlation matrix and examine the first two eigenvalues), an IRT model can essentially always be fitted. In this instance, previous factor-analytic results had already established that this externalizing domain had a single-common-factor structure. Hence these types of IRT models tell us relatively little that is new regarding the question of categories versus dimensions.

Use of the IRT Model in Competition with an LCA Model

Finally, Krueger, Markon, Patrick, and Iacono (2005) made a direct comparison between IRT models and LCA models in terms of their ability to fit a single data set. Again, the behaviors examined were alcohol problems, drug abuse, and antisocial personality and behavior—the so-called externalizing spectrum. The best-fitting (by the BIC [see below]; Schwarz, 1978) LCA model had two classes (BIC = 3, 746.638), while the best dimensional model under consideration had an indefinite number of values (i.e., arrayed along a single normal distribution; BIC = 3, 735.18). As the BIC for the dimensional model was smaller than that for the LCA model, the dimensional model was to be preferred (3, 746.638 – 3, 735.18 = 11.45). Assuming equal priors for each model under consideration (which we regard as nearly always inappropriate), the posterior probability that the univariate dimensional model was the "best" model under consideration was .9998—a hefty proposition.

This type of analysis involves a substantially different mode of reasoning from the previous ones, which reach no farther than significance/hypothesis testing and goodness-of-fit tests. Here the scientific theory evaluation problem is idealized as the (much simpler) model selection task. There are presumed to be two or more theories, each one spelled out as a fully parameterized statistical model (in this case, IRT model vs. LCA model). In an LCA model with (as is usual) a local-independence assumption, the cooccurrence of positive scores on signs, symptoms, or abnormal behaviors is accounted for entirely by the existence of latent-group admixture. m latent classes and a vector of Bernoulli parameters are postulated, m – 1 of which are independently identifiable. To enable us to choose between competing models, a figure of merit for each one is calculated: BIC = $-2 \log(\mathcal{L}) + k \log(n)$, where $\mathcal{L}$ is the sample likelihood on n observations. k is the number of independent free parameters fitted in the model, so that the second term is a fairly stiff penalty on the likelihood for complexity measured in this way. The model with the lowest BIC is selected, and the theory associated with this model is considered the better.

We wish to take strong issue with this synonymization of model selection with theory testing. Therefore, we must dissect the BIC and attend to its structure and properties in some detail.

The overall structure of the BIC is $-f_1(\mathcal{L}) - f_2(n)\, k$, where $f_1(x)$ can be a decreasing function of x and $f_2(n)$ can be a constant function. In the well-known AIC (Akaike, 1974) $f_2(n) = 2$, and the AIC is generally written as AIC = $-2 \log(\mathcal{L}) + 2k$. Generally $f_1(x)$ is $-2 \log(x)$, so that small values are avoided and instead of products we have sums. For example, with the normal likelihood we have

$$\mathcal{L} \propto \prod_{i=1}^{n} \exp - \frac{(x - \mu)^2}{2\sigma^2}$$

because the constant term involving $1\sqrt{2\pi}$ does not matter. Therefore,

$$-2 \log \mathcal{L} \propto \sum_{i=1}^{n} - \frac{(x - \mu)^2}{2\sigma 2} \qquad (15.2)$$

so that $-2 \log \mathcal{L}$ is the kernel of the normal likelihood. When it increases, so does the size of the normal likelihood.

The second term involving k, the number of free parameters estimated, is what distinguishes the BIC from the AIC. The BIC is essentially always more than the AIC (because in practical circumstances $n > 7.4$), so when model 1 and model 2 have df_1 and df_2, respectively ($df_1 > df_2$), the BIC discriminates more sharply between the models than does the AIC.

Although the justification for the BIC is in terms of Bayes factors (Burnham & Anderson, 2005; Kass & Raftery, 1995), there is for exponential distributions a strong asymptotic justification, as follows. Assume that $m \geq 2$ models are compared by their BIC values. Assume further that one of the models is the correct model. Then as $n \to \infty$, with $p \to 1$ the correct model is selected.

Now suppose everything is as before, but the correct model is not in the candidate set of models. As $n \to \infty$, with $p \to 1$ the model with the least Kullback–Leibler (K-L) divergence (Kullback & Leibler, 1951) is selected by the BIC. We attempt to explain K-L divergence in the next section.

The first result establishes a strong rationale based on the likelihood principle (i.e., the principle that a hypothesis is favored to the extent that it is likely to occur, given the underlying probability density), Bayesian statistical reason, and appeal to the long-run property of statistics (the frequency principle). Suppose the BIC is attacked with the argument that it is impossible to be sure that the correct model is in the selection set, and so the BIC has only theoretical virtue. The answer, based on the second property of the BIC, comes to the rescue in the form of a guarantee (asymptotically) of the smallest K-L divergence for the "best" BIC-selected model among those being subjected to examination.

We contend that the true model is virtually *never* in the set of candidate models, and with Burnham and Anderson (2005) suggest that it is philosophically difficult even to conceptualize what it would mean to have the *true* "model." (Would it even have parameters? Or are parameters an artificial creation we use to describe the natural world?) Especially in psychopathological science, it is difficult to accept that any model even approaches the full true theory of mental and personality pathology. In this case, one invariably justifies use of the BIC through K-L divergence. The BIC is far from the only estimate of K-L divergence; other examples abound (see Burnham & Anderson, 2003, for a list). We discuss the AIC as one alternative to the BIC.

K-L Distance and the BIC

Kullback and Leibler (1951) established an information measure, now often referred to as K-L divergence or distance, between a true or reference distribution on the one hand and a distribution generated by a model on the other hand. This is so important to understanding the BIC that it is worth spelling out:

$$I(f, g) = \int f(x) \log \left(\frac{f(x)}{g(x|\theta)} \right) dx$$

where $I(f, g)$ is the K-L distance between the true distribution f and the predicted or modeled distribution g. θ is the parameter (possibly vector) of the model that generates $g(x|\theta)$, the modeled distribution. $f(x)$ does not take parameters because it is not a model; it is reality itself as it is observed to occur.

Problems with the BIC in testing scientific theories fall into two groups: (1) use of the BIC as an estimate of K-L divergence, and (2) use of K-L divergence in testing the verisimilitude ("truthiness") of a scientific theory.

Our reply to the rationale for use of the BIC (as opposed to, say, the AIC) as a measure of K-L divergence is taken closely from Burnham and Anderson (2003, 2005). The AIC and the BIC are extremely similar; as defined in this chapter, they are approximately equal to $n = 7.4$. As n grows, so does the ability of the BIC to choose with high probability between two candidate models. Asymptotically, the AIC and the BIC will always agree, and select the candidate model with the lowest K-L divergence from the truth. In practice, they will not always agree, as was the case in Krueger and colleagues (2005), cited above. Using the BIC, Krueger and colleagues found that the normally distributed unidimensional latent-trait model was the model with the least estimated expected K-L divergence; the posterior probability that this was the best model was .9998

(assuming equal priors). The BIC-derived posterior for the four-class LCA model was 1.5×10^{-25}. The BIC-derived odds ratio for the unidimensional model compared to the four-class model was 94,845:1. On the other hand, we computed the AIC for each candidate model in Krueger et al., and a very different result was obtained: The LCA model with four classes is now the *best* model (in K-L divergence); the posterior weight assuming equal prior weights that this was the best model is .84. The AIC-derived posterior for the normally distributed unidimensional latent-trait model was .0043. The AIC-derived weight ratio for the four-class model to the unidimensional model was 195:1. Had Krueger et al. used the AIC instead of the BIC, a *very* different conclusion would have been drawn—namely, that four latent categories best accounted for the observed nonindependence of the manifest variables. Their conclusion would have been in the categorical spirit, and not dimensional.

At present, it is unclear how such differences should be reconciled, especially as both the AIC and the BIC are ostensibly measuring the same thing. Burnham and Anderson (2005) note this difficulty, and in effect state that each method outperforms the other under various circumstances, which are of course never known outside of computer simulations. In particular, they contend, and provide Monte Carlo evidence, that BIC may perform better (in terms of more often selecting the best model in K-L divergence) than AIC when there are a few large effects in the population (e.g., there are only two classes or one latent trait). On the other hand, the AIC may perform better when, in addition to larger effects, there are also many tapering effects in the data set that argue for fitting more complicated models. We conjecture that any verisimilar psychopathological model will predict tapering effects, as psychopathological constructs are most likely very complicated in a causal/structural sense, with many causal inputs and interrelations with other psychological and physical states and events. However, we freely admit that at this point it is unclear whether the AIC or the BIC is to be preferred as the measure of estimated expected K-L divergence.

We wish to consider limitations of the BIC (or the AIC) in scientific theory testing in four parts:

1. A philosophical analysis of the limits of the BIC as a figure of merit for a scientific theory (and of the limits of two BICs as figures of comparative merits of two theories).
2. A caveat regarding the additive combination of the penalty for k, the number of parameters estimated, with $\mathcal{L}$ the likelihood. Since the penalty is part of the BIC, then problems with the penalty necessarily create problems with the BIC.
3. Some perhaps heretical comments on the operation of the likelihood principle, both in theory and in practice. Since this is part of the BIC, problems with the likelihood principle imply problems with the BIC.
4. The argument that the "best" model as selected by information criteria (including the BIC and the AIC) is never as useful as a multimodel approach, where each "decently good" model in the candidate set is retained, and all such models are used jointly to make predictions (each with a weighting relative to their estimated expected K-L divergence).

$\mathcal{L} - f$(Parameters) as a Figure of Merit for Theory

In his cliometric metatheory work, Meehl (2002) advocated studying numerous representatively sampled episodes from the history of science to determine (1) what theory was ultimately vindicated; and (2) which theories, winners and losers, stood where on each of the 11 features listed in Table 15.1. Then discriminant analysis or some similar statistical method could be used to assign weights to the 11 features in order optimally to predict the "long-term winner." Even if parameter count as in BIC and AIC were assumed to be the appropriate measure of parsimony1, it would seem prima facie a miracle if the weight ultimately assigned to all criteria in Table 15.1 were zero except for feature 1, the simplest curve.

The BIC gives no place for the three other senses of parsimony: parsimony2, number of theoretical postulates; parsimony_3, number of theoretical entities (e.g., factors, taxa, neural circuits); or parsimony_4, Occam's razor. It further gives no direct weight to five features of theory performance regarding derived facts: number of corroborating

facts derived, number of discorroborating facts derived, qualitative diversity of facts derived, novelty of facts derived, or numerical precision of derived facts. Finally, the BIC pays no attention to the theory as reduced or to the theory as reducer.

We are not criticizing the BIC for not being an all-encompassing figure of merit for scientific theories. We are criticizing the BIC for starting down the road to being a figure of merit, and not going far enough. This is of course unsurprising, and is obviously the position adopted even by Krueger et al., proponents of the BIC. As noted at the outset of this chapter, Krueger and colleagues have instituted a *program* of research suggesting that some psychopathological entities previously thought as categories are actually dimensions. Their program attacks this goal from multiple angles, and they are not so naive as to suppose that any statistic, let alone one so obviously deficient as the BIC (or the AIC), is a truth-grinding machine.

It is absolutely crucial to distinguish the theory at hand, which the working scientist is attempting to falsify, from a mathematical model representing the theory. It is often, if not always, the case that the mathematical model carries with it extra assumptions, idealizations, and simplifications that are not "core" to the theory. For example, Krueger and colleagues (2005) compared LCA models with latent-trait models; both models assumed local independence of the manifest variables (conduct disorder, antisocial personality disorder, alcohol dependence, drug dependence, marijuana dependence), given the latent variable(s). Simple counterexamples can be constructed to show that local independence is not required for the existence of taxa, although local independence is one framework under which taxonicity can arise (and is perhaps the most intuitive one). In true Lakatosian style, models carry idealizations that are perhaps at best considered "peripheral" to the "hard core" of the theories; local independence is an acceptable, but unnecessary and disposable, peripheral aspect of taxonic theories. Equivalently, any dimensional theory can stand to deduce non-normally distributed latent traits (e.g., they could be logistic, or even much more exotically distributed), although that is what Krueger et al. specifically investigated.

Recall the Quine–Duhem thesis from the beginning of this chapter (equation 15.1). Any test of the theory T is necessarily also a test of the *ceteris paribus* clause (C_p), auxiliary theories (T_A), theories of instrumentation (T_I), and boundary conditions (C_B). C_P and C_B always exist as nuisance considerations in any theory test. (Because C_P is a *ceteris paribus* clause and does not enumerate all influences it encompasses, it is impossible for the representative model to include all parts of the conjunction.) The difficulty of the Quine–Duhem thesis is that when the material implication $O_1 \supset O_2$ is false (i.e., the predicted result of the experiment did not pan out), it logically entails that the conjunction $\{T \cdot T_A \cdot T_I \cdot C_P \cdot C_B\}$ is false. Note that the *entire* conjunction is false, not just T alone. Recall from freshman logic that the negation of a conjunction is the disjunction of the negations, and $-\{T \cdot T_A \cdot T_I \cdot C_P \cdot C_B\} = -T \vee -T_A \vee -T_I \vee -C_P \vee -C_B$. That is, we know one of the five conditions is false, but we do not know which one it is. In fact, all five conditions could be false. This is the difficulty with testing any scientific theory: The theory is never tested in isolation.

To take the line of reasoning further, one can also break T into constituent parts ($T_1, \ldots, T_n$), all of which must hold true for the right-hand side $O_1 \supset O_2$) to hold true. If $O_1 \supset O_2$ is false, no single part of T is implicated, and indeed there may be one (unidentifiable) false postulate T_i causing the right-hand side to fail. Assume then that the theory test is such that we have good evidence that T, the substantive theory, has been discorroborated (or falsified, in Popperian parlance). Well, in the model-testing scenario, T is actually an approximating model, and the theory test is not of the (possibly true) substantive theory T, but the model M, which is likely to be expressed with various departures from the substantive theory (e.g., idealizations such as local independence). Proponents of T can legitimately maintain that M failed the test not because of the hard core of the theory T (e.g., $T_1, \ldots, T_8$), but rather because of the peripheral and disposable idealizations necessary to express T as M. To take a concrete example, a proponent of a dimensional model can, in the face of a poor BIC value, legitimately argue that the substantive theory T really does not require the assumption of local independence used in model M.

The BIC and AIC (obviously) do not test how $\log(\mathcal{L})$ is computed, or what assump-

tions, mathematical foundations, and departures in M from the original theory T (we are assuming that the model and theory are not isomorphic) were necessary to arrive at the single, potentially obfuscating number $\log(\mathcal{L})$ that is considered so credulously by the BIC and AIC. In this sense, Meehl's (2002) classification of the BIC and AIC as (potential) solutions to the *model-fitting* (not theory-testing) problem is correct, and other tools for theory appraisal must be concocted fully to address differences between competing *theories*. We thus argue that neither the BIC, the AIC, nor the maximized $\log(\mathcal{L})$ do any more than begin to address Meehl's (2002) criteria in Table 15.1.

As the reader may imagine, we commend researchers for mathematizing and quantifying psychopathological constructs; we encourage more psychologists to adopt sophisticated quantitative methods. We believe that the Quine–Duhem thesis poses a very substantial hurdle in scientific theorizing, and that mathematically minded research programs will bear more fruit in the face of it. However, researchers need to use mathematical approaches to theory testing in a circumspect manner; no statistic should ever be used as a truth-grinding machine.

The BIC, and other estimates of K-L divergence, are not the only defensible attempts at mathematical definitions of parsimony. Consider the very simple functional form of $\text{AIC} = \log(\mathcal{L}) - k$. (This is the way Akaike, 1974, derived it, before he multiplied it by the –2 constant, for historical reasons.) The log of the likelihood is penalized by the number of parameters k, and aside from this penalty the AIC pays no attention to form of the function contributing to the other term, the $\log(L)$. However, some nonlinear models can result in substantially increased likelihood, and with fewer parameters than are required with linear decompositions such as those used in LCA and IRT analyses.

For lack of space, and lack of expertise, we consider here for the sake of argument only the trignometric sine function. With only two scalar parameters, the sine function can be made to fit arbitrarily well any set of data points in two-dimensional space (Kecman, 2001). If the AIC (or BIC) is applied to such functional forms, it will fail to account sufficiently for potentially massive overfitting, and will penalize the function for only two parameters—an obvious mistake.

We grant that investigators will not blindly compare linear models with trignometric ones, but it is important to consider that this simple example suggests that the BIC and AIC cannot be adopted as a panacea to the parsimony problem. There are other mathematical characterizations of parsimony that do not rely on K-L divergence, including the Vapnik–Chervonenkis (VC) dimension (Vapnik & Chervonenkis, 1971; for a tutorial, see Burges, 1998). The VC dimension uses a concept called shattering to understand the "flexibility," as it were, of a functional form. This of course includes flexibility inherent in the sine function, as well as that connoted by number of parameters in a linear decomposition; VC dimension parsimony would then penalize the sine function because it is so flexible, not just on the basis of parameter number. In fact, the VC dimension (i.e., penalty) of the two-parameter sine function in two-dimensional space is infinite. The VC dimension is not a new concept, and is central to support vector machines, a method gaining some popularity in pattern recognition and prediction (Burges, 1998).

Log(n)k as a Penalty for Model Parameters in the BIC

We have already remarked that the motivation for the multiplier of k, the count of independent parameters—namely, $\log(n)$—is that the BIC is an approximation to the Bayes factor. Furthermore, with the penalty in this form, the BIC functions asymptotically to pick the correct model from a set, as long as the distribution is in the exponential family.

The problem with $\log(n)k$ is that it is not the only competing adjustment to $\log(\mathcal{L})$. The AIC (Akaike, 1974) arrives at a correction of k rather than $\log(n)k$ by asymptotic reasoning involving information theory. Unless one is committed to Bayesian theory, the argument that the BIC approximately equals a Bayes factor has no force. The argument about correct model selection is a toss-up because both the AIC and BIC have arguments of this kind, and both can disagree just as they did in Krueger and colleagues (2005).

Problems with Reliance on the Likelihood in the BIC

Since Fisher, it has been accepted as a powerful argument (and among Bayesians, it has been accepted as a knock-down argument) that the likelihood function carries the entirety of the relevant information for evaluating a statistical hypothesis. The mainstream approach to data analysis in soft sciences like clinical psychology considers the scientific theory test as isomorphic with statistical hypothesis testing; very frequently the test statistics involve likelihoods and/or their ratios. The BIC is clearly one such statistic, and so is the difference between BICs belonging to two competing theories, such as an IRT model versus an LCA model.

To make things concrete, assume as before that the likelihood is the normal probability density function, as we had in equation 15.2—but now with an additional, independent variable in a fixed-effects regression situation. Then twice the negative of the log-likelihood—which is handier to work with, as it does not involve tiny decimal fractions and can be minimized to obtain a maximum-likelihood estimator—is (ignoring an irrelevant constant) $\sum_{i=1}^{n} (y - b_1 x)^2 / \sigma^2_{Y|X}$. Note that this is the residual sum of squares. Given that we can assume, without loss of generality, that $\sigma_{Y|X} = 1$, it is simple to show that the log-likelihood (as usual, ignoring the leading constant) equals $1 - R^2$, where R^2 is the coefficient of determination.

Forster (2006) gives a bevy of examples that violate the likelihood principle, formulated by Birnbaum (1962) essentially as the thesis that once the data have made their contribution to inference through the likelihood, they have no further information to add. One example is simple enough and so telling as to bear repeating here. Imagine that we have models F and G:

$$F\text{: } Y = \alpha + \beta X + \sigma U$$
$$G\text{: } X = a + bY + sZ$$

U and Z are standard Gaussians, U is independent of X, and Z is independent of Y. Suppose

$$Y = \frac{10}{\sqrt{101}} X + U$$

This guarantees that σ_X and σ_Y are the same when the data are clustered around $X = -10$ and $X = +10$. If one fits F and G separately within data clusters, one can compare model parameters α to a, β to b, and σ to s. But it is easy to let X_1 be the cluster of data near $X = -10$ and Y_1 be the cluster near $X = +10$. We can then rewrite F as two stochastic equations:

$$Y_1 = \alpha_1 + \beta_1 X_1 + \sigma U_1$$
$$Y_2 = \alpha_2 + \beta_2 X_2 + \sigma U_2$$

with two constraints, $\alpha_1 = \alpha_2$ and $\beta_1 = \beta_2$. After fitting, it will be found that independent estimates of the compatible F model parameters will agree very closely. However, estimates of related parameters from the B model will fail to coincide. For example, α_1 will be about –10 and a about +10. Inference through the likelihood to the values of the underlying setup is completely misleading, while graphic inspection of the data allows reading out the approximate values of all parameters right off the graph.

Illustration of Wrong Likelihoods and Their Effects on BICs

Consider a gamma distribution ($\lambda = 5$, $\sigma = 3$) plus 5, which is misperceived by the researcher as possibly representing a mixture of two normal distributions. If one samples 3,000 observations from such a distribution (this being in the upper range of sample sizes used by Krueger et al.) and subjects them to a univariate normal mixture model (constraining the solution to two components with equal variances), one obtains, in the single sample we tried, a BIC of 19715.44. If one fits only one component (H_0, as it were), the BIC becomes 19944.83, a decrease of 229 with a difference of just two free parameters. In this instance we have BIC theory (i.e., choose the lowest BIC) leading to a glaringly incorrect result (the posterior probability for the two-component model would be, for all intents and purposes, 1.0). Asymptotic χ_2 distribution theory, whereby the value $\mathcal{L}_2 - \mathcal{L}_1 = 245.4$ is referred to a χ^2_2distribution, with a resulting (extremely low) p value, also gives the wrong answer. The misreading of an unmixed gamma distribution as a mixture of Gaussians occurs, of course, because the

normal likelihood produces large deviations where the gamma has an asymmetry and a large tail.

A statistical purist would respond, "So what? If you use the wrong likelihood, you will get erroneous results." We reply that a statistical analysis is supposed to start with assumptions that are either reasonably certain to be true, or can be readily checked; to rely on calculations from the data; and to yield sound inferences to new knowledge. However, there is no way to know at the outset whether a right-skewed sample is such because it simply is so, or because it comprises an admixture of symmetrical distributions, with the smaller mode lying to the right.

This is our primary objection to the use of finite-mixture models, even though we have employed them in our own work. They are very powerful techniques, but their power comes from their very demanding assumptions—chiefly the assumptions made about the parametric form of underlying admixed distribution components. If those assumptions are materially false, one has the classical situation called "garbage in, garbage out."

We surmise that this is much less of a problem with the type of mixture model called LC analysis, and contrasted with IRT models using the BIC metric by Krueger and colleagues. The reason for this is simple. There is relatively little that can go wrong with the specification of the underlying distributions in LCA, compared to continuous-variable mixture models. There are two main ways things can go awry with respect to assumptions: (1) excessive piling up of observations in the all-zero cell of the contingency table (zero inflation); and (2) nonindependence within latent classes, frequently confined to low-order interactions. We submit that these two sources of error are not likely systematically to affect the BIC for IRT models much more or less than the BIC for LC models (based on consideration of the BIC algebra as largely a proxy for the likelihood in this situation), at least as far as BIC differences are concerned, because parameter count differences are small. As a result, we expect that the distribution misspecification problem, which we believe to be a bane of work with continuous variables, is a small albeit nonnegligible problem with the kind of dichotomous (rarely, polytomous) test item indicators used by Krueger and colleagues.

A Fresh Approach: Meehl's Taxometrics

The foregoing discussion establishes that it would be a great boon to have a method of studying admixture problems for continuous variables (and possibly dichotomous variables as well) that makes weaker assumptions about the variables than do mixture analyses. In particular, we want a mode of analysis that does not require an assumption about the variables' probability density functions.

Paul E. Meehl, starting in 1965, developed and published a method he called "coherent cut kinetics taxometrics" for doing precisely this. It is exemplified by several taxometric procedures: MAXCOV-HITMAX (Meehl, 1965), MAMBAC (Meehl & Yonce, 1994), MAXSLOPE (Grove, 2004), and MAXEIG (Waller & Meehl, 1998), as well as a couple of others that were never published. The central idea is this: Rank-order observations on values of an input variable Z. Either create intervals on Z, or treat the value of Z moving from low to high as a cutoff score on Z (depending on the procedure). Calculate a statistic that is a function of one (X) or two (X, Y) output variable(s) within each interval, or above and below the moving cutoff score, again depending on the procedure. In other words, each function of X or X, Y is conditional on Z. Finally, examine values of the calculated statistic conditional on Z. If there exists a taxon, then the function shape will show a particular form; if there exists a factorial latent space, the function shape will show a markedly different form. This deceptively simple idea (combined with "consistency tests," or checks and balances along the way) generates the risky test of taxon or factorial hypotheses/theories.

Grove, Waller, and Vrieze (2009) have recently given a set of jointly sufficient mathematical assumptions required to prove the main theorems of one such coherent cut kinetics procedure, the MAXCOV-HITMAX taxometric procedure. Without significant changes, this set of assumptions also apply to other such taxometric methods. The key assumption is that when the observations are sorted on input variable Z from low to high, they are also sorted on probability of taxon membership. We call this the monotone increasing taxon probability (MITP) assumption. It contrasts with the cognate assumption of LCA, uniform monotone la-

tent variable (UMLV). UMLV states that when one sorts on the latent class (i.e., puts all members of latent class 0 first, then all the class 1 members), one arranges things so that the average score on manifest variable 1 is in increasing order, the average on variable 2 is increasing, and so on. The two assumptions are in some sense mirror images.

We explain just one taxometric procedure here—the first invented, most studied, and most implemented to test taxonic/factorial theories: MAXCOV-HITMAX. This procedure sorts on input variable Z as explained above. Nonoverlapping narrow (by default, 0.25 *SD*) intervals on Z are set up. The covariance between X and Y is calculated for observations in each interval, denoted $\sigma_{mXY}(z)$ where the subscript m denotes the fact that the covariance may be calculated in an admixed population. The resulting covariances, one per interval, are graphed against the midinterval value of z. This is called the MAXCOV graph. It can readily be shown that on a taxonic conjecture and several other assumptions, including MITP, the function $f(X, Y|Z_{\text{interval}})$ should have a single maximum; that is, there should be a strong peak (in ideal circumstances, the graph of $f(X, Y|Z_{\text{interval}})$ is sombrero-shaped). How high the peak is depends on how big the difference is between taxon and complement class on X and Y compared to the X and Y variances. As the presence of taxonicity or factorial structure should not depend on the conditioning variable, one typically examines the form of all combinations of variables. For the three-variable setup, one examines the functions $f_1(X, Y|Z)$, $f_2(X, Z|Y)$, and $f_3(Y, Z|X)$ for single-peakedness.

If all the graphs indicate that there is a taxon, it is next of interest to estimate parameters of the latent distributions. Some algebra allows manipulation of quantities, such as the heights of the three MAXCOV curves (more, if there are more than three manifest variables) at their peaks, to solve for the latent distribution means, variances, and density functions. Assumptions made during the estimation process can then be checked, such as the assumption that X and Y are uncorrelated within taxon and complement class; if this particular assumption is false, corrections to parameter estimates can be made.

There is frequently more than one way to estimate a particular parameter in taxometrics, and the second way is never mathematically reducible to the first. This leads to the concept of a consistency test. If the assumption of the existence of a taxon is needed to derive each of the two parameter estimates, so that in the absence of this assumption the parameter estimators do not exist or cannot be derived by using only general algebra (algebra unsupplemented by the mathematics of taxometrics), then the comparison of the two parameter estimates is a so-called "consistency test." Absent sampling and (what is more important, when working with large samples) approximation error, two estimates of the same thing should agree. If they do not, this is a blow to the taxonic conjecture used to derive their equality. Meehl (1965, 1968) initially invented 35 consistency tests for MAXCOV-HITMAX—some simple equality tests for parameter estimates, and some rather more involved. As Meehl was, we are adamant about the centrality of consistency tests to the taxometric enterprise; we return to this point below.

The taxometric procedures themselves are advised to be used together, not singly, in order to be able to use the results of one versus another as another sort of consistency test. The procedures taken together, plus consistency tests that work "within" taxometric procedures, (1) provide neo-Popperian risky tests of scientific theories that postulate the existence of a natural category, and (2) fulfill a number of the criteria listed in Table 15.1. They derive corroboratable facts, including ones that could well be discorroborated but perhaps will not be (the risky tests); they derive novel facts in certain of the consistency tests; and they derive varied facts, if varied measures are put into the taxometric analysis—something Meehl also recommended and illustrated in his examples.

Based on the mathematics of taxometrics, the logic of consistency tests, and a neo-Popperian philosophy of science, Meehl advised the following ingredients for a sound taxometric study:

- Having a sound theoretical rationale for positing the existence of a taxon. Taxometrics is not well designed as a "taxon-sifting" exploratory data analysis tool.
- Employing not dichotomous but continuous measures, as these contain much more theory-discriminating information.
- Employing qualitatively diverse measures (different "methods" factors). For ex-

ample, in a study of depression, suppose we use one rating scale comprising the sum of no less than eight 5-point ratings but in which mostly only the 0–3 range gets used, giving an effective range of 0 to 24. As a physiological measure, let us use time for serum cortisol to escape from dexamethasone suppression. As a pharmacological measure, let us use the degree to which the subject's active drug response (weeks 2–12) exceeds that during the placebo washout period (weeks –2 to 0). As a family history measure, let us use the total number of depressive episodes documented in all first-degree relatives, taken together. And so forth.

- Studying a population that, if a taxon does exist, will have a base rate of the taxon that is not too low (say, $p > .1$).
- Employing measures that differentiate taxon from complement class as much as possible—2 within-class *SD*s or more (1.5 *SD*s if necessary, but if the preliminary work has been done properly, this compromise should not be needed).
- *Ceteris paribus*, employing measures correlated within latent classes as little as possible. (Using qualitatively diverse measures, so that shared method variance is minimized, will go far toward ensuring this.) With postulated values for δ_X and δ_Y (the difference between taxon and complement class means), one can bound the expected correlation ρ_{XY} from above to check this on the actual data. The difference between the observed correlation and the expected correlation gives the within-class correlation. Alternatively, some part of the measures can be used to sort the observations along a continuum from confidently conjectured complement class to confidently conjectured taxon membership; then one can calculate the correlations between other, held-back measures among just low scorers and high scorers; then one can swap the held-back measures for the ones used to sort the observations, and repeat.

The use of taxometrics has been growing literally exponentially, with over 80% of applications being in psychopathology. As of October 2009, there were over 300 taxometric investigations in the literature (including both substantive and methodological papers), starting in 1965 with Meehl's MAXCOV-HITMAX technical report. Of these, those concerning schizotypy/schizophrenia and those regarding depression are approximately tied for most popular psychopathological topics, with the latter having about 25 studies. Beach and Amir (2003), in a typical taxometric investigation of depression, tested whether finding "taxonicity" (a positive indication that a taxon was present in the data) was a function of the content of the manifest indicators. The hypothesis was that dimensions would be found with mild cognitive symptoms, whereas taxa would emerge when severe vegetative symptoms were studied. This was conducted in part because Ruscio and Ruscio (2000), leading taxometric investigators, had confidently described their own investigation as indicating that a mixed literature on the question of the taxonicity of depression should be interpreted as indicating dimensionality.

What Beach and Amir (2003) did was separate what were deemed to be "distress" or subjective/cognitive symptoms of depression from somatic/vegetative symptoms on the Beck Depression Inventory (BDI). The former had been used as the depression measures by Ruscio and Ruscio (2000), who found them to indicate a dimension rather than a taxon. The technique of using *X* for a single item and *Y* likewise, and computing their covariance over intervals on *Z* (the sum of all other items) as a MAXCOV-HITMAX variant, was used. College undergraduate subjects were used, who would be expected to have a low rate of depression (if there was an entity called depression at all). Just as in the Ruscio and Ruscio analyses, Beach and Amir found no evidence at all for taxonicity in the "distress" items from examining the covariance curves. Consistency tests were not calculated, as these are relevant for deciding when an apparent finding of a taxon is misleading (a false positive). However, in other analyses indicating a taxon under different measures (not discussed here), the consistency tests calculated were quite limited, barely scraping the surface of the numerous tests Meehl worked out for MAXCOV-HITMAX.

What we see here is typical of taxometric reports; indeed, it is slightly better than usual. A single source of data is used to yield all the information in the taxometric analysis, violating Meehl's precept about diversity of variables. The population studied is chosen without regard to the likely prevalence of the putative taxon (i.e., current major de-

pression), which should by any reasonable account be present in considerably less than 10% of the population, violating Meehl's advice about the base rate. The variables studied are eight individual questionnaire items summed (less two kept out to serve as *X* and *Y* variables), so that the key *Z* variable used to order observations on probability of taxon membership has only seven possible scores. This either violates or grazes the boundary of the dictum about studying continuous variables. The advice about obtaining indicators that differentiate the taxon from the complement class by 2 *SD*s each is almost surely violated, as it seems hardly conceivable that eight questionnaire items each distinguish depressed from nondepressed individuals by this much. (As a rough check on this, one can look at mean BDI scores among diagnosed depressed and control subjects: Depressed subjects coming into therapy trials commonly score about 35 [*SD* about 6], and control subjects commonly score 4–6 [*SD* about 8]. On a per-item basis—the full BDI has 21 items—that is about 0.2 *SD* per item in pooled *SD* units.) Finally, the consistency test advice is violated, in that the 40-odd consistency tests Meehl originated for MAXCOV-HITMAX are not used, and instead just one consistency test of unclear structure and dubious derivation is used in Beach and Amir (2003).

What is surprising and dismaying is that the situation described above does not appear to be representative of the average taxometric study in the literature. It seems representative of almost *every* taxometric study in the literature. The promised benefits of taxometrics are not being delivered in the empirical literature by practitioners of taxometrics because, quite simply, scientists are not following the tough advice Meehl offered.

Clinical Utility of Dimensions versus Taxa

Dimensional models of psychopathology have been supported by Krueger and colleagues' research program, for those disorders they have included within their ambit. Although the scientific bases for these dimensional models is still nascent, given the considerations described above, we speculate in what follows about the impact of dimensional models on clinical practice if they were ultimately to be widely adopted and reimbursed by patients' insurance.

Apparently, after *one particular* dimensional model has been validated, and ensconced in psychological theory, it will be adopted in the clinic, and clinicians eventually will find it old hat to locate their patients in *d*-dimensional spaces. Patients' score vectors will be entered into the model for the sake of predicting treatment placement and prognosis, and everything from malingering to dangerousness may well be predicted by a dimensional model space.

Krueger and colleagues draw conclusions throughout their research about the "best" model, and argue that DSM-V should adopt the model best supported by the current evidence. At first blush, this approach appears reasonable—and, as scientific realists, we are inclined to believe that there exists reality, and that one particular model will come to represent that reality best. On the other hand, because we are discussing the "soft" field of clinical psychology, we surmise that *any* "best" global model of psychopathology is at best a considerable simplification of the true nature of psychopathological constructs, regardless of the model fit index.

Often there will be two or more candidate models that fit relatively well (e.g., their BIC values will be very close), and in these cases the posterior probability assigned to the best model will be very close to that of the second-best and perhaps even the third-best. Burnham and Anderson (2005) provide evidence that single models, even the "best" models, have inferior predictive accuracy compared to "averaged" models, where averaging takes the form of weighting each model by its BIC posterior probability (or AIC posterior weights). When BIC is expressed as $-2\log(L) + \log(n)K$, the posterior probability of model g_i is

$$\Pr(g_i(\theta)|X = x) = \frac{\exp\left(-\frac{\Delta\mathrm{BIC}_i}{2}\right)q_i}{\sum_{j=1}^{n}\exp\left(-\frac{\Delta\mathrm{BIC}_j}{2}\right)q_j}, \; i, j = 1, \ldots, n \tag{15.3}$$

(Raftery, 1995), where q is the prior probability, and $\Delta\mathrm{BIC}_i = \mathrm{BIC}_i - \min(\mathrm{BIC}_1, \ldots, \mathrm{BIC}_n)$, the BIC difference between the *i*th model and the best model. If $\Delta\mathrm{BIC}_i$ is small for each model under consideration (e.g., ≤10), then an averaged model (with each

model weighted by $\Pr(g_i(\theta)|X = x)$) often results in more accurate prediction than the single best model. Burnham and Anderson claim that this effect will hold generally, and does hold for the research and simulation studies with which they are familiar and/or have conducted. This result should not be all that shocking. Multimodel inference (model averaging) uses more information than the single "best" (but typically far from true) model, and infelicities in the best model may be countervailed by including different models that have nonoptimal, but still valid, variables/parameters not included in the single best model. That is, the multimodel approach throws away less information, and retains the uniquely valid portions of valid but nonoptimal models. Therefore, as the models are being used to make novel predictions, the multimodel approach is best equipped to deal with novel infelicities in the data because it contains more structural flexibility than the single-model approach. Note that we have said nothing about what kinds of models are averaged. The multimodel method is a vegetable soup concerned only with the predictive validity of the final averaged model. It may very well be the case that dimensional and categorical models are included in the set of "good" candidate models, and that each is incorporated into the averaged model.

Unfortunately, we are not aware of any investigations in the psychopathological literature that use multimodel methods, and are unaware of the expected gain in predictive accuracy obtainable through the multimodel approach. In any event, if Burnham and Anderson's (2005) results are truly general, then a purely instrumentalist argument for adoption of a particular dimensional model for DSM-V fails, as no single dimensional model will predictively outperform an averaged model.

On the other hand, we imagine that it will be extremely difficult for many scientific realists (including, we believe, a large proportion of practicing clinicians) to adopt a multimodel approach. The notion of averaging across structurally disparate models, and dispensing with the idea that any single model works best, is likely to be a foreign idea to nearly all researchers and practicing clinicians; the complete lack of multimodel approaches in clinical psychology attests to this.

In a similar albeit single-model spirit, Grove (1991) compared the predictive accuracy of dimensional models versus categorical ones. In short, he compared the predictive accuracy of two decision strategies from dimensional data: (1) the predictive validity of continuous (i.e., dimensional) predictor x for criterion y; and (2) sorting individuals by diagnosis on the basis of continuous predictor x, then using these dichotomous diagnoses alone to predict y. Grove found, for nearly the entire parameter space, that predicting y straight from the dimensional variable x outperformed using the intermediate diagnostic categories. These results are of great importance in clinical psychology, as dimensional models will predictively outperform categorical models, regardless of true latent structure. In our opinion, this is a very strong practical argument for use of dimensions in DSM-V. However, it must be remembered that Grove's analysis was in no way evidence that the true structure of psychopathology is dimensional (or categorical).

The accurate prediction of past, present, and future events is central to psychology, and theories or models that allow clinicians to make more accurate determinations of future behavior, diagnosis, treatment placement, and prognosis would, *ceteris paribus*, be more useful. The construction of DSM-V is, in the broad sense, political, and the allocation of large sums of grant money and other resources will be influenced by the book produced. As such, considerations other than the substantive grounding of any theoretical or model-based approach will play only a partial role in its construction. Careful analysis of nonscientific influences is beyond the scope of this chapter, but we wish to conclude with some remarks on day-to-day aspects of clinical practice that would change—and perhaps become more difficult—if DSM-V were radically transformed from the categorical approach currently espoused to a dimensional one.

Shortcomings of Dimensional Models in Day-to-Day Clinical Activity

Current clinical decisions are typically (always?) sets of dichotomous decisions. The decision to administer one particular treat-

ment regimen instead of another is dichotomous, as is the decision to discharge a patient to outpatient mental health follow-up, or to discharge a patient to a domestic violence support group.

When categories (as opposed to dimensions) are used for clinical description/diagnosis, the descriptions are structurally isomorphic to decisions. Such a simple mapping makes it easy for today's clinician (as well as the hospital's financial wing) to operate, whatever the obtained decrement in predictive accuracy.

Categories not only map onto clinical decisions, but they are easier to work with from the outset. A simple illustration is that it takes 1 bit of information to encode a category (when dichotomous). Compare this to 6 bits for the BDI score, 7 bits for an MMPI-2 scale score, and 8 bits for a Wechsler Adult Intelligence Scale—Third Edition Full Scale IQ score. To encode the entirety of a recent MMPI-2 report we saw, it would take about $63 \times 7 = 441$ bits, not counting Harris–Lingoes scales (not that we personally recommend interpreting all 63 of these scales). As is obvious, depending on the dimensionality and number of discrete values per dimension under consideration, the ability of a human judge to consider dimensional information becomes prohibitively difficult—especially in comparison to the very little effort necessary to remember and process categorical information. In addition, models such as those Krueger and colleagues propose are not simply *n* orthogonal dimensions. Rather, they are hierarchical, and nonindependence between scales must be considered by the clinical judge. Obvious in this computing illustration is that categories can throw away tremendous amounts of information about the patient; the effect of this loss of information is precisely what Grove (1991) investigated, as described above.

Shortcomings of Dimensional Models as General Theories of Psychopathology

The range of sampling convenience of research on the dimensional approach is mostly within the normal to mildly abnormal range—that is, from populations not selected for psychopathology (such as college students, twins and their relatives, and outpatient psychiatric populations). The problem range of convenience consists largely (if not wholly) of depressive, anxious, substance abuse/dependence, and conduct problems. Other personality pathologies, eating disorders, somatoform disorders, and other kinds of impulse control problems (e.g., pathological gambling, trichotillamania) are much less often studied.

More to the point, we have yet to see an attempt at validating internalizing and externalizing models with severe psychopathology such as that seen on inpatient psychiatry units. Never has any effort been made to incorporate schizophrenia into the range of convenience of such a theory. It seems also to exclude, as a corollary, at least the form of schizotypal personality disorder that is familially associated with schizophrenia. Bipolar I disorder is not included, and perhaps bipolar II is out as well. We do not know the status of cyclothymia among proponents of overarching dimensional models, but it is not a diagnosis we have seen mentioned in the more influential papers on this topic. It is even unclear at this point what would be the status of syndromes like psychotic depression, for which one of us (William M. Grove) has seen frequencies among inpatients with major depression of several percent to 10%.

As a result of these exclusions, it may be fair to describe current influential dimensional theories as rather less than overarching or complete theories of the structure of psychopathology. If current dimensional models are truly scientifically legitimate, they would be pointedly restricted to substance use, conduct problems, and the more common (and typically milder) forms of depression and anxiety.

There are quite significant measurement problems associated with the dimensional approach to assessment, which do not exist with the current categorical approach. At least this is true if the dimensional approach is tied to the use of self-report instruments, as it seems to be in virtually all research supporting this agenda. The first problem is that a not inconsiderable number of patients cannot complete the self-report assessment device, owing to cognitive or psychotic symptomatology. This may not be as high a hurdle in the end, as most of the psychiatric disorders addressed by dimensional approaches (e.g., Krueger and colleagues) do not render patients incapacitated.

A second problem is that perhaps an even larger number of patients will complete the self-report assessment device invalidly, due either to minimization or exaggeration of psychopathology, or to random or inconsistent responding. The better-constructed instruments (e.g., the MMPI-2) may reliably detect most instances of such deviant responding, but this only means that the protocols will not be erroneously interpreted as valid measures of various dimensions; telltale values on validity scales do not supply different, valid trait measures in place of the invalid ones. In many cases, a valid second testing cannot, for various reasons, be obtained to substitute for the first invalid testing. With the DSM system of categories, very few patients cannot be assessed because the categories are not tied only to self-report questionnaires but include clinician judgments.

It might be replied that problems with self-report questionnaires are not problems intrinsic to the adoption of a dimensional approach, and this reply would be quite correct. Nor is a V-8 engine intrinsic to the Cadillac automobile; GM could build it with a V-4, since it makes V-4 engines. But it so happens that GM always and only puts a V8 in the Caddy, so it is sensible to talk about the advantages and disadvantages of a V-8 along with other features of the Cadillac, until an alternative powerplant becomes widely available.

The use of self-report questionnaires does carry certain advantages. It may be easier, for example, to concoct and implement prediction and decision algorithms, which have been shown in a venerable line of research to be predictively more accurate than clinician judgments (Grove et al., 2000; Meehl, 1954). A next step in the dimensional program would be to quantify clinician judgments to augment self-report in determining a patient's psychopathology vector.

Reconciliation in Clinical Practice of Dimensional and Categorical Approaches

With few exceptions, diagnosis in psychology and psychiatry has been categorical. Patients are determined either to have some disorder or not. This basic categorical administrative structure, so embraced by clinics and insurance bureaucrats alike, is still tenable under the dimensional model.

Any dimensional model can be "cut" in various places to create administratively and practically useful categories. It is of course mathematically impossible to map a finite (and usually very small) number of treatment alternatives onto a mathematically *continuous* dimension without creating intervals (categories) along that dimension. Thus, in the face of necessarily categorical decision making (e.g., can a patient be discharged from the hospital or not?), the dimensional model will be broken into categories.

The advantage of the dimensional model is that we can retain for future clinical decisions each patient's original vector within the k-dimensional space. As Grove (1991) has shown, one loses a significant amount of predictive accuracy when dichotomizing a continuous function. If the patient's vector is retained after cutting, and that vector is clinically useful—Krueger and colleagues may venture to say that it contains most of the clinically useful information about a patient—then the same vector can be used to make treatment placement decisions, to determine prognosis within each treatment condition, and to make various other clinical decisions. The vector is never thrown away, and can be compared and/or updated with future information.

Whether substantively grounded or not, the dimensional approach will render clinical decision making more accurate, and a patient's disposition will be made on all (or a lot, at least) of the information available, instead on a possibly arbitrary distillation of the patient's suffering into diagnostic categories. Disregarding for now the multimodel approach discussed above, we believe that dimensional models will result in increased predictive accuracy and facilitation of mechanical prediction algorithms. At the same time, current clinical categories can still be useful to communicate about patients—both to medical doctors and to laity, as well as to the patient him- or herself. Even if dimensional models were clearly scientifically justified, we could never imagine asking a patient during a feedback session to visualize, for the moment, a 15-dimensional space of mild psychopathology, and to locate his or her vector $\mathbf{v} = \langle a_1, \ldots, a_{15} \rangle$.

Conclusions

Both dimensional and taxonic theories about psychopathology have a long way to go. The methods that currently dominate the scientific scene are limited to addressing Meehl's (2002) parsimony1, the curve-fitting problem. The BIC and the AIC use estimated expected K-L information loss as their guide to parsimony and model fit, but other mathematically defensible paradigms exist, including the VC dimension, which we discuss briefly above. It is currently unclear why one such method should trump another. Researchers using the BIC contend that it is also related to the Bayes factor, and that posterior probabilities of the best K-L model can be derived. We submit that the BIC is not the only Bayes solution to model selection, and it is currently unclear why the BIC should be preferred to other methods (Gelfand & Dey, 1994).

However, we are not seeing the forest for the trees. The purpose of this chapter has been to suggest that even if the BIC were the answer to model selection, which is most surely unclear at this point, it still only provides *partial* evidence for a particular substantive *theory*. Models are almost never isomorphic to theories. They come with idealizations that lie outside the substantive core of the theory. Any model test will ipso facto never directly test the core of any theory (e.g., psychopathology is dimensional). Bad K-L models may fit as such due to false peripheral assumptions (e.g., local independence) that have little to do with dimensionality or taxonicity per se.

Too often in psychology, we lose our appropriate scientific skepticism and swoon to a popular theory of the day, despite what is really a paucity of evidence either for or against it. The current dimensional push (e.g., Volume 114 of the *Journal of Abnormal Psychology* and references therein) is quantitatively sophisticated, but remains limited largely to psychometric data and analysis about nonpatient and outpatient samples. The models posited by this push are very simple, and most certainly very far from the truth, but in some cases they fit the data better than DSM-based models (at least by the BIC under assumption of equal priors).

Dimensional models probably do possess greater predictive accuracy than their categorical counterparts. However, we have seen that purely instrumentalist arguments ultimately support the use of multimodel approaches over particular dimensional models, although we presume that the multimodel approach will not be well received by practicing clinicians, despite the expected gain in descriptive and predictive accuracy. In our opinion, clinicians would do well to increase their predictive and descriptive accuracy, but certain practical problems do crop up under a dimensional model.

Dimensions can be easily turned into categories, but not the other way around. If adopted, dimensions should be cut into clinically meaningful intervals in clinical practice, thus automatically norm-referencing multivariate scores as well as minimally taxing existing clinical and administrative infrastructure. In practice, each *n*-dimensional vector would always be retained, and all dimensional information could be used in future predictions. Information gathered thenceforth could be used to update each patient's vector.

DSM-V will be far from a purely scientific document. The framers of the final manual must balance scientific, practical, and political concerns in developing diagnostic criteria, whether dimensional or categorical. This chapter reports only on the first two concerns; we defer political discussions to more adventurous authors. Substantively, we have argued throughout that neither taxonic nor dimensional approaches are anything but nascent, that multiple methodological approaches should be adopted, and that reliance on any particular statistic is insufficient to test a theory. Multimethod approaches in numerical taxonomy should proceed in the taxometric spirit, and consistency between methods should be taken to be evidence of a taxonic structure. "Consistency tests" need not be taxometric in origin. The results of any bona fide detector of latent structure (e.g., the BIC or taxometrics) can be compared. Consistency between results is increased evidence for a particular structure, whereas inconsistency can suggest many things, such as (1) that the assumptions of one or more methods is not being met; (2) that the *type* of latent structure (either dimensional or taxonic) is not within the range of detection of one or more methods; or even perhaps that (3) the

latent structure truly is not taxonic (or dimensional).

Acknowledgments

We would like to thank Andreas O. Robinson for helpful discussions, and to acknowledge valuable editorial feedback from Niels G. Waller.

References

Akaike, H. (1974). A new look at the statistical model identification. *IEEE Transactions on Automatic Control*, *19*, 716–723.

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Bartholomew, D. J. (1987). *Latent variable models and factor analysis*. London: Griffin.

Beach, S. R. H., & Amir, N. (2003). Is depression taxonic, dimensional, or both? *Journal of Abnormal Psychology*, *112*, 228–256.

Birnbaum, A. (1962). On the foundations of statistical inference. *Journal of the American Statistical Association*, *57*, 269–326.

Brantly, M. L., Paul, L. D., Miller, B. H., Falk, R. T., Wu, M., & Crystal, R. G. (1988). Clinical features and history of the destructive lung disease associated with alpha-1antitrypsin deficiency of adults with pulmonary symptoms. *American Review of Respiratory Disease*, *138*, 327–336.

Burges, C. J. C. (1998). A tutorial on support vector machines for pattern recognition. *Data Mining and Knowledge Discovery*, *2*, 955–974.

Burnham, K. P., & Anderson, D. R. (2003). *Model selection and multi-model inference*. New York: Springer.

Burnham, K. P., & Anderson, D. R. (2005). Multimodel inference: Understanding AIC and BIC in model selection. *Sociological Methods and Research, 33*, 261–304.

Cox, D. W. (1989). Alpha-1 antitrypsin deficiency. In C. R. Scriber, A. L. Beaudet, W. S. Sly, & D. Valle (Eds.), *The metabolic basis of inherited disease* (7th ed., pp. 2409–2437). New York: McGraw-Hill.

Elliott, P. R., Bilton, D., & Lomas, D. A. (1998). Lung polymers in Z α_1-antitrypsin deficiency-related emphysema. *American Journal of Respiratory Cell Molecular Biology*, *18*, 670–674.

Forster, M. R. (2006). Counterexamples to a likelihood theory of evidence. *Minds and Machines*, *16*, 319–338.

Gelfand, A. E., & Dey, D. K. (1994). Bayesian model choice: Asymptotics and exact calculations. *Journal of the Royal Statistical Society*, *56*, 501–514.

Gillies, D. (1993). The Duhem thesis and the Quine thesis. In M. Curd & J. A. Cover (Eds.), *Philosophy of science in the twentieth century* (pp. 98–116). Oxford, UK: Blackwell.

Grove, W. M. (1991). When is a diagnosis worth making?: A comparison of two statistical prediction strategies. *Psychological Reports*, *68*, 3–17.

Grove, W. M. (2004). The MAXSLOPE taxometric procedure: Mathematical derivation, parameter estimation, consistency tests. *Psychological Reports*, *95*, 517–550.

Grove, W. M. (2007). Dimensional analyses of taxonic data. *Psychological Reports*, *101*, 617–635.

Grove, W. M., Waller, N. G., & Vrieze, S. I. (2009). *Reply to Maraun and Slaney (2005) "An analysis of Meehl's MAXCOV-HITMAX procedure for the case of continuous indicators."* Manuscript submitted for publication.

Grove, W. M., Zald, D. H., Hallberg, A. M., Lebow, B., Snitz, E., & Nelson, C. (2000). Clinical versus mechanical prediction: A meta-analysis. *Psychological Assessment*, *12*, 19–30.

Kass, R. E., & Raftery, A. E. (1995). Bayes factors. *Journal of the American Statistical Association*, *90*, 773–795.

Kecman, V. (2001). *Learning and soft computing: Support vector machines, neural networks, and fuzzy logic models*. Cambridge, MA: MIT Press.

Krueger, R. F. (1999). The structure of common mental disorders. *Archives of General Psychiatry*, *56*, 921–926.

Krueger, R. F., Hicks, B. M., Patrick, C. J., Carlson, S. R., Iacono, W. G., & McGue, M. (2002). Etiologic connections among substance dependence, antisocial behavior, and personality: Modeling the externalizing spectrum. *Journal of Abnormal Psychology*, *111*, 411–424.

Krueger, R. F., Markon, K. E., Patrick, C. J., Benning, S., & Kramer, M. (2007). Linking antisocial behavior, substance use and person-

ality: Towards a comprehensive quantitative model of adult externalizing spectrum. *Journal of Abnormal Psychology*, *116*, 645–666.

Krueger, R. F., Markon, K. E., Patrick, C. J., & Iacono, W. G. (2005). Externalizing psychopathology in adulthood: A dimensional-spectrum conceptualization and its implications for DSM-V. *Journal of Abnormal Psychology*, *114*, 537–550.

Kullback, S., & Leibler, R. A. (1951). On information and sufficiency. *Annals of Mathematical Statisics*, *22*, 79–86.

Lakatos, I. (1980). *Philosophical papers: Vol. 1. The methodology of scientific research programmes*. Cambridge, UK: Cambridge University Press.

Meehl, P. E. (1954). *Clinical versus statistical prediction: A theoretical analysis and a review of the evidence*. Minneapolis: University of Minnesota Press.

Meehl, P. E. (1965). *Detecting latent clinical taxa by fallible quantitative indicators lacking an accepted criterion* (Report No. PR-65-2). Minneapolis: University of Minnesota, Research Laboratories of the Department of Psychiatry.

Meehl, P. E. (1968). *Detecting latent clinical taxa: II. A simplified procedure, some additional HITMAX cut locators, a single-indicator method, and miscellaneous theorems* (Report No. PR-68-4). Minneapolis: University of Minnesota, Research Laboratories of the Department of Psychiatry.

Meehl, P. E. (1978). Theoretical risks and tabular asterisks: Sir Karl, Sir Ronald, and the slow progress of soft psychology. *Journal of Consulting and Clinical Psychology*, *46*, 806–834.

Meehl, P. E. (1990). Why summaries of research on psychological theories are often uninterpretable. *Psychological Reports*, *66*, 195–244.

Meehl, P. E. (1992). Cliometric metatheory: The actuarial approach to empirical, history-based philosophy of science. *Psychological Reports*, *71*, 339–467.

Meehl, P. E. (2002). Cliometric metatheory: II. Criteria scientists use in theory appraisal and why it is rational to do so. *Psychological Reports*, *91*, 339–404.

Meehl, P. E. (2004). Cliometric metatheory: III: Peircean consensus, verisimilitude, and asymptotic method. *British Journal for the Philosophy of Science*, *55*, 615–643.

Meehl, P. E., & Yonce, L. J. (1994). Taxometric analysis: I. Detecting taxonicity with two quantitative indicators using means above and below a sliding cut (MAMBAC procedure). *Psychological Reports*, *74*, 1059–1274.

Norman, W. T. (1963). Toward an adequate taxonomy of personality attributes: Replicated factor structure in peer nomination personality ratings. *Journal of Abnormal and Social Psychology*, *66*, 574–583.

Popper, K. R. (2002). *Logic of scientific discovery* (J. Freed & L. Freed, Trans.). London: Routledge & Kegan Paul. (Original work published 1937)

Raftery, A. (1995). Bayesian model selection in social research. In P. V. Marsden (Ed.), *Sociological methodology* (pp. 111–195). Oxford, UK: Blackwell.

Ruscio, J., & Ruscio, A. M. (2000). Informing the continuity controversy: A taxometric analysis of depression. *Journal of Abnormal Psychology*, *109*, 473–487.

Salmon, W. C. (1984). *Scientific explanation and the causal structure of the world*. Princeton, NJ: Princeton University Press.

Schwarz, G. (1978). Estimating the dimension of a model. *Annals of Statistics*, *6*, 461–464.

Tellegen, A., & Waller, N. G. (2008). Exploring personality through test construction: Development of the Multidimensional Personality Questionnaire. In G. J. Boyle, G. Matthews, & D. H. Saklofske (Eds.), *The Sage handbook of personality theory and assessment: Vol. 2. Personality measurement and testing* (pp. 261–292). Thousand Oaks, CA: Sage.

Vapnik, V., & Chervonenkis, A. (1971). On the uniform convergence of relative frequencies of events to their probabilities. *Theory of Probability and Its Applications*, *16*, 264–280.

Waller, N. G., & Meehl, P. E. (1998). *Multivariate taxometric procedures: Distinguishing types from continua*. Newbury Park, CA: Sage.

Whewell, W. (1858). *The history of scientific ideas* (2 vols.). London: Parker.

CHAPTER 16

A Short History of a Psychiatric Diagnostic Category That Turned Out to Be a Disease

ROGER K. BLASHFIELD
JARED KEELEY

To all the People and Inhabitants of the United States and all the outlying Countries, Greetings:

I, John Michler, King of Tuskaroras, and of all the Islands of the Sea, and of the Mountains and Valleys and Deserts; Emperor of the Diamond Caverns, and Lord High General of the Armies thereof; First Archduke of the Beautiful Isles of the Emerald Sea, Lord High Priest of the Grand Lama, etc., etc., etc.: Do issue this my proclamation. Stand by and hear, for the Lord High Shepherd speaks. No sheep have I to lead me around, no man have I to till me the ground, but the sweet little cottage is all of my store, and my neat little cottage has ground for the floor. No children have I to play me around, no dog have I to bark me around, but the three-legged stool is the chief of my store, and my neat little cottage has ground for the floor.

Yea, verily, I am the Mighty King, Lord Archduke, Pope, and Grand Sanhedrim, John Michler. None with me compare, none fit to comb my hair, but the three-legged stool is the chief of my store, and my neat little cottage has ground for the floor. John Michler is my name. Selah!

I am the Great Hell-Bending Rip-Roaring Chief of the Aborigines! Hear me and obey! My breath overthrows mountains; my mighty arms crush the everlasting forests into kindling wood; I am the owner of the Ebony Plantations; I am the owner of all the mahogany groves and of all the satin-wood; I am the owner of all the granite; I am the owner of all the marble; I am the owner of all the owners of Everything. Hear me and obey! I, John Michler, stand forth in the presence of the Sun and of all the Lord Suns and Lord Planets of the Universe, and I say, Hear me and obey! I, John Michler, on this eighteenth day of August, 1881, do say, Hear me and obey! for with me none can equal, no, not one, for the three-legged stool is the chief of my store, and my neat little cottage has ground for the floor. Hear me and obey! Hear me and obey! John Michler is my name.

John Michler, First Consul and Dictator of the World, Emperor, Pope, King and Lord High Admiral, Grand Liconthropon forever! (quoted in Hammond, 1883/1973, pp. 603–604)

This proclamation, when shown to modern psychiatrists and clinical psychologists, is typically diagnosed as having been written by someone who either has schizophrenia or is having a manic episode with grandiose delusions. Interestingly, even with the year of 1881 listed in the proclamation, virtually no modern clinicians speculate on the

actual diagnosis of John Michler. According to William Hammond, who included this proclamation in his 1883 textbook on psychopathology, Michler was an example of a patient with "general paresis," or what might now be called "neurosyphilis." At that time, general paresis was a common form of psychosis that accounted for as much as one-quarter of all psychiatric inpatient hospitalizations (Dowbiggin, 1991; Shorter, 1997).

Paresis has been largely ignored in modern times, despite its potential use as an informative example in some of the current major debates in the field. In this chapter, we attempt to outline one of those debates—the view of psychopathological disorders as essentialistic categories—in the context of the discovery, investigation, and eventual clarification of paresis as a disorder. As we demonstrate, paresis is a prototype of an essentialistic category, but even then essentialism has several shortcomings in its description of the disorder. Instead, the story of paresis suggests that the models of investigation that led to its resolution may be "blind alleys" for the progress of the field with less essentialistic disorders.

In her anthropological analysis of psychiatry, Luhrmann (2000) has represented the field as being split into two theoretical camps: one that favored the mentalistic approach to psychiatry of psychoanalysis and the resulting emphasis on psychotherapy as the preferred mode of treatment, and a second, which favored the neuroscientific view of biological psychiatry and its emphasis on drug therapies as the preferred mode of treatment. Haslam (2000) has extended Luhrmann's analysis to suggest that the biological approach to psychiatry favored a view of mental disorders in which mental disorders were assumed to be the psychological and behavioral results of "essential," underlying biological malfunctions in the brains of individuals with these disorders. He has commented: "Luhrmann argues [that] the struggle [between psychoanalysis and biological psychiatry] is coming to an end, not in a grand and hopeful synthesis or a comfortable pluralism, but in the overwhelming victory of the biomedical orientation" (p. 1031).

Haslam and his colleagues have published a series of papers describing their view of essentialism and the relevance of this concept for how we understand psychopathology (Haslam, 2000; Haslam & Ernst, 2002; Haslam, Rothschild, & Ernst, 2000). The biomedical approach to psychopathology conceptualizes mental disorders as diseases, or at least as potential diseases, with causes that ideally will be determined through biological research. This approach assumes that mental disorders represent "natural kinds" in which there are underlying essences or causal mechanisms, which will explain each disorder and the ways in which the disorders are behaviorally, socially, and interpersonally represented.

From Haslam's perspective, essentialism involves a set of nine associated beliefs that he and his colleagues (Haslam et al., 2000; Haslam & Ernst, 2002) have distilled from the writings of others who have attempted to enunciate what "essentialism" means (Hirschfeld, 1996; McGarty, Haslam, Hutchinson, & Grace, 1995; Rothbart & Taylor, 1992; Yzerbyt, Rogier, & Fiske, 1998). These nine beliefs are as follows (Haslam et al., 2000):

1. *Discreteness*—the category has "clear and sharp boundaries."
2. *Uniformity*—members of the category are "very similar to one another."
3. *Informativeness*—knowing that someone is a member of the category "tells us a lot about the person."
4. *Naturalness*—the category corresponds to something that exists in the real world, "rather than just being an artificial product of people's efforts" to categorize.
5. *Immutability*—the characteristics defining the category cannot be changed.
6. *Historical invariance*—the category has existed over time and across cultural contexts.
7. *Necessary features*—the category can be defined by characteristics that an individual must have in order to belong to the category.
8. *Inherence*—there is an "underlying reality" to the category.
9. *Exclusivity*—an individual cannot be a member of more than one category in the classification. (This item was dropped in Haslam & Ernst, 2002.)

Although there have been various theoretical discussions about the meaning of the

related concepts of essentialism, disease, and natural kinds (see Kendell, 1975; Reznek, 1987; Zachar, 2000), one of Haslam's distinctive contributions to this literature has been to study the views of laypeople about the relative degree of essentialistic thinking about natural categories. In this regard, Haslam's research drew on an interesting earlier empirical study by Campbell, Scadding, and Roberts (1979). These investigators asked four groups of subjects (nonmedical academics, high school students, medical academics, and general practitioners) to rate their degree of certainty that each of 38 different medical conditions was a "disease." At the top of the list for all four groups were medical conditions that resulted from bacterial infections (e.g., malaria, tuberculosis, syphilis); in the middle were conditions that the medical and nonmedical groups disagreed about (including hemophilia, cirrhosis of the liver, schizophrenia, and depression); and at the bottom were medical conditions that most of the groups either did not view as diseases, or at best were unsure about (e.g., color blindness, drowning, and starvation). In other words, conditions defined by bacterial infections are the best representatives of "diseases" as most people use the term.

An ideal prototype of a disease associated with psychopathology is the disorder known as paresis. The cause of paresis is tertiary syphilis, in which the syphilitic bacillus, *Treponema pallidum*, has been present in the body of the infected individual for 20–30 years and the infection has invaded the central nervous system. When bacilli enter the brain, the course of what had been a dormant disease changes relatively quickly. The initial symptoms of the disorder are psychotic symptoms, such as delusions, disordered thought processes, behavioral disruptions, and hallucinations. As the disorder progresses, the patient begins to develop progressive paralysis of the extremities. The dementia of the patient will increase over time. If the disease goes untreated, the course of paresis is typically rather short, progressing from the initial onset to death in 2–5 years. Without treatment, death is the inevitable result of the disorder. Because paresis is known to be caused by a bacterial infection, paresis can be viewed as a "poster child" for an essentialistic view of psychopathology. Paresis fits the 19th-century dictum proposed by Griesinger that all mental disorders are diseases (Shorter, 1997).

Paresis has been known by a variety of names, including "chronic meningitis," "dementia paralytica," "general paralysis of the insane" (GPI), "general paresis," "tertiary syphilis of the central nervous system," and "progressive paralysis." We use these terms interchangeably in this chapter.

The discovery of paresis, the eventual determination of its etiology, and attempts to treat this disorder represent an instructive example of how thinking about a mental disorder evolved as knowledge about this disorder changed. As far as we know, paresis was the first mental syndrome that was associated with a known change in the brains of the individuals who had the disorder. Before the initial discovery of paresis, physicians working with these individuals were aware that autopsies conducted on a number of them showed abnormalities in their brains. However, the discovery of paresis was the first instance in which a collection of symptoms prior to death was correlated with a type of brain pathology found after death.

The Discovery of Paresis

Bayle's Early Work in France

In 1822, a young French physician named Antoine-Laurent Bayle published a thesis describing autopsies he had performed on a small set of patients who initially presented with grandiose delusions and excited, disruptive behaviors (Brown, 1994). Many of these individuals were men in their 30s to 50s who held middle-class or upper-class positions in France and who suddenly became psychotic. Motor paralysis invariably occurred as the disorder progressed before these individuals died. When Bayle examined the brains of these men, he found that the size of their brains had markedly shrunk relative to the size of their skull vaults. In addition, these individuals had notable changes in the "skin" of the brain, known as the meninges, which appeared to be inflamed. Bayle theorized that these individuals had some disease of the meninges that had led to their mental disorder. Hence the name that he initially gave to the disorder was "chronic meningitis."

Bayle's discovery attracted considerable attention in France at the time. His discovery occurred at a period when French medicine was very concerned with the treatment of insanity. Discussions of insanity were part of the popular press of that era. Paresis was a somewhat "fashionable" disorder in early 19th-century France; it was viewed as primarily affecting middle- to upper-class men, whereas many of the other mental disorders of the time seemed to occur more frequently in working-class individuals (Brown, 1994). A few years after publishing his initial finding, Bayle had analyzed a much larger collection of mental patients (a total sample size of almost 200 individuals). In a controversial paper that he published in 1826, Bayle claimed that all cases of paralysis in patients with insanity were caused by the lesions in the brain that he had documented. Interestingly, within a year after publishing this paper, Bayle lost his position as a physician working at a large French mental hospital. He eventually found a job as a medical librarian and remained in this role for the rest of his life.

The reason Bayle's work was not well received was that it went against the views of the dominant figures in French psychiatry: Philippe Pinel and his student Jean Esquirol. Pinel and Esquirol had gained political favor in the French Revolution because of their sympathetic stance toward the philosophy of Rousseau. This sympathy translated into their psychiatric work in what has come to be known as the "moral treatment" movement. Pinel, even though he was a physician, had been impressed by the impact of nonphysicians ("charlatans") when working with patients in insane asylums. The common-sense, often theatrical interventions by these nonphysicians appeared to Pinel to have a greater impact on these patients than did bleeding and purging—the standard treatments that physicians had to offer. In his asylum, Pinel gathered statistics on the effectiveness of his treatment approach. He documented a 93% cure rate for individuals with acute mania and depression when his moral treatment was applied. He did not keep data on the application of his moral treatment to chronic patients, however, because he believed that these individuals had some type of brain degeneration that would make them nonresponsive to moral (i.e., psychological) interventions (Goldstein, 1987).

Esquirol, as Pinel's student, continued to study the application of moral treatment. In particular, he studied a diagnosis he termed "monomania." Monomania was differentiated from the other manias because a patient's delusions were specific and limited in scope, and the patient could function quite normally except in reference to the subject matter of the delusions.

In this context, Bayle's thesis about paresis became a focus of controversy among the adherents of Esquirol. Bayle was discussing patients who appeared from his descriptions to have something similar to Esquirol's representation of monomania. However, Bayle was arguing this was not a moral/psychological disorder at all, but instead that the disorder was caused by an inflamation of the skin of the brain. As a young physician at the time, Bayle worked at one of the larger asylums in France. The medical director of this asylum was a conservative supporter of the French monarchy and an avowed opponent of the republican views of Pinel and Esquirol. When this leader died in 1825, Esquirol, probably because of his extensive analyses of the conditions of asylums across France, was appointed the new director of this asylum. Shortly afterward, Bayle was fired, and he left the profession of psychiatry. Interestingly, the antipathy toward Bayle was so strong among adherents of the Esquirol school that in the 1850s, they challenged the primacy of Bayle's discovery of paresis and argued that paresis was actually first described by one of their own. This political claim never gained general acceptance, and, as far as we can tell, is not viewed as a credible claim by modern historians of medicine.

General Paralysis

By the middle of the 19th century, paresis had become an internationally recognized mental disorder. The most commonly used name for the disorder in the mid-1800s among English-speaking professionals was "general paralysis of the insane" (which often was shortened to "general paralysis," or later abbreviated to GPI; Austin, 1859/1976). The exact origin of this name is unknown. The word "paralysis" emphasized that the definitive descriptive symptom of this mental

disorder, prior to death and confirmation by autopsy, was the progressive motor paralysis associated with this disorder. The root word for "paralysis" also appeared in another common 19th-century name for this disorder, "dementia paralytica."

During the mid-1800s, epidemiology was becoming an area of study and research within all areas of medicine. Statistics gathered by the physicians who worked in asylums suggested that general paralysis was a prevalent and frequently observed mental disorder. For instance, data from an asylum in Berlin, Germany, showed that one-third of the patients there were diagnosed with dementia paralytica (Engstrom, 2003). An asylum in Lancashire, Great Britain, reported that 17% of its admissions and 30% of its deaths were caused by general paralysis. In contrast, a rural asylum in Wales in 1896 only had 5% of its patient mix diagnosed with this disorder (Healy et al., 2005). A British psychiatrist reviewing the literature on general paralysis noted that it was a prevalent disorder in the 19th and early 20th centuries, especially in urban asylums (Robertson, 1913). He also commented that general paralysis accounted for half to two-thirds of the cases of insanity among German military officers.

General paralysis was also found to vary in frequency across countries and within sociocultural groups. For instance, general paralysis appeared to have its highest rates of prevalence during the latter half of the 19th century in Italy and Ireland. Since these two countries were also known to have high rates of alcoholism, this finding led some to speculate that general paralysis was a brain disorder caused by excessive alcohol use (Anonymous, 1894). General paralysis was also viewed as occurring most frequently in middle-aged men, especially men in the military (Austin, 1859/1976). Women were known to have the disorder, though less frequently than men (Austin, 1859/1976). The disorder was unheard of among Quakers and Roman Catholic priests.

Another interesting finding was the observation that the disorder was occasionally found in early adolescence. For instance, Wiglesworth (1883) reported a case of general paralysis in a 15-year-old girl who died 3 months after admission to a British asylum. The observation that general paralysis could occur in adolescents was initially used to reject the theory that general paralysis was caused by syphilis. Adolescents who were only 12–14 years old and who had developed general paralysis were unlikely to have acquired syphilis through prostitutes or other likely sexual carriers of this disease. By the early 20th century, however, the transmission of syphilis from mothers to children was recognized; thus a syphilitic cause of general paralysis was acknowledged as possible (Middlemass, 1904).

Syphilis as a possible cause for paresis was first proposed in France in 1857 by Esmarch and Jensen (DeFursac & Rosanoff, 1916; Nitrini, 2005). The basis for this proposal was correlational evidence: Paresis seemed to be prevalent in groups (especially males in the military) in which syphilis was also prevalent. Many individuals with general paralysis reported having had a history of syphilis. Nevertheless, Mercier (1902), in a British textbook about mental disorders, argued that syphilis could not be the only cause of general paralysis; he contended that negative histories of syphilis were obtained from about 20% of all individuals with general paralysis, and that some persons with untreated cases of syphilis did not eventually develop this mental disorder.

A German View of Dementia Paralytica: Kraepelin

Emil Kraepelin was a major figure in European psychiatry at the turn of the last century, and his ideas on the classification of mental disorders came to influence much of later psychiatry (Engstrom & Weber, 2007; Jablensky, 2007). In fact, the current classification system in use in the United States owes much of its organizational scheme to Kraepelin's ideas. Kraepelin's classification system was represented in the organization of chapters in his textbooks on psychopathology. In 1896, Kraepelin published the fifth edition of his textbook on psychiatry (*Psychiatrie: Ein Lehrbuch für Studirende und Aerzte*); the sixth edition was published in 1899. A psychiatrist working at the Connecticut Hospital for the Insane, A. R. Diefendorf, published an English translation of Kraepelin's textbook in order to bring Krae-

pelin's controversial but increasingly influential ideas to the United States (Kraepelin, 1918).

In the seventh edition of his textbook, Kraepelin covered dementia praecox (now known as schizophrenia) and dementia paralytica (which Kraepelin also labeled as paresis) in adjacent chapters (Kraepelin, 1918). These two sister forms of dementia, in Kraepelin's view, were prevalent disorders, with dementia praecox accounting for 14–30% of all admissions to asylums, and dementia paralytica occurring in 36–45% of such admissions. Kraepelin discussed three subtypes of dementia praecox: (1) hebephrenic, (2) catatonic, and (3) paranoid. His observations suggested that the gender distributions, ages of onset, and courses of these subtypes were varied. For dementia paralytica, Kraepelin noted four subtypes: (1) demented form, (2) expansive form, (3) agitated form, and (4) depressed form. Again, he argued that demographic and course differences were associated with these four forms.

Kraepelin argued that the important underlying characteristics of dementia paralytica were found in autopsy analyses of the brains of individuals with this disorder. He noted that the major gross anatomical finding was a distinct change in the meningeal tissues. In addition, there was a correlated reduction in brain weight, which Kraepelin noted as averaging between 1,150 and 1,300 grams, with some brains weighing as little as 800–900 grams. The ventricles were enlarged; the frontal lobes were often massively reduced; and atrophy was often noted in associated brain structures, such as the cerebellum, the basal ganglia, and the spinal chord.

Kraepelin listed many symptoms of paresis, including decreased ability to understand one's environment, clouding of consciousness, disorientation, increased sense of fatigue, hallucinations, defects of memory, replacement of memories by imagined events, impaired judgment, and delusions. Because these symptoms often occurred in other disorders (neurasthenia, dementia praecox, etc.), Kraepelin believed that the signs (physical symptoms) were more useful diagnostically. The behavioral indicators of dementia paralytica were frequent paralytic attacks (occurring in 46–60% of the cases); sensory changes, including optic atrophy and loss of skin sensation (analgesia); motor disturbances of the eye (including sluggish pupillary response to light in about one-third of the cases—i.e., Argyll–Robinson pupils); and marked behavioral disruptions in speech. Kraepelin noted that the course of the disorder invariably led to death in about 2 years after the initial onset, although the course could be marked with remissions, especially in the expansive subtype.

Kraepelin believed that dementia paralytica was caused by some type of toxin that was leading to the destruction of central nervous system tissues. He thought that the formation of this toxin could be stimulated by syphilis, since he noted this disease in slightly over one-third of the patients he observed. Kraepelin thought that syphilis was associated with hereditary forms of paresis, since most juveniles with paresis had parents with syphilis and with alcoholism. Kraepelin noted that Krafft-Ebing had performed an experiment in which he attempted to inoculate nine patients who had paresis with the blood from patients known to have syphilis, and none developed secondary syphilitic lesions (e.g., genital sores). This was significant, since at this time it had been established that patients infected with syphilis could not develop the disease a second time. However, Kraepelin also reported a study by Marchand, Gabiana, and Garbini (no reference was given in Kraepelin, 1918) in which seven patients with paresis developed syphilis after dementia paralytica was diagnosed. Other possible causes of the toxin that could lead to dementia paralytica, according to Kraepelin, were alcoholism (noted in 60% of his cases); head injury (23% of the cases); and the stresses of modern life with its "overactivity and insufficient relaxation, coincident with the struggle for existence in large cities" (Kraepelin, 1918, p. 279).

Kraepelin devoted a section in his chapter on dementia paralytica to differential diagnosis. Kraepelin separated dementia praecox from dementia paralytica on the basis of motor paralysis in the latter, as well as the different ages of onset in the two disorders. Dementia praecox, according to Kraepelin, had an age of onset before 25 in both men and women, whereas dementia paralytica generally occurred in men (4:1 ratio of men to women) between the ages of 30 and 50.

Determining the Etiology of Paresis

At the turn of the last century, a prominent British neurologist named F. W. Mott (1897, 1901) published a series of articles in the *Journal of Mental Science* (now known as the *British Journal of Psychiatry*) about paresis. He was a strong proponent of the syphilitic theory of paresis. Mott (1901) stated four arguments in favor of this theory: (1) Epidemiological statistics from various countries showed that where syphilis was relatively rare, paresis was also less frequently found; (2) epidemiological studies showed that paresis was very unusual in Catholic priests and in Quakers; (3) hereditary studies had not shown a strong constitutional factor, and hence the disorder must be caused by an active disease agent; and (4) in cases of adolescents with paresis, virtually all had parents who were discovered to have syphilis.

Mott (1897) also proposed a theory about the specific mechanism by which syphilis affected the brain. He performed a series of neuroanatomical studies on the degeneration of the heart in patients with paresis. From this research, he concluded that paresis was a general degeneration of neurons due to a long-standing toxic influence of syphilis. Mott proposed that paresis was a type of "meningio-encephalitis"—a name that harked back to Bayle's name for this disorder three-quarters of a century earlier.

Mott's theory, however, did not immediately gain widespread acceptance. Two lines of evidence against the syphilitic theory were commonly mentioned. First, descriptive studies of paretics, as Kraepelin noted in his textbook, were not able to confirm a history of syphilis in all patients with paresis. Clearly many such patients had been infected by syphilis, but many apparently had not. Second, the best treatment for syphilis at the time was the use of mercury compounds. Mercury, a toxic substance, seemed to arrest the course of the syphilitic disease. However, most attempts to use mercury with patients who has paresis failed to show any effect.

Between 1903 and 1907, two British researchers gathered evidence supporting a new etiological theory of paresis. Robertson and McRae (1907) argued that paresis was caused by a diphtheroid bacillus, which they named *Bacillus paralyticans*. They offered three pieces of evidence for their theory. First, they claimed that rats inoculated with this bacillus developed paresis-like symptoms. Second, sheep inoculated with the bacillus had a long gestation period before there was a high count of these bacilli in their blood (i.e., like the long gestation period for paresis). Third, they stated that blood samples taken from humans with paresis had shown the presence of these bacilli, but that blood samples taken from normal humans had not.

The resolution of the etiological questions about paresis occurred through a remarkable series of discoveries in the first decade of the 20th century (Quetel, 1990; Sherman, 2006). Despite many previous failures to generate animal models for syphilis, Metchinkoff and Roux were the first to transmit the infection to monkeys successfully in 1903. Schaudinn and Hoffman, working in Berlin, discovered the bacillus causing syphilis in 1905. They named this bacillus *Spirochaeta pallida*. In 1906, another German research group including Wassermann, Plaut, Neiser, and Bruck used a complement fixation test to analyze the serum of monkeys that had been infected with syphilis. Later the Wassermann research group was successful in discriminating humans who were known to have syphilis from those who did not. This test rapidly became a standard laboratory diagnostic test for the presence of syphilis. Later research, however, showed that the Wassermann test did lead to "false positives" (i.e., the test suggested that some individuals were positive for syphilis, but in fact these individuals did not have the disease).

The Wassermann group applied their test to the blood serum of individuals with paresis. The percentage of positive findings was high, but not perfect. Plaut (1911), who primarily worked for Kraepelin in Munich, decided to apply the Wassermann test to samples of cerebrospinal fluid from patients clinically diagnosed with paresis. He found that most of these patients had positive findings for the cerebrospinal fluid, whereas there were no false positives on tests of control individuals.

The final evidence for the syphilitic theory of paresis was published by Noguchi and Moore (1913), who were working at Columbia University in the United States. They

found the syphiltic bacilli in the brain sections of 12 of 70 patients with paresis that they examined. All 12 cases in which the bacilli were located had positive Wassermann tests of the cerebrospinal fluid. In addition, Noguchi and Moore were able to infect rabbits with syphilis by inoculating them with brain tissue from humans with paresis. This publication was viewed as convincing evidence for the syphiltic theory. The etiological mystery, which psychiatrists from the 1800s had spent a century trying to understand, was now solved.

Treatment of Paresis

With the etiology resolved, the search for a treatment began. Before it was definitely established that GPI was caused by syphilis, many of these patients were being treated with mercury, as noted above. A promising new pharmacological approach was proposed in the early 20th century. Ehrlich and Hata (see Brown, 2000) experimented with various chemical substances and their effects on bacterial organisms. In 1908, they conducted tests on arsephenamine (sold under the commercial name Salvarsan in 1910). Arsephenamine was a synthetic preparation containing arsenic and was lethal to *T. pallidum*. Although treatment with Salvarsan was a considerable improvement over treatment with mercury, the effect of this chemical on GPI was not as great as had been hoped (Brown, 2000; Nitrini, 2005).

Wagner-Jauregg, an Austrian, received the Nobel Prize in Medicine in 1927 for his treatment of paresis (Brown, 2000). Wagner-Jauregg noted the correlation between curing a mental disorder and fever. In 1917, he inoculated nine patients who had paresis with malaria, allowed them to develop a high fever, and then treated them with quinine, which was known at the time to be an effective treatment for malarial infections. Six of the nine patients showed full remission, and the remaining three cases showed vast improvement. Many of Wagner-Jauregg's patients were given neoarsphenamine, a tweaked compound of the original arsphenamine, in conjunction with the malaria treatment.

The malaria treatment proved promising, but it was also noted that many of those who recovered were in the early stages of paralysis. This stage was easy to determine, as the latent syphilis could be detected with a positive Wassermann test.

The malaria treatment and the arsenic-derived drugs remained the treatments of choice until the advent of World War II. The next advance in treatment started when Alexander Fleming discovered penicillin in 1928. However, he quickly discovered that penicillin was not able to stay in the body long enough to have any effect on bacteria, due to the rapid renal clearance of the drug. He stopped studying it after this discovery in 1931, but resumed his research in 1934.

It was not until World War II that penicillin was used en masse. With such rapid clearance of the drug (3–4 hours), it was manufactured in large quantities and given in high doses very often. Researchers studied ways to slow down the rapid secretion and excretion of penicillin. It was the discovery of uricosurics such as probenecid that really magnified the effects of penicillin. Probenecid is capable of competing with secretion of penicillin in the kidneys and is therefore able to hold penicillin in the body longer, giving it the opportunity to have a greater effect on bacteria.

Discussion

To us, the most important reason for reminding the field about the story of paresis is that paresis is consistent with a categorical, essentialist, "disease" model of psychopathology. Paresis is clearly a disease. Historically, the importance of paresis is that this disorder was a prevalent disease in the 19th century, but the mystery of its etiology was solved and it was largely eradicated during the first half of the 20th century. Psychiatry does not have many success stories, but the story of paresis is one of them.

Paresis appears to fit a categorical model of mental disorders. In most discussions of the categorical model (e.g., Cantor, Smith, French, & Mezzich, 1980), it is associated with a nominalistic measurement system in which stimuli (patients) are sorted into categories (disorders) on the basis of their observable characteristics (symptoms). Most discussions of a categorical model assume that there is a list of necessary and sufficient

characteristics defining whether a stimulus belongs in a category or not. For example, all squares must have four sides, the sides must be straight lines, the lines must intersect at 90-degree angles to each other, and the sides must be the same length. If any of these characteristics are missing, an object is not a square; if all four are present, it must be a square. More recent discussions of the categorical model have (grudgingly) admitted that for most natural categories (e.g., species of animals), the definitions of categories do not require a necessary and sufficient list of characteristics. Instead, categories can be defined by using polythetic definitions in which a certain number of a group of correlated characteristics (syndrome) are sufficient to make an assignment (diagnosis) to the category (disorder).

So how does this view of the categorical model apply to paresis? Initially, this view of the categorical model seemed to work well. Patients either have paresis or they do not. In Haslam's view of essentialism, paresis appears to be a concept that is discrete. Its boundaries are reasonably clear. Moreover, the definition of paresis does seem to be associated with necessary and sufficient characteristics. Paresis is tertiary syphilis of the central nervous system. If the syphilitic bacillus is in central nervous system tissue, then the patient has paresis; if not, then the patient does not have paresis.

However, when we examine the meaning of paresis as a concept in modern DSM-like terms, neither the discreteness nor the necessary/sufficient conditions hold up terribly well. The modern DSMs are built around syndromal representations of categories. The signs (observed behaviors) and symptoms (verbal expression of difficulties) are the characteristics of patients, and these are ideally assessed through a structured interview that has relatively high interclinician reliability. In terms of its signs and symptoms assessed at one time point, paresis was hardly a discrete category. In fact, the syndromes of paresis, as discussed by Kraepelin, were a depressive subtype, a grandiose subtype (which Bayle initially believed was the only syndrome associated with this disorder), and a paranoid subtype. All of these syndrome presentations are present in other forms of mental disorders, including such serious mental illnesses as schizophrenia, bipolar disorders, Alzheimer's disease, and others. From a contemporary descriptive perspective, paresis was hardly a "discrete" category.

What made the category appear somewhat more discrete from a 19th-century clinical perspective was a longitudinal view of this disorder. Patients would be admitted with clinical syndromes such as the ones described above. When followed over time, these patients would start to show the characteristic neurological signs of the disease (the changes in pupil dilation as well as motor paralysis). In a few years, the neurological deterioration into dementia (as that word is used in current clinical work) and the invariable death of the patient set the course of the disorder apart from other disorders. Stated more succinctly, paresis appeared to be a relatively discrete disorder when viewed in terms of its course, but it was not discrete when viewed simply in terms of symptoms.

Even the course (longitudinal pattern) of paresis could be misdiagnosed. That is, patients without paresis could present with a similar course. A good example was a case that Plaut (1911) discussed of a depressed patient who was admitted with motor paralysis. Plaut had performed a Wassermann test on the cerebrospinal fluid of the patient, and the test was negative (indicating, most likely, that the patient did not have syphilis in the central nervous system). The patient died. An autopsy was performed, and the patient was found to have a massive glioma (a type of brain tumor that can grow quite rapidly). If this patient had presented to the same hospital 10 years earlier, most likely no autopsy would have been performed, and certainly no Wassermann test would have been available. From the symptoms and the course, this patient would have been diagnosed (incorrectly) with paresis.

It is also difficult to define paresis in terms of necessary and sufficient characteristics. Clearly, from the current psychiatric approach of measuring psychopathology in which semistructured interviews are emphasized, paresis does not have a set of necessary or sufficient conditions. Motor paralysis (for which the disorder was named) was common, but Kraepelin noted that this symptom was present in only about 60% of patients, and thus not necessary. Laboratory tests for the presence of syphilis in the cerebrospinal

fluid would be much more likely to be diagnostic of paresis. However, there could be patients with positive results who would not have paresis. The process by which syphilis invades human tissue and leads to its destruction appears to be relatively slow (i.e., measured in decades of life). Just as being HIV-positive does not mean that one has AIDS, having syphilitic bacilli in the central nervous system does not mean that a patient has developed dementia or any of the possible forms of psychotic symptoms that characterized the onset of paresis when it was diagnosed in 19th-century psychiatry.

Let us return to Haslam's outline of the meaning of essentialism when applied to categories. Paresis is positive or negative for the following beliefs:

1. *Discreteness*—the category has "clear and sharp boundaries."
   - *Negative*. In terms of the current descriptive measurement system used in DSM-IV, paresis would not present as a clearly separable category.
2. *Uniformity*—members of the category are "very similar to one another."
   - *Negative*. Again, in terms of the symptom presentations currently used to describe severe psychopathology, patients with paresis can appear "manic" (grandiose), depressed, or paranoid.
3. *Informativeness*—knowing that someone is a member of the category "tells us a lot about the person."
   - *Neutral*. Knowing that a person has paresis does not tell us a lot about the individual. Friedrich Nietzsche and John Michler had paresis, but they were hardly similar individuals. Knowing that they had paresis does not tell us much about them or their places in history. However, knowing that they had paresis was *very informative* about their medical condition and their prognosis.
4. *Naturalness*—the category corresponds to something that exists in the real world, "rather than just being an artificial product of people's efforts" to categorize.
   - *Positive*. Paresis, as tertiary syphilis, does represent something in the real world. Almost certainly the disorder existed before Bayle ever named it. And Bayle's naming of the disorder did not change the manifestation of the disorder.

Immutability—the characteristics defining the category cannot be changed.

- *Negative*. Syphilis, sometimes called "the great pox," led to death in a matter of hours to a few days when it first appeared in Europe. Syphilis as a modern disease has changed in such a way that, even when untreated, its progress is relatively slow and it leads to death in decades rather than days.

6. *Historical invariance*—the category has existed over time and across cultural contexts.
   - *Positive*. Paresis was recognized across a range of cultures, countries, and continents. The disorder was extremely rare in priests and Quakers, but was much more frequent in the 19th-century Irish and Italian populations.
7. *Necessary features*—the category can be defined by characteristics that an individual must have in order to belong to the category.
   - *Positive*. To have paresis, the patient must have tertiary syphilis of the central nervous system. If there is no evidence of syphilis in the brain of a patient with the symptoms of paresis, then some other form of dementia must be occurring. Notice, however, that the decision about this characteristic would be *negative* if the focus was entirely on signs and symptoms. There are no self-reported characteristics or any behaviors that are necessary for a patient to have GPI.
8. *Inherence*—there is an "underlying reality" to the category.
   - *Positive*. There was an "underlying reality" to paresis that led to death for individuals with this disorder during the 19th century. The mystery of this disorder was solved by discovering its etiology. In modern times, there are treatments that can be effective for the symptoms associated with the syndromes of depression, mania, and paranoia. None of those treatments would have changed the "underlying reality" of paresis. Even if the depression, mania, or paranoia had been treated, the course of the disease would have led to death.

9. *Exclusivity*—an individual cannot be a member of more than one category in the classification. (This item was dropped in Haslam & Ernst, 2002.)
   - *Clearly negative.* Although retrospective studies cannot be done, almost certainly if a retrospective study of a patient sample from the 1800s could be conducted with the Structured Clinical Interview for DSM-IV and these patients were assigned DSM-IV diagnoses, they would also be diagnosed as having high rates of alcoholism, major depressive disorder, and probably antisocial personality disorder.

Thus, even though paresis is a prototypic mental disorder "disease" because its etiology is clearly known to be a bacterial infection, it is not a good example of an essentialistic category. Nonetheless, paresis is an important mental disorder for the modern conceptualization of psychopathology. This is the first mental disorder in which a descriptive syndrome was empirically documented to be associated with clear changes in the brains of individuals with the syndrome. Gradually, understanding of the concept grew through longitudinal views of the disorders (i.e., the disorder progressed from mental symptoms to occurrences of motor paralysis to increased mental deterioration to death). Finally, when the technology existed to determine the cause of syphilis, the etiological issue associated with paresis was quickly resolved. Still, it took almost another half century to find highly effective treatments (i.e., penicillin, antibacterial drugs) that could affect the etiological basis of the disorder.

However, should the investigation of modern mental disorders follow the example of paresis? We think that this would be a mistake for several reasons. First, the disease model, based on a bacterial view of "disease," is an inappropriate model for many mental disorders. Second, because mental disorders are not essentialistic concepts, the paradigms that have been used so successfully to investigate diseases are likely to have no payoff if used to investigate mental disorder concepts. Third, if the two previous points are true, then the validity of our current mental disorder concepts must be questioned.

Modern conceptions of disorder all too often fall into the trap of essentialism. As we have stated in the introduction to this chapter, there is a strong movement within psychiatry toward biological, disease-model-based explanations for mental disorders. Many authors have argued that the disease model is a misleading representation of the nature of mental disorders, and that it may be inappropriate in and of itself (e.g., Cantor et al., 1980; Zachar, 2000). In short, the essentialistic assumptions outlined above fail when applied to most mental disorder categories. Even if we grant that a disease model is appropriate, the story of paresis emphasizes these authors' points, in that the model fails to capture many aspects of the reality of a prototypic disease like paresis. If the disease model cannot even fully capture a disease such as paresis, then it is extremely unlikely that it will be useful in describing more amorphous concepts that may lack a clear underlying pathology.

Furthermore, the way in which the mental health field studies disorders and treatments embraces several essentialistic assumptions. The current gold-standard paradigm for treatment evaluation is the double-blind, placebo-controlled, randomized clinical trial. This paradigm assumes an essentialistic definition of disorder because, for the effects of the study to be interpretable, the groups must be assumed to be discrete and homogeneous. In fact, many studies employ exacting inclusion criteria: The disorder of interest must be the only condition present, and individuals with other commonly comorbid conditions are excluded. This practice is necessary; otherwise, the results could be realistically attributed to the confounding effects of the presense of another disorder rather than to the pathology of the disorder of interest. The story of paresis reminds us that even a prototypic example of a disease-model-based mental disorder does not adequately meet the assumption of descriptive discreteness. Thus the results of these sorts of studies may be based on an inaccurate model of the disorder and therefore do not represent the actual effects of the nature of the disorder.

Finally, modern understandings of validity encompass aspects of essentialism. As a result, the way in which the field investigates its subject matter may be inherently misdi-

rected. When a new disorder is proposed, its proponents enumerate the ways in which this disorder is different from other disorders in terms of its signs and symptoms, thus justifying its investigation. As we have stated previously, signs and symptoms at the syndromal level are imperfectly correlated with underlying pathology. Many conditions evidence similar symptom patterns, in the same way that paresis resembled such conditions as schizophrenia and bipolar disorders. However, the pathology of the conditions may be very different, therefore implying different approaches to treatment. Within mental health, very few disorders have etiologies that are understood. In the absence of this knowledge, a disease model of classification that is based on essentialistic assumptions is inappropriate. Therefore, some other description of the nature of disorder is necessary if the field is to advance its understanding.

As the next edition of DSM is being written, the story of paresis is a cautionary tale. Although there have not been many success stories within the field of psychiatry, emulating the story of paresis through an implicit, essentialistic view of mental disorders will not necessarily lead to further success. Disorders like schizophrenia, bipolar disorders, or posttraumatic stress disorder are even less likely than paresis to meet the assumptions of essentialism. Although the field has progressed using old models of investigation and description, we propose that the field sorely needs new models of disorder and new ways of investigating mental disorder concepts if future progress is to occur.

References

Anonymous. (1894). Psychological retrospect. *Journal of Mental Science*, *40*, 464.

Austin, T. J. (1976). *A practical account of general paralysis*. New York: Arno Press. (Original work published 1859)

Brown, E. M. (1994). French psychiatry's initial reception of Bayle's discovery of general paralysis of the insane. *Bulletin of the History of Medicine*, *68*, 235–253.

Brown, E. M. (2000). Why Wagner-Jauregg won the Nobel Prize for discovering malaria therapy for general paresis of the insane. *History of Psychiatry*, *11*, 371–382.

Campbell, E. J. M., Scadding, J. G., & Roberts, R. S. (1979). The concept of disease. *British Medical Journal*, *ii*, 757–762.

Cantor, N., Smith, E. E., French, R. &, Mezzich, J. (1980). Psychiatric diagnosis as prototype categorization. *Journal of Abnormal Psychology*, *89*, 181–193.

DeFursac, J. R., & Rosanoff, A. J. (1916). *Manual of psychiatry*. New York: Wiley.

Dowbiggin, I. R. (1991). *Inheriting kadness: Professionalization and psychiatric knowledge in nineteenth century France*. Berkeley: University of California Press.

Engstrom, E. (2003). *Clinical psychiatry in imperial Germany: A history of psychiatric practice*. Ithaca, NY: Cornell University Press.

Engstrom, E., & Weber, M. (2007). Making Kraepelin history: A great instauration? *History of Psychiatry*, *18*, 267–273.

Goldstein, J. (1987). *Console and classify: The French psychiatric profession in the nineteenth century*. New York: Cambridge University Press.

Hammond, W. A. (1973). *A treatise on insanity in its medical relations*. New York: Arno Press. (Original work published 1883)

Haslam, N. (2000). Psychiatric categories as natural kinds: Essentialist thinking about mental disorder. *Social Research*, *67*, 1031–1058.

Haslam, N., & Ernst, D. (2002). Essentialist beliefs about mental disorders. *Journal of Social and Clinical Psychology*, *21*, 628–644.

Haslam, N., Rothschild, L., & Ernst, D. (2000). Essentialist beliefs about social categories. *British Journal of Social Psychology*, *39*, 113–127.

Healy, D., Harris, M., Michael, P., Cattel, D., Savage, M., Chalasni, P., et al. (2005). Service utilization in 1896 and 1996: Morbidity and mortality data from North Wales. *History of Psychiatry*, *16*, 27–42.

Hirschfeld, L. A. (1996). *Race in the making: Cognition, culture, and the child's construction of human kinds*. Cambridge, MA: MIT Press.

Jablensky, A. (2007). Living in a Kraepelinian world: Kraepelin's impact on modern psychiatry. *History of Psychiatry*, *18*, 381–388.

Kendell, R. E. (1975). *The role of diagnosis in psychiatry*. Oxford, UK: Blackwell Scientific.

Kraepelin, E. (1918). *Clinical psychiatry: A textbook for students and physicians* (A. R. Diefendorf, Trans.). New York: Macmillan.

Luhrmann, T. M. (2000). *Of two minds: The growing disorder in American psychiatry*. New York: Knopf.

McGarty, C., Haslam, S. A., Hutchinson, K. J., & Grace, D. M. (1995). Determinants of perceived consistency: The relationship between group entitativity and the meaningfulness of categories. *British Journal of Social Psychology*, *34*, 237–256.

Mercier, C. (1902). *A textbook of insanity*. London: Swan Sonnen-Shein.

Middlemass, J. (1904). A case of developmental general paralysis. *Journal of Mental Science*, *50*, 76–78.

Mott, F. W. (1897). Some cases of syphilitic brain disease resembling general paralysis. *Journal of Mental Science*, *53*, 649–650.

Mott, F. W. (1901). Observations upon the etiology and pathology of general paralysis. *Journal of Mental Science*, *57*, 386–388.

Nitrini, R. (2005). The cure of one of the most frequent types of dementia: A historical parallel. *Alzheimer Disease and Associated Disorders*, *19*, 156–158.

Noguchi, H., & Moore, J. W. (1913). A demonstration of *Treponema pallidum* in the brain in cases of general paralysis. *Journal of Experimental Medicine*, *17*, 427–438.

Plaut, F. (1911). *The Wassermann sero-diagnosis of syphilis in its application to psychiatry*. New York: *Journal of Nervous and Mental Disease* Publishing Company.

Quetel, C. (1990). *The history of syphilis* (J. Braddock & B. Pike, Trans.). Baltimore: John Hopkins University Press.

Reznek, L. (1987). *The nature of disease*. London: Routledge & Kegan Paul.

Robertson, G. M. (1913). General paralysis of the insane. *Journal of Mental Science*, *59*, 185–221.

Robertson, W. F., & McRae, D. (1907). Further bacteriological and experimental investigations of the pathology of general paralysis and tabes dorsalis. *Journal of Mental Science*, *53*, 590–615.

Rothbart, M., & Taylor, M. (1992). Category labels and social reality: Do we view social categories as natural kinds? In G. R. Semin & K. Fiedler (Eds.), *Language and social cognition* (pp. 11–36). London: Sage.

Sherman, I. W. (2006). *The power of plagues*. Washington, DC: American Society for Microbiology Press.

Shorter, E. (1997). *A history of psychiatry: From the era of the asylum to the age of Prozac*. New York: Wiley.

Wiglesworth, J. (1883). Case of general paralysis in a young woman, commencing at the age of fifteen. *Journal of Mental Science*, *29*, 241–244.

Yzerbyt, V., Rogier, A., & Fiske, S. T. (1998). Group entitativity and social attribution: On translating situation constraints into stereotypes. *Personality and Social Psychology Bulletin*, *24*, 1089–1103.

Zachar, P. (2000). Psychiatric disorders are not natural kinds. *Philosophy, Psychiatry, and Psychology*, *7*, 167–182.

CHAPTER 17

Concepts and Methods for Researching Categories and Dimensions in Psychiatric Diagnosis

HELENA CHMURA KRAEMER

Once again a seismic occurrence in mental health is in the making: The fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-V) is currently due to come out in 2012. Once again mental health clinicians will be expected to relearn psychiatric diagnosis definitions, and researchers to switch from use of one diagnostic system to another in their studies. In what ways the new system will change, and whether these changes will be for the better, remain to be seen. However, on one issue there currently does seem to be agreement: Dimensional diagnoses, as well as the traditional categorical diagnoses, will be included. Exactly how that will happen, and whether adding dimensional diagnoses will eventually improve mental health care (as predicted), also remain to be seen.

Traditionally, DSM diagnoses have been "categorical"; in other words, a patient either is or is not assigned a particular diagnosis. However, a diagnosis may also be "dimensional"—that is, made on an ordinal scale with more than two values. Such a diagnosis could minimally be made on a 3-point scale ("definitely yes," "possible," "definitely no"); at the other extreme, it could be expressed on a continuum (much as systolic and diastolic blood pressure are measured). Both "categorical" and "dimensional" can be somewhat misleading terms, since "categorical" technically can refer to having *more than* two nonordered responses, and "dimensional" can also refer to having two *or more* binary or ordered responses, to characterize a patient. However, these terms have been consistently used in this context as corresponding to univariate binary and ordinal responses, respectively. For the sake of discussion, I adhere to that usage in this chapter.

Why dimensional diagnoses have long had strong advocacy is clear. From researchers' perspectives, access to valid, reliable dimensional diagnoses—sensitive to the heterogeneity of response among those with a disorder, as well as to the heterogeneity of response among those who might later develop the disorder—increases the power to detect risk factors, to identify causal risk factors, and thus to develop and document efficacious and effective treatments for the disorder. With access only to categorical diagnosis, not only is the power to detect "signals" of any kind diminished, requiring large (and typically unavailable) sample sizes; even when signals are detected as "sta-

tistically significant," their effect sizes are attenuated. Thus progress in mental health clinical research may very well have been slowed by exclusive reliance on categorical diagnoses.

Moving from a categorical to a dimensional diagnosis elucidates clinically significant heterogeneity among those with the categorical diagnosis and of clinically significant heterogeneity among those without the categorical diagnosis. This amplifies the "signals" and should facilitate better "tuning" and "clarification" of the "detector" of the signals. The present discussion focuses on the conceptual and methodological issues involved in developing categorical and dimensional diagnoses of mental disorders.

Disorder versus Diagnosis

Crucial to any discussion of diagnosis is the distinction between a "diagnosis" and a "disorder." A "disorder" is something wrong in a patient that is of clinical interest and concern (a disease, a malfunction, an injury, a disability, etc.). The patient experiences a disorder, whether or not any clinician recognizes it. The disorder is the "signal" to be detected. A "diagnosis" of the disorder, on the other hand, is the informed opinion of a clinician that a certain disorder is present in the patient. A patient has a diagnosis only when a clinician delivers it, and that diagnosis may be right or wrong as to the presence of the disorder—a fact many patients discover when they seek independent second opinions.

An illustration will show how crucial the distinction between diagnosis and disorder is. To reduce the prevalence or incidence of a *diagnosis* is easy: Simply make the criteria for diagnosis more stringent. To reduce the prevalence or incidence of a *disorder*, on the other hand, requires new knowledge of the causal factors leading to the disorder and the development of new strategies either to prevent or to successfully treat the disorder. That is not so easy.

A specific disorder is either present or absent in each person. Among those in whom that disorder is present, there is typically heterogeneity in terms of duration, severity, which specific signs and symptoms are expressed, how these are expressed and for how long, and any resulting impairment. If one could conceive of some measure of "salience" of a disorder (D) that reflects all such clinical heterogeneity in expression, one might imagine tracking this salience for each individual patient over time. Within an individual patient, over stretches of time, the disorder may be absent ($D = 0$). However, the disorder may be present ($D > 0$), but to a degree and duration that might not stimulate concern either in the individual (who would not seek clinical attention for it) or in the clinician (D is not "clinically significant").

The earliest time at which the disorder becomes of clinical significance is the "onset" of the disorder—the beginning of the first "episode" or "bout." The span of time after onset over which the disorder remains of clinical significance is the "episode" or "bout" of the disorder. At the end of an episode, when the salience falls once again below clinical significance, there is "remission." If at some later time, the salience once again rises into the region of clinical significance, this will signal a "relapse" and the beginning of a new episode or bout. If a remission lasts long enough that a relapse of the same disorder becomes unlikely, the patient may be said to have "recovered." Another episode after recovery, thought to be a separate, new occurrence of the disorder, would be a "recurrence." All these terms (Frank et al., 1991; Rush et al., 2006)—"onset," "episode/bout," "remission," "relapse," "recovery," "recurrence"—are in common use among medical consumers (patients), clinicians, clinical researchers, and health care policymakers, all referring to the course of the *disorder* over time in a subject.

Moreover, these terms are important in specifying the topics around which clinical research centers. Identifying risk factors for a disorder focuses on the characteristics of individuals as yet free of the disorder that predict subsequent onset. Prevention focuses on manipulation of causal risk factors for an individual prior to onset that would delay onset (perhaps even forever). Early diagnosis issues focus on identification of individuals in the early stages of a disorder, when the salience is clinically significant but low, because experience indicates that early treatment is often most likely to be effective treatment. Treatment studies focus on individuals in the midst of an episode, to attempt to shorten the duration of the episode and induce early remission, as well as

to dampen the salience during the episode. Maintenance studies focus on individuals who are in remission, to attempt to lengthen the duration of the remission and thus to prevent relapse, or even to induce recovery. Once there is recovery, how to prevent recurrence may become an issue, and successful such efforts may or may not be the same as successful efforts to prevent initial onset.

The problem, of course, is that in the vast majority of clinical situations (particularly in the context of mental health), there is no direct way to ascertain whether a disorder is present or not, much less what its salience is. "Disorder" and "salience of disorder" are what are called "latent constructs." That is, they exist, but they cannot be directly observed or measured. They can only be inferred by what can be observed or measured: a diagnosis that is valid for the disorder in question for a specified population (the observable expression of a disorder may not be the same over time, or in different populations) and variation in severity, impairment, duration, expression, and so forth (the elements of salience).

Minimally required for validity of a diagnosis is that the greater the salience of the disorder, the more likely a positive diagnosis of that disorder is. But how, in the absence of any "gold-standard" diagnostic procedure to detect the presence of the disorder, or to measure its salience, do we assess and compare different proposals for diagnosis? And how should we go about improving diagnosis?

Categorical and Dimensional Diagnoses

Categorical Diagnosis

For a categorical diagnosis (*CDX*), validity has long been described in terms of "sensitivity" and "specificity" (Bossuyt et al., 2003; Galen & Gambino, 1975; Kraemer, 1992a). Table 17.1 is a 2 × 2 table relating a particular categorical diagnosis (*CDX* = 1 when "yes," 0 when "no") to the presence or absence of the disorder (*D* = 1 or 0). The sensitivity (*Se*) of the diagnosis is the probability of a positive diagnosis (*CDX* = 1) when the disorder is present (*D* > 0). The specificity (*Sp*) of the diagnosis is the probability of a negative diagnosis (*CDX* = 0) when the disorder is absent (*D* = 0). To be valid, the probability of a positive diagnosis when the disorder is present must be greater than when it is absent ($Se > 1 - Sp$). Ideally, $Se = Sp = 1$; that is, the diagnosis is an error-free indicator of the disorder.

Figure 17.1 shows the same information graphically in a receiver operating characteristic (ROC) plane—an essential tool of signal detection methods (Kraemer, 1992a)—with four different *CDX*s, numbered 1–4. Each *CDX* that could be proposed for a certain disorder in a certain population can be located as a single point in the ROC plane by its sensitivity (*Se* on the *y*-axis) and the complement of its specificity ($1 - Sp$ on the *x*-axis).

As noted above, the ideal diagnostic rule is one with both sensitivity and specificity equal to 1, at the "ideal point." This never happens because of the less than perfect reliability of diagnoses. The poorest possible diagnosis occurs when the probability of a positive diagnosis does not depend on whether or not the disorder is present ($Se = 1 - Sp$), and thus lies on the diagonal line called the "random ROC" (Figure 17.1). Every valid *CDX* lies above the random ROC, but any *CDX* that lies below the random ROC can be made valid, simply by switching the "yes" and "no" labels.

If the probability of a positive diagnosis in the population of interest (*Q* in Table 17.1)

TABLE 17.1. Descriptors of Validity for a Categorical (Binary) Diagnosis (*CDX* = 1 vs. *CDX* = 0) for the Disorder (*D* > 0 vs. *D* = 0)

	CDX = 1	*CDX* = 0	
D > 0	*A* (true positive)	*B* (false negative)	$P = A + B$
D = 0	*C* (false positive)	*D* (true negative)	$P' = 1 - P = C + D$
	$Q = A + C$	$Q' = 1 - Q = B + D$	

Note. *A*, *B*, *C*, *D*, *P*, *P′*, *Q*, *Q′* are the probabilities of the indicated events in the population of interest. Sensitivity (*Se*) = *A*/*P*. Specificity (*Sp*) = *D*/*P′*.

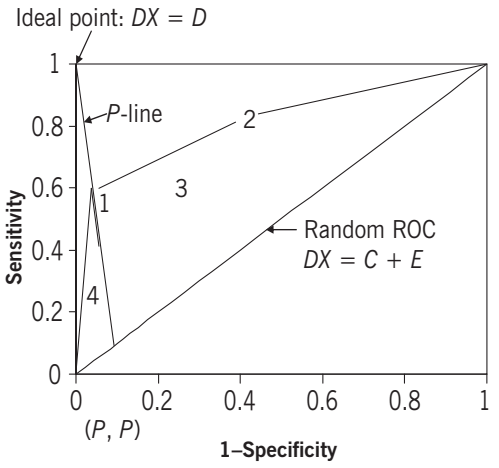


FIGURE 17.1. The receiver operating characteristic (ROC) plane showing the "location" of a single categorical diagnosis for a disorder with probability *P* in the population of interest.

equals the probability of having the disorder (*P* in Table 17.1), that point will lie on the line connecting the ideal point with the point *P*,*P* on the random ROC. Any *CDX* having a value of *Q* unequal to *P* lies on a line parallel to the *P*-line, cutting the random ROC at the point *Q*,*Q* (see Figure 17.1). This is a geometric version of Bayes's theorem. Thus in Figure 17.1, it can be seen that *P* is about .1; that in order of *Q* (lowest to highest) are *CDX*s 4, 1, 3, and 2; and that *Q* for *CDX* 1 is approximately equal to *P*.

Thus highly sensitive *CDX*s (the kind one would prefer for screening tests) lie above the *P*-line above the random ROC (like *CDX* 2), and highly specific *CDX*s (the kind one would prefer for a definitive diagnosis) lie below the *P*-line above the random ROC (like *CDX* 4). A discriminative *CDX* would generally lie near the *P*-line and above the random ROC (like *CDX* 1). In all cases, the further a *CDX* is from the random ROC, and the nearer to the ideal point, the more valid the *CDX*.

When there are various *CDX*s under consideration in a particular population for a single disorder (*P* fixed), as there are in Figure 17.1, such a graphic view permits easy comparisons among them. If one draws straight lines joining each pair of *CDX* points, the upper boundary of that set of lines connected to the two corners of the ROC plane is the ROC curve related to that set of *CDX*s—the location of all possible optimal tests for that disorder in that population. For any *CDX* that lies below the ROC curve (like *CDX* 3), there is another available *CDX* (or a combination of two *CDX*s) on the ROC curve that has better sensitivity *and* specificity (here *CDX* 1) and is therefore more valid.

There are well-developed geometric (McNeil, Keeler, & Adelstein, 1975) and analytic (Kraemer, 1992a; Kraemer et al., 1999; Kraemer, Periyakoil, & Noda, 2002) methods to locate the optimal *CDX* from the subset of *CDX*s lying on the ROC curve for a specific clinical purpose. It is seldom true that one *CDX* can optimally serve all clinical purposes in a population, although traditionally we have acted as if it could.

Moreover, if the same *CDX* is used in a different population, one in which *P* is different, the entire ROC picture may change. Not only does the *P*-line shift, but the points locating the different *CDX*s also shift. Thus a *CDX* that is valid in one population may not be in another. It is not necessarily true that the best *CDX* for assessing any disorder is the same for males and females; for preteens, adolescents, adults, and seniors; or for different ethnic groups—although traditionally we have acted as if one *CDX* is optimal, regardless of the population of concern.

In particular, although the declared intent of DSM is for clinical uses, decisions concerning the definition of *CDX*s have often been based on community samples. Whether a *CDX* developed in a community sample is optimally sensitive and specific for a clinical sample (or vice versa) is highly questionable.

Dimensional Diagnosis

Suppose instead that a dimensional diagnosis (*DDX*) is proposed—an ordinal scale of some type. For *DDX* to be valid, those with the disorder, and especially those with the disorder of higher salience, must be more likely to have higher values of *DDX*. To show this, we dichotomize *DDX* at every possible cutoff point ($DDX \geq d$ versus $DDX < d$), thus producing a *CDX* at every possible cutoff point; obtain the sensitivity and specificity for each dichotomization; locate these points in an ROC plane; and draw the ROC curve (Figure 17.2). If the ROC curve so generated from a *DDX* coincides with the random ROC, the *DDX* has no valid-

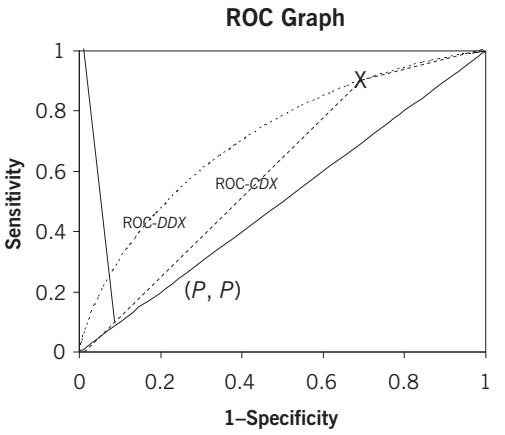


FIGURE 17.2. The ROC curve for a dimensional diagnosis (*DDX*) with an absolutely continuous distribution in a population with probability *P* or 10%, and the ROC curve for a categorical diagnosis (*CDX*) obtained by dichotomizing that *DDX*.

ity. The higher the arch of the ROC curve above the random ROC, the more valid the *DDX*. One useful measure of the validity of a *DDX* is the area under that ROC curve, or *AUC* (Acion, Peterson, Temple, & Arndt, 2006; Brownie, 1988; Grissom, 1994; Hanley & McNeil, 1982; Kraemer & Kupfer, 2006; McGraw & Wong, 1992). The *AUC* estimates the probability that someone with the disorder has a higher *DDX* than someone without the disorder (Acion et al., 2006; Kraemer & Kupfer, 2006). Thus, for an invalid *DDX*, *AUC* = .5. The closer *DDX* is to 1.0, the more valid the *DDX*. (The *AUC* for a single *CDX* is $.5(Se + Sp)$.)

Since each point on the ROC curve of a *DDX* is itself a *CDX*, the same *DDX* can be used for different clinical purposes, simply by varying the cutoff point. Thus instead of trying to develop a different optimal *CDX* for each possible clinical use, one may be able to use a single *DDX* and vary the cutoff point for different clinical uses—a distinct practical advantage. It may even be possible to develop an optimal *CDX* for each possible clinical use in both a clinical and a community population, simply by varying the cutoff point.

However, if one chooses to use *one* of these dichotomizations of a *DDX* rather than *DDX* itself in a research study, there is usually a loss of information, which translates into loss of power for statistical tests and loss of accuracy in estimating population parameters. How much loss there is depends on exactly where the cutoff point is set. In Figure 17.2, for example, the ROC curve based on a continuous *DDX* is compared with the ROC curve based on a *CDX* based on a single dichotomization of that *DDX*. A cutoff point located at or near the peak of the ROC curve entails less loss in *AUC*, while one located in a corner of the ROC plane (as in Figure 17.2) may completely undermine its validity. This is the source of concern about the selection of cutoff points for earlier versions of DSM, which appear to have been arbitrarily set.

Lest there be an impression that the loss incurred would be trivial, consider the following illustration. Let us suppose that in a randomized clinical trial with equal sample sizes in the treatment (*T*) and control (*C*) groups, what is required is a sample size per group adequate to have 80% power to detect a moderate difference (standardized mean difference of .5, *AUC* = .64) between *T* and *C*, using a 5% two-tailed significance test. If the *DDX* at the end of treatment is used as the outcome measure, and satisfies the usual *t*-test assumptions, the necessary sample size will be 63 subjects per group ($N = 126$). However, if instead a cutoff point is applied to the *DDX* to generate a *CDX* to be used as the outcome measure, the minimal sample size needed per group will be 101 ($N = 202$), and that will be only if the cutoff point is exactly halfway between the two means. If the cutoff point is a standard deviation below or above that midpoint, the sample size will be 183 ($N = 366$); if it is two standard deviations away, the sample size will be 770 ($N = 1{,}540$); and so on. It is not unusual that in moving from using a *DDX* to using a *CDX*, the minimal sample size increase is about 50% to get the same power to detect the same effect, and the sample size may be 5- or 10-fold larger for less optimally selected cutoff points. This only emphasizes what researchers have long known (Cohen, 1983; Kraemer, 1991; Kraemer & Thiemann, 1987; MacCallum, Zhang, Preacher, & Rucker, 2002; Veiel, 1988): With CDX, signals can be harder to detect, the cost of doing studies greater, and results that are not statistically significant more common.

To make matters worse, suppose one here uses as an effect size the number needed to treat (*NNT*) (Altman, 1998; Cook & Sack-

ett, 1995). *NNT* is the number of subjects one will need to give *T* in order to get one more success than if they have all been given *C* ($NNT = 1/(2AUC - 1)$). Thus $NNT = 1$ indicates that every *T* subject has *DX* greater than every *C* subject (no overlap between the two groups). The larger the *NNT*, the less the advantage of *T* over *C*. Using the *DDX*, $NNT = 3.6$. With optimal dichotomization for a *CDX*, $NNT = 5.1$, and as the cutoff point moves to one and two standard deviations away from the midpoint, *NNT* rises to 10.9 and 60.5. Thus even if statistically significant results are found with such a *CDX* (which can be assured by having larger sample sizes), the resulting effect sizes may appear to be so low as to be viewed as not clinically significant.

Conversions between Categorical and Dimensional Diagnoses

Any *DDX* can obviously be dichotomized in a variety of ways to generate *CDX*s. What is less obvious is that every *CDX* can also be converted into a corresponding *DDX* in a variety of ways. In fact, many of the current *CDX*s are already based on dichotomizing ordinal measures. A *CDX* is often defined as having at least *n* of a long list of signs/symptoms presented. The number of symptoms endorsed in a patient is a *DDX*. The cutoff point *n* is often arbitrarily, rather than optimally, set. Other current *CDX*s rate severity as "mild," "moderate," or "severe"—a 3-point *DDX*. Yet others impose cutoff points on age or duration. In all such cases, simply moving back to the ordinal scale dichotomized to generate the *CDX* converts the diagnostic rule to a corresponding *DDX*.

Even if the *CDX* is completely qualitative, one can always have the *CDX* obtained by $m > 1$ independent diagnosticians. The number of positive diagnoses (0, 1, 2, . . . *m*) is a simple *DDX* that will be more reliable and valid than any one of those individual *CDX*s (Kraemer, 1992b). Just as conversion from a *DDX* to a corresponding *CDX* loses power and precision, a conversion from a *CDX* to a corresponding *DDX* will generally increase power, precision, and sensitivity to patient heterogeneity.

It is, of course, not true that every *DDX* for a disorder is better than every *CDX* for the same disorder. But it is true that for every *CDX* there exists a *DDX* based on the same patient information that is better in terms of power, precision, and sensitivity. Why, then, do we not simply do away with *CDX*s and focus only on *DDX*s?

The Argument for Coexistence

The original motivation for the DSM system, and the reason for "statistical" as the central word in its title, appears to relate to the original emphasis on its use in counting tasks (such as estimating incidence and prevalence, in order to consider trends over times/places and in subpopulations defined by gender, age, ethnicity, etc.) and in administrative tasks (such as estimating resources needed to deal with patients). Moreover, for clinicians to communicate effectively with patients, such brief labels are necessary. These motivations continue to apply, even now that uses of the DSM system have spread to, for example, randomized clinical trials, biochemical research, genetic studies, and imaging research. Indeed, brief labels may be even more necessary today, when patients need specific keywords in order to get information about their disorder from medical websites.

It would be a mistake, however, to think that clinicians, patients, and administrators always prefer categorical diagnoses, while researchers always prefer dimensional diagnoses. The all-important decisions as to inclusion–exclusion from a randomized clinical trial, or decisions on matching and stratification in research designs, require *CDX*s. Studies of such issues as onset, remission, relapse, recovery, and recurrence require definitions that can only be based on a *CDX*. Thus researchers need *CDX*s as well as *DDX*s. Clinicians monitoring response to treatment need *DDX*s to detect whether the patient is improving or not. Clinicians need *DDX*s as well as *CDX*s.

In summary, both categorical and dimensional diagnoses are necessary to cover all the clinical and research purposes for which diagnoses might be pertinent. Which is preferable depends on the situation. What is necessary, however, is that there be a close connection between the *CDX* and the *DDX* for the same disorder, and that each be used in the situation in which that form of diagnosis is best.

The Process of Signal Detection Development of a Diagnosis

A Working Definition of Mental Disorder

What is needed first is an agreed-upon definition of a "mental disorder" to identify what types of signals are being sought. It would be presumptuous for a biostatistician to declare such a definition, but just for illustration, let us say that a "medical disorder" is a condition existing in an individual that (1) causes distress and/or impairment within that individual; (2) cannot be controlled by the will of the individual; and (3) in the absence of intervention, is likely to persist and develop. Thus being Jewish in Germany during the 1930s was not a disorder despite satisfying criteria 2 and 3 because the distress and impairment experienced were imposed from the outside, not generated by the condition within any Jewish individual. A condition that society finds unusual or does not approve, and to which it reacts by imposing distress or impairment on the individual, is not a medical disorder. Chronically getting beaten by a domestic partner (once briefly proposed as a mental disorder) is not a medical disorder because one can choose to remove oneself (even if this is not easy) from the situation. The common cold is an illness, but by the definition above it is not a disorder, since generally it will resolve within a week or so with or without intervention. Thus the models for medical disorders here are heart disease, cancer, diabetes, and other serious chronic disorders, rather than the common cold or other conditions that usually resolve in the absence of medical intervention.

More specifically, a "mental disorder" might be defined as a medical disorder characterized primarily by expression in emotions, behavior, and/or cognition. Thus diabetes mellitus is a disorder, but not a mental disorder. Alzheimer's disease is a disorder that is a mental disorder, as well as a neurological disorder. Depression, schizophrenia, and autism are mental disorders.

The definitions articulated here may be completely unacceptable to experts in the medical and mental health fields, but they illustrate an important point: Any articulated definition sets the minimal criteria for evaluating the validity of proposed diagnoses. With this definition, one would have to demonstrate that any proposed diagnosis is substantially (not necessarily totally) influenced by emotions, behavior, and/or cognition; that it is associated with distress and/or impairment stemming from within the individual (not imposed by the environment); that these expressions are not within the willful control of the individual; and that there is some consistency to the diagnosis over time in absence of intervention. None of these criteria require a gold-standard diagnostic procedure for evaluation of validity.

Compiling a Diagnosis from Multiple Diagnostic Indicators

Let us start, as each iteration of DSM has started, by identifying diagnostic indicators (currently symptoms, signs, and responses, but in the future perhaps also including results of genotyping, imaging, and biochemical tests) that tend to cluster within patients, each of which is a weak *DX* of a disorder of interest. Early in this process, such diagnostic indicators are based completely on clinical observations and intuitions, but later, one would expect new indicators arising from basic and clinical research findings based on earlier versions of the diagnosis of the disorder.

Then the location of the ROC curve for each diagnostic indicator (and later the diagnosis itself), *DX*, meant to detect the disorder, *D*, is determined by three components, symbolically:

$$DX = D + C + E$$

where *DX* is the diagnostic indicator or diagnosis (categorical or dimensional), *D* is the unobservable presence/absence of the disorder of interest, *C* represents information about that individual completely unrelated to the disorder (contaminants[1]), and *E* represents random error of measurement. The position of the ROC curve for any *DX* depends on how much of the information in *DX* depends on *D*, and how little on *C* or *E*.

Let us now review some classic definitions. "Reliability" is the proportion of the total variance among individuals in the population of interest (due to *D*, *C*, and *E*) that is due to information about the individual (*D* and *C*). Reliability can generally be esti-

mated by (1) taking a representative sample from the population of interest; (2) having two observations of the indicator per subject taken by different raters, each working independently of the other, over a span of time in which the disorder is likely to remain stable, but the errors are likely to be independent of each other; and (3) using a parametric or nonparametric intraclass correlation coefficient (for dimensional measures), or an intraclass kappa (for categorical measures) to estimate the reliability. Even in the absence of a gold standard, reliability can be estimated and has been the field trial mainstay of DSM development since about the time of DSM-III. When test–retest reliability is near zero, the ROC will coincide fairly well with the random ROC ($DX = E$). However, the ROC may coincide fairly well with the random ROC even when test–retest reliability is very high ($DX = C$). Thus low test–retest reliability guarantees poor detection, but high test–retest reliability does not guarantee good detection.

"Validity" of the diagnosis for the disorder is the proportion of the total variance (D, C, and E), due to the disorder (D alone). The validity of the diagnosis for the disorder theoretically is the correlation between DX and D in the population, but practically it cannot be directly estimated because we cannot directly observe D. In such cases, what is done instead is to challenge the validity of DX in a variety of ways. The more such challenges it survives, the more likely DX is to be a valid diagnosis for D. The position of the ROC curve is determined by the validity of DX for D.

The reliability of a diagnosis is always at least as great as its validity for any disorder, and equals it only in the absence of any contaminants. Thus the emphasis in DSM development has long been on establishing the reliability of diagnoses, even though reliability is not a definitive indicator of the quality of the DX. However, reliability is easy to document, and in the absence of reliability, validity is not possible. That certainly makes sense, but it must be remembered that efforts to improve the reliability of a diagnosis may occur at the cost of its validity.

When most of the variance of an indicator in the population is due to contaminants or to error ($DX = C + E$), its ROC curve will coincide with the random ROC. When there are neither contaminants or errors, the ideal point in the ROC plane will be on the ROC curve ($DX = D$). In practice, neither extreme is likely to pertain. The goal then is to combine multiple diagnostic indicators to improve diagnosis, and to do so in such a way as to move the resulting DX away from the random ROC and toward the ideal point. To do this, there are only three options: increase the variance due to D, decrease the variance due to C, or decrease the variance due to E.

The only available strategies to increase variance due to D are (1) to add indicators based on new information related to D; and (2) to move, as much as possible, from such categorical indicators to dimensional indicators, in order to pick up as much of the heterogeneity of the disorder as is possible. Thus beyond (1) incorporating the scientific knowledge gained from replicated studies using the earlier version of the diagnosis, and (2) moving to dimensional diagnoses, most strategies are directed to reducing E and C.

The Problem of Redundant Measures

If one simply independently measures the *same* indicator multiple times (perhaps in multiple different ways), and uses those multiple measures separately, these will generate multiple ROC curves differing only because of error of measurement. This will not raise the ROC curve. On the other hand, if one averages those multiple indicators to reduce them to *one* composite indicator, the errors (E) of measurement in the multiple indicators will cancel each other out. Then the error (E) of the average of the indicators will be smaller that that of any single measurement of the indicator, which will raise the ROC curve.

Averaging multiple independent measures of the same indicator always increases the reliability, according to the Spearman–Brown formula (Brown, 1910; Spearman, 1910), approximately:

$$r_m = mr_1/[(m - 1)r_1 + 1]$$

where r_m is the reliability of the average of m independent measures of the same indicator. As long as a single indicator has nonzero reliability ($r_1 > 0$), one can theoretically increase the reliability of an average to almost perfect, simply by averaging enough independent measures of that indicator.

However, doing so may not raise the ROC curve much at all, for the upper limit of ROC curves so achieved will depend on the relative size of the variance due to the disorder (*D*) versus that due to contaminants (*C*). If the variance due to *C* is much larger than that due to *D*, not much increase in validity can be achieved by averaging multiple measures of the same indicator.

The situation can be summarized as follows:

- It is *never* worthwhile using multiple measures of the same indicator *separately* in a diagnostic procedure. That only adds more error and no new information.
- If the reliability of an indicator is near zero, it may as well be set aside; multiple poor-quality measures do not necessarily add up to a good-quality measure.
- If the reliability of an indicator is nonzero but low because of error of measurement, *and* there is reason to believe that most of the remaining variance is due to *D* and not to *C*, it may be worthwhile to obtain multiple independent measures of the indicator and to average them.
- If the reliability of a single measure of an indicator is moderate to high, there is little practical advantage to obtaining multiple such measures. The focus may then shift to considerations of the validity of that indicator.

But how can one tell that there are multiple measures of the *same* indicator, since redundant measures may not be in response to exactly the same question? If the square of the correlation coefficient between two measures approximates the product of the reliability coefficients of the two separate measures, either the reliability of one and/or the other is zero, or the two measures are redundant to each other. Thus the initial removal of indicators with near-zero reliability, and then the detection of redundancy using this rule, are essential. Where there are redundant reliable indicators, one should somehow reduce these to one.

Combining Multiple Nonredundant Indicators

Since most mental disorders have multiple expressions (e.g., multiple signs and symptoms), and individual subjects with the same disorder may not have the same expressions, it is essential to seek multiple, reliable, nonredundant indicators. The next logical step would be to cluster or combine such multiple indicators to form a diagnosis.

Several analytic methods are available for this task (cluster analysis, factor analysis, latent-class analysis, item response theory, etc.). What they all have in common are that (1) there is no external criterion, and (2) they examine the correlational structure of the indicators offered and cluster those that in some sense are most closely related to each other. If redundant reliable measures are included, even if they were completely invalid for the disorder, they may well dictate the results of such analyses, resulting in an invalid diagnosis. However, the same problem arises with multiple reliable indicators that are not redundant, but have strong shared contaminants.

For example, suppose that in a mixed-gender population, one selects a number of indicators of a non-gender-specific disorder that, regardless of the presence or absence of the disorder, differentiate males from females. In this case, gender is a contaminant in trying to develop a valid *DX* for *D*. Any clustering method may well result in a *DX* that simply differentiated males and females in that population, but may be totally invalid for the disorder itself. The same will be true for indicators very strongly associated with age, ethnicity, or another distinct disorder highly epidemiologically comorbid with the disorder of interest.

It is important to realize that a different distribution of *DX* in different strata—for example, different prevalences of a *CDX* in different strata—is not evidence per se of contamination by whatever determines the strata. It may well be that the disorder is more prevalent among females than among males, and that the *CDX* correctly reflects this difference. Alternatively it may be that the *CDX* is biased because gender is a strong contaminant—that the prevalence based on the *CDX* differentiates males and females, but the disorder itself does not.

Like the disorder *D* itself, the contaminant *C* is latent (i.e., not directly observable). There does not seem to be any simple rule, like the rule for identification of redundant indicators, to help identify indicators with strongly shared contaminants. However, if one suspects a specific contaminant, such as

gender, one can stratify the population on that contaminant and assess reliability and validity of the diagnosis within each such stratum. If the major source of variability in an indicator is gender, such indicators will now have very low reliability and/or little evidence supporting validity within each gender stratum. One may, however, find that indicators that are reliable and have some evidence of validity differ from one stratum to another, which will suggest that *DX* may have to be different for males and females. Finally, one may find that the *DX* appears comparably valid in both groups, suggesting that while the disorder itself is gender-specific, gender is not a strong contaminant in the diagnosis, and the same *DX* may be valid for both genders.

For example, it is known that below the age of about 50, the prevalence of heart disease is much less among females than among males. However, if one changed the diagnosis so as to match the prevalence of men to that of women, one might find many more men dying because they were inappropriately denied treatment, or many more women subjected to unnecessary treatment—neither of which is an acceptable option.

Challenging the Validity of a Diagnosis

Once indicators based on clinical and research experience are proposed and measured on ordinal scales as much as possible; once those indicators are checked for reliability and discarded if not reliable; once redundant indicators are identified and redundancy is removed, by selection of either one indicator, some combination of several, or all; once serious consideration is given to possible contamination of the indicators; and once some method is used to identify the latent construct (by this stage presumably closely related to the disorder *D*) underlying the remaining indicators, the final question has to do with establishing validity of the resulting *DX* for *D*.

Since a mental disorder is defined as being chronic in the absence of effective treatment, how stable is *DX* over months or years? Since each individual mental disorder is likely to be relatively rare in a clinical population and even rarer in a community population, those without the disorder will predominate in any sample. If one simply correlates *DX* at one time with *DX* a year later, the result is likely to appear stable, only because the majority do not have a high *DX* at any time. However, if one accepts the definition of a mental disorder as chronic in the absence of effective treatment, one can expect that those with high values of a valid *DX* at the earlier time will continue to have high values of *DX* at the later time; some with low values of *DX* at the earlier time will experience onset and will have high values of *DX* at the later time; and the majority will have low values of *DX* at both times. Very few with high values of *DX* at the earlier time will have low values of *DX* at the later time. Either a total lack of stability over time, or stability only within those likely not to have the disorder, will be evidence against validity of *DX* for the disorder.

Similarly, correlation of *DX* with measures of concomitant or future distress or impairment, by the definition above, should be relatively high. Absence or low levels of correlation will be evidence of lack of validity for a disorder.

In short, the preliminary challenges to the validity of a *DX* are based on whatever definition of mental disorder is set a priori.

Challenge: One Disorder or Two?

The final challenge is to establish diagnoses related to *different* mental disorders. There are undoubtedly many. Some diagnostic indicators may be associated with several different disorders. Certain risk factors, such as gender, age, or poverty, may be risk factors for multiple such disorders. Different such disorders may be comorbid (i.e., occurring in the same person). Different disorders may result in very similar levels of distress and disability. How, then, do we distinguish one disorder from two, or recognize that what was envisioned as two disorders is merely one?

It is said that at one time, what we now call syphilis was thought to be multiple different disorders, depending on which organ system was primarily affected. (See Blashfield & Keeley, Chapter 16, this volume, for the history of one of these disorders: paresis.) However, when an organism was found to cause one of these disorders, it was

found to be the causal mechanism for all; when an effective treatment was discovered for one of these disorders, it was found to be effective for all. Consequently, multiple disorders became one. Conversely, diabetes was originally considered one disorder but is now recognized as two different ones, Type I and Type II—initially because the age-of-onset distribution seemed bimodal, but later because the causal mechanisms and effective treatments were shown to differ as well.

To adapt this model to the present situation, if those identified by diagnostic procedures as having two disorders share multiple risk factors (some causal), share diagnostic indicators, and are epidemiologically comorbid (those with one are more likely to be diagnosed with the other) (Kraemer, 1995), and if the same interventions appear effective for both, all these things raise the probability that the two diagnoses correspond to one disorder. Suitably combining the two diagnoses will raise the ROC curve for that disorder. For instance, are depression and anxiety two different mental disorders, or are the diagnostic procedures for both identifying the same underlying disorder? If the latter is the case, clinical research should be strengthened by recognition of the fact and suitable adjustment of diagnostic procedures.

On the other hand, mixing two different disorders as one will result in major difficulty in identifying any strong risk factors associated with a particular diagnosis, or in developing more than marginally effective treatments. Thus if there is little research progress over time in understanding either the cause, course, or cure for a disorder, this raises the suspicion that two (or more) different disorders are concealed under the same diagnostic label. For example, is schizophrenia two or more different disorders, as separate from each other as Type I and Type II diabetes?

Since ultimately the utility of a diagnosis depends on whether it helps either in prevention or treatment of a disorder, either subsuming two disorders under one diagnostic label, or diffusing one disorder under two diagnostic labels, will compromise the utility of a diagnosis in clinical decision making as well as clinical research. Thus it is important not to "split" when one should "lump" or to "lump" when one should "split." Inappropriately taking either action carries serious clinical and research consequences.

Discussion

The evolution of a successful diagnostic system is not a straightforward process, but one of successive approximations. Initially, a diagnostic system may be based entirely on clinical observation and intuition, as was probably true of DSM-I. Ideally, when such a diagnostic system is then used in subsequent clinical practice and in clinical research, new information is generated from the research that, when replicated and confirmed, stimulates modifications resulting in the next iteration of the diagnostic system. Then the process repeats itself over and over again. Ideally, then, each iteration (e.g., DSM-I, its successors, and soon DSM-V) should produce more valid diagnoses than the iteration before, approaching more and more closely to the disorders themselves. At the same time, the clinical research that follows each iteration, since it is based on a more valid diagnostic system than available before that iteration, should be able to identify stronger risk factors (perhaps causal risk factors), and if so to suggest more effective prevention programs. Among those with each disorder, it should be possible to identify the disorder earlier with an improved diagnostic system, and easier to find and document effective treatments. With better diagnosis, it should be easier for clinicians to monitor response to treatment, and thus clinical decision making should be facilitated. If this is not what has been happening, a close look at the process by which diagnoses are developed is in order.

It is difficult to know exactly how decisions were made in earlier DSMs, and it is much too early to evaluate how decisions are being made for DSM-V. However, it appears that the single most important conceptual decision is one clear definition of "mental disorder" to guide the search of all the work groups focused on different mental disorders. If we don't know what "signals" we're trying to build a detector for, it is difficult to build an excellent detector. Then evaluation of the specific diagnostic categories should take place within the population of those with mental disorders. It is easy enough to distinguish between someone with a mental disorder and someone who is a completely healthy "control." The most valuable uses of any diagnostic system are to distinguish

among those with different disorders in order to make appropriate clinical decisions for individual patients, and to further researchers' understanding of the cause, course, and cure of each such disorder.

The most salient methodological deviation in the process described above has been the exclusive reliance on categorical diagnoses, which will apparently change with DSM-V. Relying exclusively on *CDX*s is analogous to using a signal detector with the gain turned down to a very low level: We can't hear the signals, much less tell whether the settings on the detector, its directional orientation, and its tuning are optimal or not.

Certainly pointing the detector in the proper direction would help. Optimal diagnosis may be quite different in community and clinical populations, and, within any population, for screening, discrimination, and definitive diagnosis. To date, results from community studies have been used to make decisions about diagnosis in clinical populations, and vice versa. As a result, little attention has been paid to the different clinical uses to which a diagnosis is likely to be put (screening, discrimination, definitive diagnosis) because a single categorical diagnosis has been proposed for each disorder. Finally, little attention has been paid to the validity of a particular diagnosis for different subgroups defined by gender, age, or ethnicity.

Powerful statistical methods that enhance detection of disorders have long existed, but these have had little impact on diagnosis. However, the results such methods produce depend crucially on the quality of the indicators offered for application of those methods. Removing unreliable indicators, redundant indicators, and indicators that share strong contaminants prior to application of such methods, and measuring all indicators as much as possible on a dimensional scale, would all enhance the outcomes of such application. Current lists of signs and symptoms frequently include unreliable, redundant, and contaminated indicators. Thus it is not clear how much would have been accomplished had excellent statistical methods been applied to poor-quality data. Finally, what results from application of such statistical methods does not guarantee validity in the future. There must be subsequent documentation both of reliability, which would be nearly a foregone conclusion, and validity, which would not.

When a diagnosis is proposed, the emphasis to date on reliability is necessary, of course, but the exclusive emphasis in the past on interobserver reliability rather than test–retest reliability has exaggerated the quality of the diagnosis. Moreover, the exclusive focus on reliability, to the exclusion of validity, is a continuing major conceptual and methodological problem. If we are in the process of constructing a very fine detector for the wrong signal, how do we recognize this? The major worry among those anticipating DSM-V has been that the errors of the past will simply be repeated in the new revision. Moving from exclusive reliance on categorical diagnosis to inclusion of a corresponding dimensional diagnosis would be one major step away from that possibility—one that might facilitate other steps away from such errors.

Above all, it has not been usual practice to challenge the validity of the diagnostic definitions, but that would be an important addition to the evaluation of DSM-V. How to present such challenges is, of course, the major question to be answered.

Perhaps the primary impediment to a major advance in diagnosis is the statement "But that's the way we've always done it!" If in the last 30 years, with DSM-III, DSM-III-R, and DSM-IV, there had been major advances in identifying the specific etiology of specific mental disorders, major advances in preventing their onset, and major advances in inducing early remission or even recovery among those with mental disorders, such a statement might be understandable. However, few would claim that such major advances have been made. What we need now instead is a consciousness of the weaknesses of approaches used in earlier iterations, a new focus on the resulting weaknesses of the system, incorporation of new information gained over the last 30 years about mental disorders, and new methodological approaches to the problem.

Note

1. The term "confounder" is often used here, but here *C* and *D* are defined as completely inde-

pendent of each other—a restriction often not satisfied by variables called "confounders."

References

Acion, L., Peterson, J. J., Temple, S., & Arndt, S. (2006). Probabilistic index: An intuitive nonparametric approach to measuring the size of treatment effects. *Statistics in Medicine*, *25*(4), 591–602.

Altman, D. G. (1998). Confidence intervals for the number needed to treat. *British Medical Journal*, *317*(7168), 1309–1312.

Bossuyt, P. M., Reitsma, J. B., Bruns, D. E., Gatsonis, C. A., Glasziou, P. P., Irwig, L. M., et al. (2003). Towards complete and accurate reporting of studies of diagnostic accuracy: The STARD Initiative. *Annals of Internal Medicine*, *138*, 40–44.

Brown, W. (1910). Some experimental results in the correlation of mental abilities. *British Journal of Psychology*, *3*, 296–322.

Brownie, C. (1988). Estimating Pr(X < Y) in categorized data using "ROC" analysis. *Biometrics*, *44*, 615–621.

Cohen, J. (1983). The cost of dichotomization. *Applied Psychological Measurement*, *7*(3), 249–253.

Cook, R. J., & Sackett, D. L. (1995). The number needed to treat: A clinically useful measure of treatment effect. *British Medical Journal*, *310*, 452–454.

Frank, E., Prien, R. F., Jarrett, R. B., Keller, M. B., Kupfer, D. J., Lavori, P. W., et al. (1991). Conceptualization and rationale for consensus definitions of terms in major depressive disorder: Remission, recovery, relapse, and recurrence. *Archives of General Psychiatry*, *48*, 851–855.

Galen, R. S., & Gambino, S. R. (1975). *Beyond normality: The predictive value and efficiency of medical diagnoses*. New York: Wiley.

Grissom, R. J. (1994). Probability of the superior outcome of one treatment over another. *Journal of Applied Psychology*, *79*, 314–316.

Hanley, J. A., & McNeil, B. J. (1982). The meaning and use of the area under a receiver operating characteristic (ROC) curve. *Radiology*, *143*, 29–36.

Kraemer, H. C. (1991). To increase power without increasing sample size. *Psychopharmacology Bulletin*, *27*(3), 217–224.

Kraemer, H. C. (1992a). *Evaluating medical tests: Objective and quantitative guidelines*. Newbury Park, CA: Sage.

Kraemer, H. C. (1992b). How many raters?: Toward the most reliable diagnostic consensus. *Statistics in Medicine*, *11*, 317–331.

Kraemer, H. C. (1995). Statistical issues in assessing comorbidity. *Statistics in Medicine*, *14*, 721–733.

Kraemer, H. C., Kazdin, A. E., Offord, D. R., Kessler, R. C., Jensen, P. S., & Kupfer, D. J. (1999). Measuring the potency of a risk factor for clinical or policy significance. *Psychological Methods*, *4*(3), 257–271.

Kraemer, H. C., & Kupfer, D. J. (2006). Size of treatment effects and their importance to clinical research and practice. *Biological Psychiatry*, *59*(11), 990–996.

Kraemer, H. C., Periyakoil, V. S., & Noda, A. (2002). Tutorial in biostatistics: Kappa coefficients in medical research. *Statistics in Medicine*, *21*, 2109–2129.

Kraemer, H. C., & Thiemann, S. (1987). *How many subjects?: Statistical power analysis in research*. Newbury Park, CA: Sage.

MacCallum, R. C., Zhang, S., Preacher, K. J., & Rucker, D. D. (2002). On the practice of dichotomization of quantitative variables. *Psychological Methods*, *7*(1), 19–40.

McGraw, K. O., & Wong, S. P. (1992). A common language effect size statistic. *Psychological Bulletin*, *111*, 361–365.

McNeil, B. J., Keeler, E., & Adelstein, S. J. (1975). Primer on certain elements of medical decision making. *New England Journal of Medicine*, *293*, 211–215.

Rush, A. J., Kraemer, H. C., Sackeim, H. A., Fava, M., Trivedi, M. H., Frank, E., et al. (2006). ACNP Task Force: Report by the ACNP Task Force on response and remission in major depressive disorder. *Neuropsychopharmacology*, *31*(9), 1841–1853.

Spearman, C. (1910). Correlation calculated from faulty data. *British Journal of Psychology*, *3*, 271–295.

Veiel, H. O. F. (1988). Base-rates, cut-points, and interaction effects: The problem with dichotomized continuous variables. *Psychological Medicine*, *18*, 703–710.

CHAPTER 18

The Integration of Categorical and Dimensional Approaches to Psychopathology

ERIK SIMONSEN

For a long time, the developers of the DSM and ICD classification systems have considered including a dimensional, quantifying component in these systems. In this chapter, I briefly describe the different taxonomic systems used in psychiatry; highlight the advantages and disadvantages of categorical versus the dimensional approaches; discuss alternative models; and give examples of the most recent taxonomic research on the major diagnostic categories (personality disorders, psychoses, mood disorders, anxiety disorders, substance abuse/dependence). Finally, I present some general recommendations for the future of psychiatric classification.

The Taxonomic Systems

Beneath the dilemmas of different taxonomic systems, there are underlying philosophical and other basic conceptual issues. The ways in which we fundamentally conceive of mental disorders and see patients as persons shape our methodology and nosology. Scientific progress in clinical psychiatry has been influenced mainly by logical positivism. In general, psychiatry has been presumed to be more scientific when causal explanations, quantification of symptoms, and operationalized concepts are in use. The "categorical approaches" used in medicine are the logical consequences of this paradigm. ICD-10 (World Health Organization, 1992) and DSM-IV-TR (American Psychiatric Association, 2000) specify the necessary and sufficient conditions for class membership, or "family resemblance." A number of attributes are listed for each diagnosis; some are necessary for class membership (i.e., general criteria), but most often a patient has to fulfill a certain number of criteria from a list of criteria for a specific diagnosis. This approach is "polythetic," compared to the "monothetic" approach, where all criteria in the list have to be fulfilled for the diagnosis. In the categorical models, a diagnosis is seen as a discrete entity, and a person either has or does not have the illness in question.

The categorical approaches have tied clinicians to the kind of doctor–patient relationship where a doctor is expected to question a patient in a medical framework and then to act. Schwartz and Wiggins (1987) argued that this "subordination of scientific knowledge to practical purposes makes psychiatry

a practical discipline rather than pure science" (p. 279). They proposed an alternative way of seeing a patient as a person. Referring to the work of German social scientist Max Weber, they expressed the view that idiographic methodology might be more fruitful in psychiatry. Karl Jaspers (1913/1963) and Kurt Schneider (1962) used Weber's conception of "ideal types" in their nosology; Schwartz and Wiggins recommended this approach under the name of "prototypes." Ideal types or prototypes are idealized definitions of typical cases—the best examples of diagnostic concepts. They are useful in orienting clinicians' questioning of their patients: In pinpointing the common concept of a particular illness, a prototype can help a clinician decide what is unique about a particular patient. The prototype approach may in fact also be used in a dimensional way: The patient under evaluation may match the description of the ideal type or prototype to a greater or lesser extent. Deviations from the ideal case raise questions regarding the nature of these deviations—for example, about genetic versus environmental influences.

"Dimensional approaches" locate individuals along one or more symptomatic or psychological dimensions (e.g., degrees of depression, degrees of negative symptoms, degrees of alcohol abuse, and/or degrees of aggression). Dimensions are most often defined as discrete from each other, but they may overlap. The dimensions can either be unidirectional or bidirectional. In the unidirectional form, the psychopathology is measured from zero to the highest point on a scale. In the bidirectional form, the endpoints of a dimension are contrasting phenomena (e.g., activity vs. passivity, depression vs. mania).

When clinicians talk about their patients after knowing them well, they normally think about the patients as persons. They will not think of the patients as categories or as rows and columns of dimensions. Diagnostic terms, whether categorical or dimensional, are ways of labeling groups of clinical phenomena for the purpose of easing the work of psychiatrists and psychologists. In the following section, I discuss the advantages and disadvantages of the two approaches.

The Pros and Cons of Categorical and Dimensional Approaches

To evaluate the advantages and disadvantages of categorical and dimensional approaches, one must bear in mind the overall goals of a diagnostic classification. One is to facilitate the clear exchange of information and discussion of ideas. Another is clinical utility: A classification system should not only be utilized as the basis for a choice of treatment, but should also be used to specify the likelihood of treatment response, as well as the probable course and outcome. Epidemiologists and those making decisions about service delivery need precise information about prevalence and incidence to identify causes, outline education programs, and develop plans for intervention; a classification system makes it possible to obtain such information. Finally, classification should provide a framework for organizing and retrieving research data.

Advantages of Categorical Approaches

Table 18.1 summarizes the advantages and disadvantages of categorical approaches. A major advantage is that categories are easy to report and remember; clinicians can employ the categories in making rapid diagnoses with numerous patients seen briefly. Another advantage is that psychiatrists are educated with this kind of conceptualization of clinical conditions. That is, from their medical training psychiatrists have become familiar with categorical concepts, which convey a large amount of information and provide direction for practical behavior. Categorical diagnoses are used to make decisions about an individual (to treat or not to treat, to treat with a drug or with psychotherapy, to use this drug or that drug, etc.). Our existing knowledge about the presentation, course, and treatment of mental disorders has been generated by using categories.

Moreover, most taxa in nature (species of living organisms) appear as being all-or-none categories. Accordingly, in our daily lives we are accustomed to this way of thinking—ranging from a child's acquisition of cognitive maps of the world to the creation of scientific theories. Certain features

TABLE 18.1. Advantages and Disadvantages of Categorical Approaches to Classification

Advantages	Disadvantages
• Familiarity from diagnostic process in somatic medicine • High reliability • Ease of use • Better correspondence with a heuristic view of phenomena • Knowledge of etiological factors, course, and outcome • Ease of clinical decision making (either–or)	• Reification • Risk of mechanical, "top-down" diagnostics • Artificial comorbidity • Overlap between different diagnoses • Inadequate and inconsistent coverage of syndromes • Striking differences of patients within same category • Unclear and arbitrary boundaries between the normal and abnormal • High proportion of atypical/unclassified cases

are given priority and centrality; others are not overlooked, but merely assigned lesser significance. In categorizing a patient in this manner, a clinician can perceive the unity of the patient's pathology. Categories also satisfy practical needs for service planning, outlining research, research designs, and requestioning.

A final important advantage of the categorical approach concerns reliability. Without reliability, it would be meaningless or impossible to address validity. Much effort has been put into strengthening reliability. It is one of the major advantages since the introduction of DSM-III (American Psychiatric Association, 1980) that psychiatric diagnoses have become more reliable.

Disadvantages of Categorical Approaches

Clinicians often claim that the better they know patients, the greater the difficulty they have in fitting them into a category. They are well aware of the limitations of categorical diagnoses. Although categories are the tools we create to help us coordinate and focus our observations, clinicians are well aware that our diagnostic concepts are constructs. One major disadvantage of categorical approaches is that these diagnostic concepts become reified. People tend to think that if a category is listed in the official classification system, it is "real," existing in nature. They succumb to the fallacious belief that syndromes comprise nosological "diseases" instead of syndromatical concepts. There is also the danger that reified diagnoses may be assigned in a "top-down," mechanical manner.

Moreover, the current categorical classifications routinely fail to meet the goal of guiding a clinician to the presence of only one specific disorder. Studies have consistently indicated that because of unclear boundaries between disorders, many patients meet diagnostic criteria for several different disorders, particularly within the group of personality disorders and within the groups of anxiety and depressive disorders. Also, most studies have failed to demonstrate that schizophrenia and depression are discrete, homogeneous clinical entities (Kendell, 1989). Different, but still equally reasonable, diagnostic criteria for schizophrenia have been shown to yield different numbers of schizophrenia cases (Jansson & Parnas, 2007). Jansson and Parnas claim that the boundaries of schizophrenia based on clinical data are only conventions, and that schizophrenia remains a "fuzzy concept."

In addition to the problems of excessive diagnostic co-occurrence and arbitrary boundaries, there is a complementary problem of inadequate coverage. One approach to this problem is to add more diagnostic categories, but there is considerable reluctance to do so—in part because this would have the effect of further increasing the difficulties of diagnostic comorbidity and differential diagnosis. The lack of homogeneity among individuals who share the same diagnosis is also a problem. Patients with the same diagnosis will vary substantially with respect to which diagnostic criteria were used to make the diagnosis, and the differences are not trivial. If the presence or absence of five out of nine criteria is noted, for example, there

are potentially 126 different syndromes. An additional problem is the arbitrary boundary between normal psychology and psychopathology or between normal behavior and maladaptive functioning. Seemingly minor changes to diagnostic criterion sets have resulted in unexpected and substantial shifts in prevalence rates with each new edition of the diagnostic manuals (Blashfield, 1993). A relatively high proportion of atypical and unclassified cases is yet another limitation of categorical approaches.

Advantages of Dimensional Approaches

Table 18.2 summarizes the advantages and disadvantages of dimensional approaches. The major advantage of dimensional models is their ability to explore and delineate the differences among individuals. They combine several clinical attributes in a single configuration; this allows a clinician to produce a comprehensive clinical description with minimal information lost, as compared to categories, where a single (albeit distinctive) characteristic or set of characteristics is brought to the forefront. Also, in the dimensional systems the representation and assignment of unusual and unique factors make a difference because cases are not "forced" into procrustean categories for which they are ill suited. Each person is represented by a profile of different scores introducing quantitative variations and graded transitions between normality and pathology, without arbitrary cutoffs. In addition, dimensional approaches facilitate the diagnosis of more nonspecific symptoms and subthreshold conditions (e.g., minor degrees of anxiety and depression, which account for a great many mental health problems in primary care settings). They provide maximum flexibility for later categorization and are also superior for reporting changes. The interval scores seem to be more stable over time and to display higher reliability than dichotomous measures. Moreover, dimensional scores are relatively unaffected by minor shifts in psychopathology, whereas modest changes can move an individual either above or below a diagnostic threshold in a categorical approach. Finally, it should be mentioned that all drug trials use dimensional measures such as observer ratings or self-reports to assess treatment effects.

Disadvantages of Dimensional Approaches

Although the advantages of dimensional approaches have been described for several decades, they have not fared well in the diagnosis of psychopathology (Millon, 1987). There are several reasons for this. There is little agreement among theorists and researchers on the number of dimensions necessary to represent psychopathological phenomena. And if the numbers of imposed dimensions become too high, they produce complex and intricate schemas that require geometric or algebraic representations. They will then become too complicated to use for routine clinical assessment or decision making. Another major limitation of the dimensional approaches is that they generate considerable difficulties both in comprehension and in communication among professionals. Dimensional descriptions of symptoms may create convenience and ease in the short run, but they need to be arranged in groups before the information they contain can be commu-

TABLE 18.2. Advantages and Disadvantages of Dimensional Approaches to Classification

Advantages	Disadvantages
• No loss of information • More nuanced descriptions of psychopathology • More individual descriptions of unique factors • Better descriptions of unusual cases • Quantitative gauging of features • Perhaps greater ease in linking to treatment • Better-graded transitions between normality and pathology • Better monitoring of changes over time	• Disagreement on which dimensions to use • Possibility of becoming closed systems of dimensions • Break with the view of seeing the patient as a whole person • Increased difficulty of communication in everyday practice • Excessive complexity for clinical use

nicated. When a population has been identified as having a similar profile, there will soon be a need to conceptualize the clinical condition in a higher-order domain—a category. One might also be concerned whether the average clinician can handle complex systems based on mathematical approaches. Finally, validity becomes difficult to obtain, since the structure and the description of the psychopathological presentations will vary extensively.

Alternative Models: Latent-Structure Models

In recent years, various sophisticated statistical techniques known as "latent-structure models" (including latent-class, latent-trait, and taxometric analyses) have been used to address the issue of categorical versus dimensional approaches. The two types of approaches are not mutually exclusive. Kraemer, Noda, and O'Hara (2004) have argued that the approaches are fundamentally equivalent, and that whether one or the other is more appropriate depends on the clinical situation or research questions to be addressed. It is not a question of deciding which is right and which is wrong, but rather of clarifying under which circumstances one should utilize one approach over the other. Kraemer and colleagues emphasize that the approaches interdigitate, and that any categorical approach can be converted to a dimensional one and vice versa. They also observe that while dimensional approaches are preferred for outcome measures, categorical approaches are needed in research to define inclusion and exclusion criteria. They show that it is not a categorical approach to diagnosis itself that forms a weak basis for research, but the missetting of cutoff points. The arbitrary and subjective choice of cutoff points is what undermines the effectiveness of categorical approaches.

Both the DSM and ICD systems have mainly adopted a polythetic categorical approach for all their diagnoses. The generation of cutoff points in these systems for categorical classification is already an instance of dimensional thinking. The major challenge is, however, analyzing the extent to which the different categories are in fact homogeneous dimensions or syntheses of a number of lower-order dimensions. In earlier research, cluster analyses were used to extract major complexes of symptoms within diagnostic entities. The newer latent-structure models, such as latent-class and latent-trait models (item response theory), have generated new ideas and data in the search for underlying coherence of psychopathological phenomena. A latent-class analysis assumes that "sick" and "well" categories exist, and that the co-occurrence of a set of symptoms is best explained by patterns of mutually exclusive underlying categories. In a latent-trait analysis, symptoms are conceptualized by their location along a continuum relative to other symptoms and by their ability to discriminate between persons at the nearby levels of severity. These mixed models have the potential for meeting the scientific need and the human desire to organize and categorize, while still accounting for individual differences.

In medicine, interval scales have been used successfully to define levels at which diagnoses are assigned and treatment is given. Examples of the close link between diagnostic criteria and thresholds include measures of blood pressure and blood glucose. The level of diagnosis is driven and determined by empirical findings, such as a strong indication of increasing rates of related disorders (in the case of blood pressure, heart disease, and apoplexy), morbidity, and mortality beyond the diagnostic threshold. At the current level of knowledge, psychiatry still has to tie classification and diagnostic criteria mainly to descriptive validity. We are not able to determine diagnostic thresholds by using interval scales, as in medicine. Nevertheless, ordinal scales would still be better to use than binary dichotomies, which are most commonly used in the current classification systems. Latent-structure models and other sophisticated statistical techniques (structural modeling, receiver operating characteristics analyses) now available for use with clinical data will probably play a more important role in the future and enhance both the clinical validity and the utility of psychiatric diagnoses.

Comorbidity

A very important issue and challenge for future classification systems is to address the

high comorbidity of some of the major categories in the DSM system—for instance, the comorbidity between schizophrenia and bipolar disorders, between major depression and generalized anxiety disorder, and within the groups of anxiety disorders and of personality disorders. In all four examples, the diagnostic categories share a number of biological, antecedent, concurrent, and predictive external validators. The hierarchical nature of the ICD system and the "horizontal" nature of the DSM system challenge this issue in different ways. The ICD classification follows the tradition of Jaspers's (1913/1963) hierarchy and ranks the disorders as follows: The lowest F digits (F0 and F1) are used for organic disorders, including psychoactive substance use disorders; F2 is used for psychotic disorders and mood disorders, which also may be psychotic; and F3–F6 are used for nonpsychotic disorders. The hierarchy gives preference to diagnoses that may imitate or include disorders with lower rank and higher F-code numbers. The advantage of this system is that the classification system gives priority to diagnoses for which the organic etiology is more clearly validated; however, the clinical utility of this distinction is unclear. By the use of the hierarchical system, ICD-10 avoids comorbidity except for psychoactive substance use disorders. If a schizophrenia diagnosis is justified, clinicians are not allowed to include "comorbid" diagnoses like anxiety disorders or personality disorders. According to exclusion criteria, these diagnoses cannot be used in the presence of a psychotic disorder. But comorbid syndromes in schizophrenia may have important relevance for treatment, treatment alliance, and response to treatment—perhaps even more than the disorder itself. Therefore, to enhance clinical utility of the classification system, it would be important to acknowledge the presence of these syndromes for the benefit of treatment planning.

In the DSM system the number of categories has steadily grown since the radical change to the polythetic system in 1980. The validity of each category is sparse, and the delineation between categories remains blurred. However, when clinicians adhere strictly to the diagnostic criteria, most patients come out with a number of diagnoses. The extensive use of diagnoses in the DSM system can pull clinicians away from a more person-focused, integrated system of diagnosis. One aim (among others) of creating Axis II was to enable clinicians to perceive the clinical syndromes within the context of a personality-explanatory system. So the high comorbidity is likely to cut the clinical problem into more pieces, without any clear advantage for clinical practice.

The Spectrum Model

The "spectrum model" is widely used by clinicians nowadays to conceptualize related clinical phenomena. Most notable are the concepts of the schizophrenia spectrum, the affective spectrum, the panic–agoraphobic spectrum, and the obsessive–compulsive spectrum (Akiskal et al., 2003; Cassano et al., 1997; Hollander, 2005; Lara, Pinto, Akiskal, & Akiskal, 2006).

The spectrum model provides an operational system for describing a range of related psychopathological phenomena—the idea of seeing disorders within a spectrum of symptoms that emerge in different patterns from core pathology. This approach appeals to clinicians because it is related to current treatment potential within a spectrum, and because it makes clinical sense that there is a gradation from milder to more severe cases within the same core pathology. One way of elaborating this system is to specify by criteria for each diagnosis the symptoms, the duration of illness, and the functional impairment for the patient's location on the spectrum. One of the weaknesses of this model is that it cannot be used as a general model for all psychiatric disorders, but only for certain clusters of disorders. However, the spectrum approach has been shown to be of clinical significance in detecting subclinical phenomena (Akiskal, 1994; Kendler et al., 1993). Symptoms in the absence of a specific disorder may play an important role as predictors of outcome (Frank et al., 2002).

Research on Specific Disorders

Personality Disorders

The personality disorders are the group of disorders for which the advantages of a dimensional approach are most obvious.

However, several different dimensional models exist. Widiger and Simonsen (2005) have found a number of similarities among these models and illustrated how most of the dimensional scales can be well integrated within four broad domains of adaptive and maladaptive personality functioning, which serve as the top level of a hierarchy: two internalizing dimensions (emotional dysregulation, and introversion vs. extroversion) and two externalizing dimensions (antagonism vs. compliance, and impulsivity vs. constraint). We have further indicated how the existing diagnostic criteria for personality disorders can be readily incorporated within this hierarchical structure, and how the existing personality disorder constructs (e.g., antisocial or borderline) can be recovered through diagnostic algorithms by using personality trait scales.

Research questions on dimensional approaches should of course be directed primarily toward maximizing clinical utility, but an integrated model that coordinates several models should also be validated with respect to the significance of genetic, neurobiological, and developmental aspects (Simonsen & Tyrer, 2005). Some studies have compared categorical and dimensional models. A recent taxometric analysis of 1,146 males with criminal records and substance abuse who were assessed by self-report and semistructured interview suggest that antisocial personality disorder exists on a continuum (Marcus, Lilienfeld, Edens, & Poythress, 2006), thus contradicting the idea that psychopathy should be a taxonomic categorical construct. In a similar study of 1,389 outpatients assessed by semistructured interview, Rothschild, Cleland, Haslam, and Zimmerman (2003) indicated that borderline personality disorder does not represent a latent category, supporting a dimensional view of the latent structure of this disorder.

For clinicians, it would be most straightforward to adopt a system in which "general personality disorder" is maintained as a separate category, most appropriately on Axis I in the DSM system. The global definition of general personality disorder should include a person's ability to adapt to everyday tasks in occupational and intimate relationships, and to maintain a stable view of self and others. Furthermore, the current system of different personality disorder categories should be elaborated and refined as prototypes. It would then be possible to include dimensional representations of these new prototypes in the forthcoming ICD and DSM systems; a clinician would then be able to determine to what extent a person resembles the features of each prototype under consideration. Refined assessments and descriptions of personality traits should then be added, using available empirically validated instruments.

Psychotic Disorders

Dimensional approaches have been used in the clinical assessment of psychotic disorders for the last decade. Cross-sectional and lifetime dimensional representations of psychopathology (e.g., severity of hallucinations), when compared to diagnostic categories, have shown to be more strongly associated with important clinical parameters (van Os et al., 1999). In a study of 980 patients with psychosis Rosenman, Korten, Medway, and Evans (2003) showed that dimensional measures of psychopathology explained more of the variance in service demand, dysfunctional behavior, social adaptation, and global and occupational functioning than did the categorical diagnoses. Only the use of support services and illness course were better predicted by categorical diagnoses. Rosenman et al. concluded that the dimensions provided significant extra information not provided by the categorical diagnoses, and that dimensions would thus be a more useful basis for clinical management. Psychotic symptoms may be more frequent in the general population than we used to think (Myin-Germeys, Krabbendam, & van Os, 2003). They seem to be distributed more widely, due to different combinations of risk factors and different kinds of phenotypic expressions on the continuum from normality to psychotic disorders (Wiles et al., 2006).

Clinicians are also getting used to adopting a dimensional view in the treatment of psychotic disorders. The rating of psychotic symptoms, negative symptoms, depressive symptoms, and manic symptoms as dimensions are more routinely used in clinical settings to ensure the best monitoring of pharmacological treatment. The introduction of a formal dimensional approach in future classification systems would facilitate this development and at the same time acknowl-

edge the dimensional nature of psychotic phenomena. Domains for quantitative scores that would be clinically useful might include deficiency in reality testing (hallucinations and severity of delusions), disorganization of thoughts, cognitive dysfunction, negative symptoms, and social impairment.

A combination of categorical and dimensional approaches would optimize the classification of psychotic disorders; facilitate both communication and decision making; and also enhance clinical utility by measuring treatment response, course, and outcome. Such a compromise, a categorical–dimensional model, will probably be a considerable advance over our current classification systems (Dutta et al., 2007)

Mood Disorders

Clinicians also conceptualize depressive symptoms as points on continua rather than as discontinuous entities. At present, there is extensive use of quantitative scores in mood disorders—most notably the use of scores on clinical rating scales (e.g., the Hamilton Rating Scale for Depression; Hamilton, 1980) or self-report measures (e.g., the Beck Depression Inventory–II; Beck, Steer, & Brown, 1996). Such instruments are often used as benchmarks for remission, improvement, and recovery, and they permit clinical audits of practice. Dimensional approaches to clinical assessment of mood disorders have also been available to clinicians for a long time (not only psychiatrists, but also general practitioners). The categorically based classification does not reflect important variations in phenomenology and phenotypes as evidenced in epidemiological data. Researchers have examined continuity in terms of whether the symptoms in DSM-IV-defined mild, moderate and severe depression differ in degree along a continuum (quantitative) or in kind (qualitative). Symptom severity has clinical and neurobiological correlates, and it is a critical dimension for predicting treatment response. There is some evidence of quantitative continuity of symptoms. In population-based community samples, more studies favor a dimensional model as the best way to conceptualize, measure, and classify depression (Hankin, Fraley, Lahey, & Waldman, 2005; Slade & Andrews, 2005). However, there is also some evidence of a possible discontinuity, where biological factors (somatic symptoms) seem to play a more important role than for psychological depressive symptoms (Grove et al., 1987).

A taxometric analysis was conducted to test the hypothesis that the latent structure of melancholia in 378 adolescents presented for depression evaluation would support a latent categorical variable (Ambrosini, Bennett, Cleland, & Haslam, 2002). The dimensional model contributed predictive validity only at a trend level over and above DSM-III diagnoses for mean level of depression in a 6-month prospective follow-up study, and this model did not contribute to incremental validity for family history variables or for recovery (Shankman & Klein, 2002). The authors pointed out that categorical diagnoses are inherently multidimensional (multifactorial). They include symptoms and duration as well as exclusion criteria, and thus incorporate a number of attributes that many dimensional approaches fail to consider.

In several studies, it has been shown that the relationship between number of symptoms and functional impairment in depressive disorders is linear (Sakashita, Slade, & Andrews, 2007; Üstün & Sartorius, 1995). No evidence supports continuity and the use of a certain number of symptoms to delineate between normality and abnormality.

Some combination of categorical and dimensional approaches to depressive disorders is already in use in many clinical settings and in primary care. The nature and validity of the dimensions, and which ones to include in future classification systems, remain to be researched. As with the dimensions underlying the psychotic disorders, the dimensions should reflect the continuum from normal to abnormal, but should also obtain information relevant for treatment.

Anxiety Disorders

Anxiety disorders are highly comorbid not only with one another, but also with depression and personality disorders. This has been the case ever since the anxiety and phobic neuroses were changed to a range of new categories with the introduction of DSM-III in 1980. This has led to much criticism of the current categorical approach. Goldberg and colleagues have conducted several stud-

ies of anxiety and depression in primary care. They have shown that depression and anxiety are highly correlated affects. Their early data suggest that there is no point of rarity between the symptoms that make up an anxiety cluster and a depression cluster, but an unbroken continuum of various combinations of these two affects (Goldberg, Bridges, Duncan-Jones, & Grayson, 1987). The structural relationship of key features in the group of anxiety disorders and the underlying higher-order trait dimensions have drawn much attention in research. Studies of fear circuitry and hypothalamic–pituitary–adrenal axis pathophysiology may well suggest or support a dimensional approach. Various DSM anxiety disorders respond similarly to the same drugs, which may also reflect a common underlying dimension such as negative affect. Taxometric analyses of patients with posttraumatic stress disorder and patients with chronic worries likewise support a dimensional approach (Asmundson et al., 2000; Ruscio, Borkovec, & Ruscio, 2001).

As in clinical research on psychotic disorders and depression, there is a tradition in clinical research on anxiety disorders of employing dimensional assessment to evaluate treatment results, but these instruments have not been part of everyday clinical practice to the same extent except for behavior therapy. The anxiety disorders seem to share a number of psychopathological phenomena, in which quantity, frequency, intensity, and/or severity are converted to unidimensionalized scores (i.e., worries, panic feeling, anticipatory anxiety, avoidance behavior, and cognitive symptoms). This alternative approach of assessing shared symptom domains among anxiety disorders also provides a way of rating anxiety symptomatology in other disorders, such as substance abuse, depression, personality disorders, and psychotic disorders.

The Yale–Brown Obsessive Compulsive Scale (Goodman et al., 1989) is an example of a multidimensional scale that rates the severity of criterion symptoms. It was developed to measure treatment change in research studies of obsessive–compulsive disorder, but it has become a rather unique example of how a dimensional scale for rating symptoms resembling DSM criteria has now been adopted by clinicians as well.

Substance Abuse and Dependence

Traditionally, alcoholism was defined in terms not only of distinct types, but of severity of alcoholism (Jellinek, 1960). Options for coding diagnoses as mild, moderate, or severe were made explicit in DSM-III-R (American Psychiatric Association, 1987). Recent research has consistently shown that alcohol problems are best arrayed along a continuum of increasing severity of illness, rather than classified into unique categories (Helzer et al., 2006; Krueger et al., 2004; Lynskey et al., 2005). Symptoms of alcohol dependence and abuse in different clinical samples and in the general population indicate a continuum of severity (Mitchell & Plunkett, 2000). Latent-class model analyses (item response theory) in population-based studies of multiple-drug abuse also indicate such a continuum (Pedersen & Skrondal, 1999). However, for dependence on substances other than alcohol, the findings (in a far lower number of, and far less sophisticated, studies) have not been that consistent. And for tobacco dependence, studies in fact have indicated that there is no dimensionality.

At least for alcohol abuse and dependence, therefore, gradation and weighting of each of the symptoms and criteria should simply be dimensionalized (i.e., "no/mild/severe" or "never/sometimes/frequent"). This quantification would enable clinicians to come to decisions about the needed level of interventions.

Conclusions

Most sciences start with a categorical classification of their subject matter, but later replace this with dimensions as more accurate measurement become possible (Hempel, 1961). Most symptoms are dimensional in nature, as are biological phenomena. Every dimensional approach can be made categorical by setting a cutoff point. The current categorical classification systems seem to impede exploration of validity issues because the systems work with dichotomous rather than continuous variables. However, if our classification systems are to incorporate more empirical data, they should be better integrated with the concepts and measuring units used in neurobiology (e.g., in genetics, neuroimaging, or neurochemistry). They should also

include the dimensional elements of neurobiological data to add construct validity to descriptions. Early markers of subtle phenomena of psychopathology and symptoms in the prodromal phase of illnesses are more easily detected within dimensions, and dimensions also permit more sensitive descriptions of course and treatment outcome. A dimensional element would not only enhance statistical power in research, but also lead to more focused treatment and improved predictive validity. The major categorical diagnoses may easily, as described above, include dimensional elements in their phenomenology and clinical presentation—either within symptoms, the number of symptoms, symptom severity, years of duration, or functional impairment.

The current categorical classification systems, however, should be preserved. A large body of important empirical data has been developed since the introduction of DSM-III criteria in 1980, and several instruments have been designed for making reliable categorical diagnoses. Criteria-based classification is a sine qua non for the reliability of psychiatric diagnoses, but it has fueled high rates of artificial comorbidity of diagnoses, which share much of the same latent structure and equivalent treatment. Patients should be described in terms of diagnostic groups, but details about their psychopathology should be recorded by quantitative profiles of different, clinically useful measures. It would be beneficial to include several of the already established dimensional instruments as supplements to the existing diagnostic procedures. This should lead to higher diagnostic accuracy, more individualized assessment, and a better background for treatment planning.

The German philosopher Hegel argued that synthesis will follow thesis and antithesis; it is not a question of choosing either one system or the other. In my opinion, we are ready for a synthesis and integration of the categorical and dimensional approaches to the diagnosis and classification of psychiatric disorders.

References

Akiskal, H. S. (1994). The temperamental borders of affective disorders. *Acta Psychiatrica Scandinavica*, *89*, 32–37.

Akiskal, H. S., Hantouche, E. G., Allilaire, J. F., Sechter, D., Bourgeois, M. L., Azorin, J. M., et al. (2003). Validating antidepressant-associated hypomania (bipolar III): A systematic comparison with spontaneous hypomania (bipolar II). *Journal of Affective Disorders*, *73*, 65–74.

Ambrosini, P. J., Bennett, D. S., Cleland, C. M., & Haslam, N. (2002). Taxonicity of adolescent melancholia: A categorical or dimensional construct? *Journal of Psychiatric Research*, *36*, 247–256.

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., rev.). Washington, DC: Author.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.

Asmundson, G. J., Frombach, I., McQuaid, J., Pedrelli, P., Lenox, R., & Stein, M. B. (2000). Dimensionality of posttraumatic stress symptoms: A confirmatory factor analysis of DSM-IV symptom clusters and other symptom models. *Behaviour Research and Therapy*, *38*, 203–214.

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Beck Depression Inventory–II manual*. San Antonio, TX: Psychological Corporation.

Blashfield, R. K. (1993). Variants of categorical and dimensional models. *Psychological Inquiry*, *4*, 95–98.

Cassano, G. B., Michelini, S., Shear, M. K., Coli, E., Maser, J. D., & Frank, E. (1997). The panic–agoraphobic spectrum: A descriptive approach to the assessment and treatment of subtle symptoms. *American Journal of Psychiatry*, *154*, 27–38.

Dutta, R., Greene, T., Addington, J., McKenzie, K., Phillips, M., & Murry, R. M. (2007). Biological, life course, and cross-cultural studies all point toward the value of dimensional and developmental ratings in the classification of psychosis. *Schizophrenia Bulletin*, *33*, 868–876.

Frank, E., Cyranowski, J. M., Rucci, P., Shear, M. K., Fagiolini, A., Thase, M. E., et al. (2002). Clinical significance of lifetime panic spectrum symptoms in the treatment of patients with bipolar I disorder. *Archives of General Psychiatry*, *59*, 905–911.

Goldberg, D. P., Bridges, K., Duncan-Jones, P.,

& Grayson, D. (1987). Dimensions of neuroses seen in primary-care settings. *Psychological Medicine*, *17*, 461–470.

Goodman, W. K., Price, L. H., Rasmussen, S. A., Mazure, C., Fleischmann, R. L., Hill, C. L., et al. (1989). The Yale–Brown Obsessive Compulsive Scale: I. Development, use, and reliability. *Archives of General Psychiatry*, *46*, 1006–1011.

Grove, W. M., Andreasen, N. C., Young, M., Endicott, J., Keller, M. B., Hirschfeld, R. M., et al. (1987). Isolation and characterization of a nuclear depressive syndrome. *Psychological Medicine*, *17*, 471–484.

Hamilton, M. (1980). Rating depressive patients. *Journal of Clinical Psychiatry*, *41*(12, Pt. 2), 21–24.

Hankin, B. L., Fraley, R. C., Lahey, B. B., & Waldman, I. D. (2005). Is depression best viewed as a continuum or discrete category?: A taxometric analysis of childhood and adolescent depression in a population-based sample. *Journal of Abnormal Psychology*, *114*, 96–110.

Helzer, J. E., Bucholz, K. K., Bierut, L. J., Regier, D. A., Schuckit, M. A., & Guth, S. E. (2006). Should DSM-V include dimensional diagnostic criteria for alcohol use disorders? *Alcoholism: Clinical and Experimental Research*, *30*, 303–310.

Hempel, C. G. (1961). Introduction to problems of taxonomy. In J. Zubin (Ed.), *Field studies in mental disorders* (pp. 3–22). New York: Grune & Stratton.

Hollander, E. (2005). Obsessive–compulsive disorder and spectrum across the life span. *International Journal of Psychiatry in Clinical Practice*, *9*, 79–86.

Jansson, L. B., & Parnas, J. (2007). Competing definitions of schizophrenia: What can be learned from polydiagnostic studies? *Schizophrenia Bulletin*, *33*, 1178–1200.

Jaspers, K. (1963). *General psychopathology*. Manchester, UK: Manchester University Press. (Original work published 1913)

Jellinek, E. M. (1960). *The disease concept of alcoholism*. New Haven, CT: Hillhouse Press.

Kendell, R. E. (1989). Clinical validity. *Psychological Medicine*, *19*, 45–55.

Kendler, K. S., McGuire, M., Gruenberg, A. M., O'Hare, A., Spellman, M., & Walsh, D. (1993). The Roscommon Family Study: III. Schizophrenia-related personality disorders in relatives. *Archives of General Psychiatry*, *50*, 781–788.

Kraemer, H. C., Noda, A., & O'Hara, R. (2004). Categorical versus dimensional approaches to diagnosis: Methodological challenges. *Journal of Psychiatric Research*, *38*, 17–25.

Krueger, R. F., Nichol, P. E., Hicks, B. M., Markon, K. E., Patrick, C. J., Iacono, W. G., et al. (2004). Using latent trait modeling to conceptualize an alcohol problems continuum. *Psychological Assessment*, *16*, 107–119.

Lara, D. R., Pinto, O., Akiskal, K., & Akiskal, H. S. (2006). Toward an integrative model of the spectrum of mood, behavioral and personality disorders based on fear and anger traits: I. Clinical implications. *Journal of Affective Disorders*, *94*, 67–87.

Lynskey, M. T., Nelson, E. C., Neuman, R. J., Bucholz, K. K., Madden, P. A., Knopik, V. S., et al. (2005). Limitations of DSM-IV operationalizations of alcohol abuse and dependence in a sample of Australian twins. *Twin Research and Human Genetics*, *8*, 574–584.

Marcus, D. K., Lilienfeld, S. O., Edens, J. F., & Poythress, N. G. (2006). Is antisocial personality disorder continuous or categorical?: A taxometric analysis. *Psychological Medicine*, *36*, 1571–1581.

Millon, T. (1987). On the nature of taxonomy in psychopathology. In C. G. Last & M. Hersen (Eds.), *Issues in diagnostic research* (pp. 3–85). New York: Plenum Press.

Mitchell, C. M., & Plunkett, M. (2000). The latent structure of substance use among American Indian adolescents: An example using categorical variables. *American Journal of Community Psychology*, *28*, 105–125.

Myin-Germeys, I., Krabbendam, L., & van Os, J. (2003). Continuity of psychotic symptoms in the community. *Current Opinion in Psychiatry*, *16*, 443–449.

Pedersen, W., & Skrondal, A. (1999). Ecstasy and new patterns of drug use: A normal population study. *Addiction*, *94*, 1695–1706.

Rosenman, S., Korten, A., Medway, J., & Evans, M. (2003). Dimensional vs. categorical diagnosis in psychosis. *Acta Psychiatrica Scandinavica*, *107*, 378–384.

Rothschild, L., Cleland, C., Haslam, N., & Zimmerman, M. (2003). A taxometric study of borderline personality disorder. *Journal of Abnormal Psychology*, *112*, 657–666.

Ruscio, A. M., Borkovec, T. D., & Ruscio, J. (2001). A taxometric investigation of the latent structure of worry. *Journal of Abnormal Psychology*, *110*, 413–422.

Sakashita, C., Slade, T., & Andrews, G. (2007).

Empirical investigation of two assumptions in the diagnosis of DSM-IV major depressive episode. *Australian and New Zealand Journal of Psychiatry*, *41*, 17–23.

Schneider, K. (1962). *Psychopathology* (6th ed.). Stuttgart, Germany: Thieme.

Schwartz, M. A., & Wiggins, O. P. (1987). Diagnosis and ideal types: A contribution to psychiatric classification. *Comprehensive Psychiatry*, *28*, 277–291.

Shankman, S. A., & Klein, D. N. (2002). Dimensional diagnosis of depression: Adding the dimension of course to severity, and comparison to the DSM. *Comprehensive Psychiatry*, *43*, 420–426.

Simonsen, E., & Tyrer, P. (2005). New developments in personality disorder research. In G. N. Christodoulou (Ed.), *Advances in psychiatry* (pp. 67–71). Chêne-Bourg, Switzerland: World Psychiatric Association.

Slade, T., & Andrews, G. (2005). Latent structure of depression in a community sample: A taxometric analysis. *Psychological Medicine*, *35*, 489–497.

Üstün, T. B., & Sartorius, N. E. (1995). *Mental illness in general health care: An international study*. Chichester, UK: Wiley.

van Os, J., Gilvarry, C., Bale, R., van, H. E., Tattan, T., White, I., et al. (1999). A comparison of the utility of dimensional and categorical representations of psychosis: UK700 Group. *Psychological Medicine*, *29*, 595–606.

Widiger, T. A., & Simonsen, E. (2005). Alternative dimensional models of personality disorder: Finding a common ground. *Journal of Personality Disorders*, *19*, 110–130.

Wiles, N. J., Zammit, S., Bebbington, P., Singleton, N., Meltzer, H., & Lewis, G. (2006). Self-reported psychotic symptoms in the general population: Results from the longitudinal study of the British National Psychiatric Morbidity Survey. *British Journal of Psychiatry*, *188*, 519–526.

World Health Organization. (1992). *International statistical classification of diseases and related health problems* (10th rev.). Geneva: Author.

CHAPTER 19

Dimensionalizing Existing Personality Disorder Categories

ANDREW E. SKODOL

Work on DSM-V is well underway. In 1999, a DSM-V Research Planning Conference was held. Twelve DSM-V Research Planning Work Groups were constituted; most have met, producing "white papers" on research needed to inform the revision process. In 2002, *A Research Agenda for DSM-V* was published (Kupfer, First, & Regier, 2002), containing the first series of these papers. In this book, Kupfer and colleagues (2002) argued that the categorical approach to the diagnosis of mental disorders in general, and of personality disorders specifically, needs reexamination. No laboratory marker has been found to be specific for any DSM-defined syndrome. Epidemiological and clinical studies have shown high rates of comorbidity within and across axes, as well as short-term diagnostic instability. And a lack of treatment specificity for individual disorders has been the rule rather than the exception. Thus the question of whether mental disorders (including personality disorders) should be represented by sets of dimensions of psychopathology and other features, rather than by multiple categories, was identified as one of seven basic nomenclature issues needing clarification for DSM-V.

In their chapter of *A Research Agenda for DSM-V*, Rounsaville and colleagues (2002) elaborated: "There is a clear need for dimensional models to be developed and their utility compared with that of existing typologies in one or more limited fields, such as personality. If a dimensional system performs well and is acceptable to clinicians, it might be appropriate to explore dimensional approaches in other domains (e.g., psychotic or mood disorders)" (p. 13). Thus personality disorders have become a "test case" for the return to a dimensional approach to the diagnosis of mental disorders in DSM-V.

In 2004, a DSM-V Research Planning Conference titled "Dimensional Models of Personality Disorder: Etiology, Pathology, Phenomenology, and Treatment" was held. Two special issues of the *Journal of Personality Disorders* were published in 2005, containing the review papers prepared for this conference. The topics reviewed included alternative dimensional models of personality disorders; behavioral and molecular genetic contributions to a dimensional classification; neurobiological dimensional models of personality; developmental perspectives and childhood antecedents; cultural perspectives; the continuity of Axes I and II; coverage and cutoffs for dimensional models; clinical utility; and the problem of severity in personality disorder classification (Widiger & Simonsen, 2005b, 2005c).

Categorical versus Dimensional Models

Considerable research has shown excessive co-occurrence among personality disorders diagnosed by using the DSM categorical system (Oldham, Skodol, Kellman, Hyler, & Rosnick, 1992; Zimmerman, Rothchild, & Chelminski, 2005). In fact, most patients diagnosed with personality disorders meet criteria for more than one. In addition, use of the DSM polythetic criteria, in which a minimum number (e.g., five) from a list of criteria (e.g., nine) are required, but no single one is necessary, results in extreme heterogeneity among patients receiving the same diagnosis. For example, there are 256 possible ways to meet criteria for borderline personality disorder in DSM-IV-TR (Johansen, Karterud, Pedersen, Gude, & Falcum, 2004). Furthermore, all of the personality disorder categories have arbitrary diagnostic thresholds (i.e., the number of criteria necessary for a diagnosis). There are no empirical rationales for setting the boundaries between "pathological" and "normal" personality functioning. Finally despite having criteria for 10 different personality disorder types, the DSM system may still not cover the domain of personality psychopathology adequately. This has been suggested by the observation that the most frequently used personality disorder diagnosis is personality disorder not otherwise specified (PDNOS) (Verheul & Widiger, 2004)—a residual category for patients who are considered to have a personality disorder, but do not meet full criteria for any one of the DSM-IV-TR types, or are judged to have a personality disorder not included in the official classification (e.g., depressive, passive–aggressive, or self-defeating personality disorder).

Dimensional models of personality psychopathology make the co-occurrence of so-called personality disorders and their heterogeneity more rational because they include multiple dimensions that are continua on all of which people can vary. The configurations of dimensional ratings describe each person's profile of personality functioning, so many different multidimensional configurations are possible. Trait dimensional models were developed to describe the full range of personality functioning, so it should be possible to describe anyone.

Dimensional models, however, are unfamiliar to clinicians trained in the medical model of diagnosis, in which a single diagnostic concept is used to communicate a large amount of important clinical information about a patient's problems, the treatment needed, and the likely prognosis. Dimensional models are also more difficult to use; up to 30 dimensions (i.e., the 30 facets of the five-factor model) may be necessary to fully describe a person's personality. Finally, there is little empirical information on the treatment or other clinical implications of dimensional scale elevations—and, in particular, on where to set cutoff points on dimensional scales to maximize their clinical utility. Thus the advantages of either the categorical or the dimensional approach are reciprocals of the other model's disadvantages.

Proposals for Dimensional Models of Personality Disorders

Widiger and Simonsen (2005a) have reviewed 18 alternative proposals for dimensional models of personality disorders. These proposals include (1) dimensional representations of existing personality disorder constructs; (2) dimensional reorganizations of diagnostic criteria; (3) integration of Axes II and I via common psychopathological spectra; and (4) integration of Axis II with dimensional models of general personality structure. Clark (2007) has recently reviewed the implications of several of these approaches for assessing personality disorders, understanding comorbidity between Axis I and Axis II disorders, and reconceptualizing personality disorders in terms of their more stable and less stable elements.

Dimensional Representations of Existing Personality Disorder Constructs

The simplest change in approach to Axis II in the future that would be likely to increase utility would be to rate (revised) personality disorder categories as dimensions. This proposal (option 1 above), therefore, is the focus of the current chapter, although it should not be taken to mean that it will be the approach

recommended by the DSM-V Personality and Personality Disorders Work Group, whose deliberations have only recently begun. It should also not be taken to reflect my own preference, even though I have participated in some of the developmental and empirical work on this model. The Work Group and I continue to consider all proposals and alternatives with open minds.

Criteria-Based Dimensional Representations

There are several ways to transform existing personality disorder categories into dimensions. The most straightforward approach is a simple count of diagnostic criteria, without regard to diagnostic threshold. Counts of criteria have been used in longitudinal studies conducted with nonclinical (Lenzenweger, 2006; Trull, 2001) and general population (Cohen, Crawford, Johnson, & Kasen, 2005) samples, and in genetic studies (Kendler et al., 2006; Reichborn-Kjennerud, Czajkowski, Neale, et al., 2007; Schurhoff et al., 2007) where prevalence rates of full-criteria personality disorders were low, thereby limiting the statistical power of analyses. An approach that has been somewhat more elaborated for clinical use allows for the rating of clinically significant traits and subthreshold disorders, as well as for disorders meeting criteria, and was first proposed over 20 years ago (Kass, Skodol, Charles, Spitzer, & Williams, 1985). Kass et al. (1985) substituted a simple 4-point scale for Axis II personality categories for routine clinical evaluations in a hospital-based outpatient clinic. They found that 51% of patients met full criteria for one or more DSM-III personality disorders, but that this number increased to 88% when patients rated as having "some traits" or "almost meets DSM-III criteria" were noted. Thus it was demonstrated that personality disorder dimensions conveyed more clinically relevant descriptive information about the maladaptive personality traits of patients than did "all-or-nothing" categories.

A further elaboration on the Kass and colleagues (1985) dimensional model was a proposal for standard dimensional representations of personality disorders (Oldham & Skodol, 2000). This proposal converted each DSM-IV personality disorder into a standard 6-point scale ranging from "absent traits" to "prototypic disorder." Significant personality traits and subthreshold disorders could be noted, in addition to full diagnoses of varying degrees of severity (see Table 19.1). This method solved the twin problems posed by the lack of uniformity in the total numbers of diagnostic criteria in the polythetic criteria lists for each DSM-IV personality disorder and different numbers of criteria required to meet each disorder's diagnostic threshold. It also provided convenient and clinically meaningful terms, such as "subthreshold," "pervasive," and "prototypic," to describe personality disorder psychopathology. This schema has been shown to be significantly associated with functional impairment of patients with personality disorders seeking treatment; it has outperformed DSM catego-

TABLE 19.1. A Dimensional Representation of DSM-IV Personality Disorders

Disorder	Absent	Traits	Subthreshold	Disorder (threshold)	Pervasive	Prototypic
Paranoid	0	1 or 2	3	4	5 or 6	7
Schizoid	0	1 or 2	3	4	5 or 6	7
Schizotypal	0	1, 2, or 3	4	5	6, 7, or 8	9
Antisocial	0	1	2	3	4, 5, or 6	7
Borderline	0	1, 2, or 3	4	5	6, 7, or 8	9
Histrionic	0	1, 2, or 3	4	5	6 or 7	8
Narcissistic	0	1, 2, or 3	4	5	6, 7, or 8	9
Avoidant	0	1 or 2	3	4	5 or 6	7
Dependent	0	1, 2, or 3	4	5	6 or 7	8
Obsessive–compulsive	0	1 or 2	3	4	5, 6, or 7	8

ries and other dimensional systems based on diagnostic criteria or on general personality traits (Skodol, Oldham, et al., 2005; see below).

Clinically meaningful dimensions have been identified within personality disorder categories by such methods as factor analysis. These efforts have focused most often on borderline, schizotypal, and antisocial personality disorders. Using longitudinal data, Sanislow and colleagues (2002) confirmed three factors comprising borderline personality disorder criteria, each of which has been viewed by different investigators as a core feature of the disorder: disturbed relatedness (Bender & Skodol, 2007), behavioral dysregulation (Paris et al., 2004), and affective dysregulation (Linehan, 1993). Gunderson, Kolb, and Austin (1981) and later Zanarini, Gunderson, Frankenburg, and Chauncey (1989) developed a semistructured interview called the (Revised) Diagnostic Interview for Borderlines, which measures borderline psychopathology dimensionally in four domains: affective, cognitive, impulsive, and interpersonal. These dimensions have been found to be reliably rated (Zanarini, Frankenburg, & Vujanovic, 2002) and to contain items, particularly in the interpersonal domain, that can discriminate patients with borderline personality disorder from patients with other personality disorders (Zanarini, Gunderson, Frankenburg, & Chauncey, 1990). Morey (1991) developed a Borderline Features Scale as part of his Personality Assessment Inventory. This inventory is a 24-item measure that dimensionally assesses four major features of borderline personality disorder: affective instability, identity problems, negative relationships, and self-harm. Although there is some variability in the content of the major subdimensions of borderline personality disorder in the models represented by these instruments, there is also considerable convergence. If we take borderline personality disorder as an example, it is therefore conceivable that the major domains of personality disorder in DSM could be represented by a limited number of dimensions measuring variability in core components of psychopathology, each reflecting different vulnerabilities, for each disorder (Paris, 2007).

Finally, individual symptoms within diagnoses have been rated along dimensions of severity (e.g., none, mild, moderate, serious, or severe) with clearly defined scale anchor points, particularly when sensitivity to change—for example, with treatment—is the goal (Zanarini, 2003). These ratings can also be summed to derive an overall continuous measure of the severity of borderline or other personality disorder psychopathology.

Dimensional Ratings of Diagnostic Prototypes

Another example of a "person-centered" dimensional approach to existing categories is the prototype-matching approach originally described by Shea, Glass, Pilkonis, Watkins, and Docherty (1987). Embedded in the Personality Assessment Form (PAF) are brief descriptive paragraphs emphasizing salient features of DSM-III personality disorders. Ratings are made by a clinical evaluator on the degree to which the descriptions are characteristic of a person's long-term personality functioning. Ratings are made for each disorder on a 6-point scale ranging from 1 ("not at all") to 6 ("to an extreme degree"). Categorical diagnoses have been retrieved from this dimensional measure by setting a diagnostic threshold of 4 ("to a considerable extent") or higher to represent substantial personality disturbance. Some evidence for convergent and predictive validity for PAF-derived personality disorder ratings has been reported by Pilkonis and Frank (1988) and by Shea and colleagues (1990). In the context of the National Institute of Mental Health (NIMH) Treatment of Depression Collaborative Research Program, the factor structures of the clinician-rated PAF and an extensive self-report battery of personality traits were similar (Pilkonis & Frank, 1988). Patients with personality disorders had significantly worse outcomes in social functioning, and were more likely to have residual symptoms of depression, than patients without personality disorders (Shea et al., 1990). The PAF has been used as an independent clinician-rated confirmation of a semistructured interview diagnosis of a personality disorder at intake in the Collaborative Longitudinal Personality Disorders Study (CLPS; Gunderson et al., 2000; see later discussion).

A prototype dimensional model has subsequently been empirically derived and

elaborated by Shedler and Westen (Shedler & Westen, 2004; Westen, Shedler, & Bradley, 2006). The SWAP prototype-matching model is a syndromal approach based on research using the Shedler–Westen Assessment Procedure–200 (SWAP-200). Twelve personality syndromes were identified from a large national sample of patients who were rated by clinicians using the SWAP-200 (Shedler & Westen, 2004; Westen & Shedler, 1999a, 1999b). Each syndrome was then represented by a paragraph-length prototype description representing the syndrome in its "pure" form. Using this system, a clinician compares a patient to the description of the prototypic patient with each disorder, and the "match" is rated on a 5-point scale from 5 ("very good match") to 1 ("little or no match"). Prototype ratings have been demonstrated to have good inter-rater reliability. They have also been shown to be easy to use and have been judged by psychiatrists and psychologists to be clinically useful (Spitzer, First, Shedler, Westen, & Skodol, 2008; see also below).

Use of Dimensional Representations in Research on Utility

First (2005) has argued that efforts to demonstrate the clinical utility of dimensional approaches to personality disorder diagnosis should be a prerequisite for their implementation. The "clinical utility" of a diagnosis has been defined by Kendell and Jablensky (2003) as its ability to provide "nontrivial information about prognosis and likely treatment outcomes, and/or testable propositions about biological and social correlates" (p. 9). Included in their definition would be information about clinical course, associated disability, treatment selection, prevalence, demographics, family history, premorbid characteristics, and etiology. One measure of the utility of dimensional representations of existing personality disorder categories has been their use in research on such clinically relevant variables, particularly in settings where full-criteria personality disorders are relatively uncommon (such as general population or genetic studies), and statistical power is consequently limited. Below, I review a selection of studies that have made use of dimensional representations.

The Children in the Community Study

The Children in the Community Study (CICS) is a longitudinal study of a sample of approximately 800 children, who were originally recruited (with their mothers) in upstate New York in 1975, when they were between 1 and 10 years of age (Cohen, Crawford, Johnson, & Kasen, 2005). They have been followed now periodically for over 30 years. Originally, the study was designed to assess the level of need for children's services in the community. At the first follow-up in 1983, the focus of the study shifted to predictors of Axis I disorders in early adolescence, but an interest in the development of personality disorders in this age group also existed. Using various methods, the researchers have assessed personality disorders in this sample four times to date: in 1983, when the children were at mean age 14; between 1985 and 1986, when they were at mean age 16; between 1991 and 1993, at mean age 22, and between 2001 and 2004, at mean age 33.

Course of Personality Disorder Symptoms

Given the prevalence of personality disorders in the general population, too few adolescents in the CICS met criteria for these disorders to allow the researchers to obtain reliable stability estimates. Therefore, the CICS examined the stability of personality disorder traits and found that levels decreased by 48% between adolescence (ages 14–16) and early adulthood (age 22) (Johnson, Cohen, Kasen, et al., 2000).

Impact on Psychosocial Functioning

The impact of personality disorder psychopathology on functioning has been examined in the CICS for each DSM personality disorder cluster. For Cluster A (the odd, eccentric cluster), adolescents with high symptom levels had lower education and achievement (Cohen, Chen, et al., 2005), greater partner conflict, and earlier childbearing (Chen et al., 2004) in early adulthood. Adolescents with high levels of Cluster B (dramatic, er-

ratic) symptoms had lower levels of intimacy (Crawford, Cohen, Johnson, Sneed, & Brook, 2004) and sustained conflict with partners (Chen et al., 2004) in early adulthood. Adolescents with high levels of Cluster C (anxious, fearful) symptoms had greater conflict with partners, if they had a partner (Chen et al., 2004). Adolescents and young adults who qualified for a diagnosis of PDNOS experienced significant educational failure and interpersonal difficulties (Johnson, First, et al., 2005).

Impact on Behavior

High levels of Cluster A symptoms during adolescence predicted subsequent violent acts and criminal behavior (Johnson, Cohen, Smailes, et al., 2000); high levels of Cluster B symptoms predicted violent behavior. In Cluster C, adolescent dependent symptoms predicted suicidality (Johnson et al., 1999). Adolescents with PDNOS were also at risk for serious acts of aggression as young adults (Johnson, First, et al., 2005). Young adults' personality disorder symptoms in all three clusters partially mediated violence against partners (Johnson, Cohen, Smailes, et al., 2000).

Impact on Quality of Life

Any personality disorder or any Cluster A, B, or C personality disorder in early adulthood (age 22) was associated with reduced quality of life at age 33 (Chen et al., 2006). Cluster B personality disorders had the greatest effect. Antisocial, borderline, and schizotypal symptoms were independently associated with quality-of-life reduction. These effects were independent of demographic characteristics, co-occurring Axis I disorders, and physical illnesses.

Impact on Axis I Disorders

In Cluster A personality disorders, adolescent or young adult symptoms increased risk of subsequent mood, eating, anxiety, and disruptive behavior disorders. Adolescent or young adult Cluster B symptoms increased risk of subsequent mood, anxiety, eating, disruptive, *and* substance use disorders. Cluster C symptoms increased risk of subsequent mood, anxiety, and disruptive behavior, but not eating or substance use, disorders (Johnson et al., 1999; Johnson, Cohen, Kasen, & Brook, 2005, 2006a, 2006b).

The Longitudinal Study of Personality Disorders

The Longitudinal Study of Personality Disorders (LSPD; Lenzenweger, 2006) is a multiwave, longitudinal study of Cornell University undergraduates. As such, it represents a study of personality psychopathology in individuals who were not identified patients (i.e., not seeking treatment). Of 1,684 eligible undergraduates, 258 were selected by screening and follow-up interviews. Of this group, 134 were identified as having probable personality disorders, and the other 124 were deemed to have no personality disorder. These participants were then followed three times over a 4-year interval.

Course of Personality Disorder Symptoms

As in the CICS, because of the insufficient number of individual personality disorders at the categorical (threshold) level to allow analysis of the stability of disorders, personality disorder symptoms were examined in the LSPD as continuous dimensions. Personality disorder dimensions showed significant levels of stability, by both interview and self-report (Lenzenweger, 1999). However, personality disorder features in this study also showed significant declines over time: Personality psychopathology decreased by 1.4 features per year over 4 years (Lenzenweger, Johnson, & Willett, 2004). Psychosocial functioning has not been measured in the LSPD to date, although its measurement is anticipated in future follow-along waves.

Norwegian Twin Study

The Axis I and Axis II Psychiatric Disorders in Norwegian Twins Study is the first and only study to date that has included structured interview data on both DSM-IV Axis I and Axis II psychiatric disorders in a population-based sample with genetically informative data (including DNA). Among the main aims of this study are to investigate the influence of genetic and environmental risk factors on DSM-IV personality disorders and their comorbidity (Czajkowski et

al., 2008; Kendler et al., 2006; Ørstavik, Kendler, Czajkowski, Tambs, & Reichborn-Kjennerud, 2007a; Reichborn-Kjennerud, Czajkowski, Neale, et al., 2007) and the comorbidity between Axis I and Axis II disorders (Ørstavik, Kendler, Czajkowski, Tambs, & Reichborn-Kjennerud, 2007b; Reichborn-Kjennerud, Czajkowski, Torgersen, et al., 2007).

Relationships among Personality Disorders

Kendler and colleagues (2006) examined the relationship between the genetic and environmental risk factors for dimensional representations (based on counts of criteria met on a semistructured interview at the level of at least subthreshold presence) of the DSM-IV Cluster A (odd, eccentric) personality disorders in 1,386 young adult twin pairs from the Norwegian Institute of Public Health Twin Panel. Importantly, prior to utilizing their dimensional representations of personality disorders in genetic analyses, Kendler and colleagues tested whether the four response options (i.e., "absent," "subthreshold," "present," and "strongly present") in their measure of each DSM-IV personality disorder criterion reflected levels of severity on a single underlying, normally distributed continuum of liability. In virtually all cases of criteria and combinations of criteria, the assumption was supported, justifying the use of dimensional representations of personality disorders to investigate genetic and environmental contributions to the liability for the disorders in the population. In this study, total heritability for these personality disorders was modest (ranging from 21% to 28%), and all shared a portion of their genetic and environmental risk factors. Schizotypal personality disorder had higher common genetic and unique environmental factors than did either paranoid or schizoid personality disorder, and most closely reflected the genetic and environmental liability common to the three Cluster A disorders.

A similar study was conducted for dimensional representations of Cluster C personality disorders (Reichborn-Kjennerud, Czajkowski, Neale, et al., 2007). Heritability ranged from 27% to 35%. Common genetic and environmental factors accounted for 54% and 64%, respectively, of the variance in avoidant personality disorder and dependent personality disorder, but only 11% of the variance in obsessive–compulsive personality disorder, which appeared to be etiologically distinct. The results did not support the validity of the Cluster C construct in its present form.

Relationships between Personality Disorders and Axis I Disorders

The relationships between dimensional representations of depressive personality disorder and major depressive disorder (Ørstavik et al., 2007a), and between avoidant personality disorder and social phobia (Reichborn-Kjennerud, Czajkowski, Torgersen, et al., 2007), were studied by estimating the extent to which the pairs of disorders were influenced by common genetic and shared or unique environmental factors versus the extent to which these risk factors were specific to each disorder. Although depressive personality disorder and major depressive disorder shared a substantial proportion of genetic and environmental risk factors, the disorders appeared to be distinct, with overlapping but not identical etiologies. A common genetic vulnerability was found for avoidant personality disorder and social phobia, and individuals with high genetic liability would develop one disorder or the other entirely on the basis of environmental risk factors unique to each disorder. These results were consistent with the hypothesis that common psychobiological dimensions underlie certain Axis I and Axis II disorders.

Comparisons of Alternative Models

It remains to be seen how alternative models of personality psychopathology compare on important aspects of clinical utility to personality disorders and their dimensional representations. Testing the associations of alternative models with a variety of antecedent (e.g., abuse, positive child experiences), concurrent (e.g., functioning, treatment utilization), and predictive (e.g., future functioning, course of Axis I disorders) validators has been the subject of ongoing studies in the CLPS, mentioned earlier in this chapter.

The CLPS (Skodol, Gunderson, et al., 2005) is a multisite, NIMH-funded lon-

gitudinal study of the natural course of personality disorders. Participating sites include Brown, Columbia (now in collaboration with the Sunbelt Collaborative and the University of Arizona), Harvard, Yale, and Texas A&M Universities. The aims of the CLPS have been to determine the stability of personality disorder diagnoses and criteria, personality traits, and functional impairment, and to determine the predictors of clinical course. The original CLPS sample recruited 668 treatment-seeking or recently treated patients who were diagnosed with one of four DSM-IV personality disorders (schizotypal, borderline, avoidant, or obsessive–compulsive), or with major depressive disorder and no personality disorder. This original sample was supplemented with the recruitment of 65 additional minority patients to ensure adequate power to test differences among white, African American, and Hispanic patients with the four personality disorders on various outcomes. The original CLPS sample has now completed its 10th year of follow-up.

Within the first 2 years of follow-up, between 33% (schizotypal) and 55% (obsessive–compulsive) of patients with personality disorders experienced a period of remission according to the 2-month standard (Grilo et al., 2004). Between 23% (schizotypal) and 38% (obsessive–compulsive) experienced a 12-month remission. In addition, on retest at 2 years by evaluators unaware of the original diagnoses, between 50% and 60% were below the threshold for a personality disorder diagnosis. The mean proportion of criteria met declined significantly for each of the personality disorders. These patterns of criteria decline and rates of remission continued over the first 6 years of follow-up, such that by year 6, over three-fourths of patients with personality disorders had had a 2-month remission, and over two-thirds had had a 12-month remission (Skodol, 2008). Relapses also occurred, however, over the first 6 years. Relapse rates varied by personality disorder diagnosis: Schizotypal personality disorder had the lowest relapse rates, and avoidant personality disorder had the highest relapse rates.

When viewed as continuous dimensions, counts of the number of criteria met correlated with baseline counts .74 at 6 months, .67 at 1 year, and .59 at 2 years (Grilo et al., 2004). These correlations are very similar to correlations of personality traits across age categories represented in the CLPS (18–45 years) as reported in a meta-analysis of 152 longitudinal studies by Roberts and DelVecchio (2000).

Studies using dimensional representations of DSM-IV personality disorders have been conducted in the CLPS. An initial study (Skodol, Oldham, et al., 2005) compared DSM-IV personality disorder categories, dimensional representations (the 6-point continuous scales described above, based on number of criteria met) of personality disorders, the five-factor model, and a three-factor model (positive affectivity, negative affectivity, disinhibition) derived from the Schedule for Nonadaptive and Adaptive Personality (SNAP) on their associations to both interviewer-rated and self-reported domains of functional impairment. The DSM-IV dimensional representations had the strongest associations to employment, social, leisure, and global functioning.

A more elaborate study was undertaken by Morey and colleagues (2007). In this study, multiple antecedent, concurrent, and predictive markers of construct validity were examined for three major models of personality disorder: the five-factor model (five factors and 30 facets), the 15-trait SNAP model, and the DSM-IV personality disorders (categories and criteria counts). Antecedent validity markers included various types of childhood abuse and neglect, positive childhood experiences reflecting resiliency, past history of medication use, and past history of psychiatric hospitalization. Concurrent markers included psychosocial functioning in multiple domains, co-occurring Axis I disorders, and current medications. Functioning, Axis I disorders, suicide attempts, hospitalizations, and medication use over 2 years and 4 years of follow-up were the predicted outcome variables. All models showed substantial validity across marker variables over time. Dimensional models, especially the dimensionalized DSM-IV personality disorders and the SNAP model, consistently outperformed DSM-IV personality disorder categories and the FFM in predicting external validators. The SNAP model, which incorporates both normal and abnormal personality dimensions, seemed best: It not only captured variables of clinical significance at

baseline, when most subjects were seeking treatment, but also maintained its predictive power over time better than the DSM-IV dimensions. The data demonstrated the importance of both stable trait and dynamic psychopathological influences in predicting external criteria over time.

Clinician Acceptability of Dimensional Models

As noted earlier, some dimensional models of personality psychopathology can appear complex and unfamiliar to clinicians used to working with diagnostic categories. Spitzer and colleagues (2008) conducted a study of the clinical relevance and utility of five dimensional systems for personality disorders that have been proposed for DSM-V: (1) a criteria-counting model based on current DSM-IV diagnostic criteria, (2) a prototype-matching model based on current DSM-IV diagnostic criteria, (3) a prototype-matching model based on the SWAP-200, (4) the five-factor model, and (5) Cloninger's psychobiological model. A random national sample of psychiatrists and psychologists applied all five systems to a patient under their care and rated the clinical utility of each system. The two prototype-matching models were judged most clinically useful and relevant. The two trait-based models were judged least useful. The authors concluded that prototype-matching systems most faithfully capture personality syndromes seen in practice and allow for rich descriptions without a proportionate increase in time or effort.

Conclusions

The simplest change in approach to Axis II in the future that would be likely to increase utility would be to rate (revised) personality disorder categories as dimensions. In this chapter, I have reviewed several ways to transform existing personality disorder categories into dimensions. The most straightforward approach is a simple count of diagnostic criteria, without regard to diagnostic threshold. Another example of a dimensional approach to existing categories is the prototype-matching approach, in which a clinician compares a patient to a description of a prototypic patient with each personality disorder, and the degree of the "match" is rated. As noted earlier in this chapter, one measure of the utility of dimensional representations of existing personality disorder categories has been their use in research, particularly in settings where full-criteria personality disorders are relatively uncommon (such as general population or genetic studies), and statistical power is therefore limited. Several examples of the use of dimensional representations in research studies have been given.

Also as noted earlier, although the dimensionalization of existing categories is the focus of the current chapter, it should not be taken to mean that it will be the approach recommended by the DSM-V Personality and Personality Disorders Work Group, whose deliberations have only recently begun. At this writing, the Work Group and I are still considering all proposals and alternatives with open minds.

References

Bender, D. S., & Skodol, A. E. (2007). Borderline personality as a self–other representational disturbance. *Journal of Personality Disorders*, *21*, 500–517.

Chen, H., Cohen, P., Crawford, T. N., Kasen, S., Johnson, J. G., & Berenson, K. (2006). Relative impact of young adult personality disorders on subsequent quality of life: Findings of a community-based longitudinal study. *Journal of Personality Disorders*, *20*, 510–523.

Chen, H., Cohen, P., Johnson, J. G., Kasen, S., Sneed, J. R., & Crawford, T. N. (2004). Adolescent personality disorder and conflict with romantic partners during the transition to adulthood. *Journal of Personality Disorders*, *18*, 507–525.

Clark, L. A. (2007). Assessment and diagnosis of personality disorder: Perennial issues and an emerging reconceptualization. *Annual Review of Psychology*, *58*, 227–257.

Cohen, P., Chen, H., Kasen, S., Johnson, J. G., Crawford, T. N., & Gordon, K. (2005). Adolescent Cluster A personality disorder symptoms, role assumption in the transition to adulthood, and resolution or persistence of symptoms. *Development and Psychopathology*, *17*, 549–568.

Cohen, P., Crawford, T. N., Johnson, J. G., & Kasen, S. (2005). The Children in the Com-

munity Study of developmental course of personality disorder. *Journal of Personality Disorders*, *19*, 466–486.

Crawford, T. N., Cohen, P., Johnson, J. G., Sneed, J. R., & Brook, J. S. (2004). The course and psychosocial correlates of personality disorder symptoms in adolescence: Erikson's developmental theory revisited. *Journal of Youth and Adolescence*, *33*, 373–387.

Czajkowski, N., Kendler, K. S., Jacobson, K. C., Tambs, K., Roysamb, E., & Reichborn-Kjennerud, T. (2008). Passive–aggressive (negativistic) personality disorder: A population-based twin study. *Journal of Personality Disorders*, *22*, 109–122.

First, M. B. (2005). Clinical utility: A prerequisite for the adoption of a dimensional approach in DSM. *Journal of Abnormal Psychology*, *114*, 560–564.

Grilo, C. M., Shea, M. T., Sanislow, C. A., Skodol, A. E., Gunderson, J. G., Stout, R. L., et al. (2004). Two-year stability and change in schizotypal, borderline, avoidant and obsessive–compulsive personality disorders. *Journal of Consulting and Clinical Psychology*, *72*, 767–775.

Gunderson, J. G., Kolb, J. E., & Austin, V. (1981). The Diagnostic Interview for Borderline Patients. *American Journal of Psychiatry*, *138*, 896–903.

Gunderson, J. G., Shea, M. T., Skodol, A. E., McGlashan, T. H., Morey, L. C., Stout, R. L., et al. (2000). The Collaborative Longitudinal Personality Disorders Study: Development, aims, design, and sample characteristics. *Journal of Personality Disorders*, *14*, 300–315.

Johansen, M., Karterud, S., Pedersen, G., Gude, T., & Falkum, E. (2004). An investigation of the prototype validity of the borderline DSM-IV construct. *Acta Psychiatrica Scandinavica*, *109*, 289–298.

Johnson, J. G., Cohen, P., Kasen, S., & Brook, J. S. (2005). Personality disorder traits associated with risk for unipolar depression during middle adulthood. *Psychiatry Research*, *136*, 113–121.

Johnson, J. G., Cohen, P., Kasen, S., & Brook, J. S. (2006a). Personality disorders evident by early adulthood and risk for eating and weight problems during middle adulthood. *International Journal of Eating Disorders*, *39*, 184–192.

Johnson, J. G., Cohen, P., Kasen, S., & Brook, J. S. (2006b). Personality disorder traits evident by early adulthood and risk for anxiety disorders during middle adulthood. *Journal of Anxiety Disorders*, *20*, 408–426.

Johnson, J. G., Cohen, P., Kasen, S., Skodol, A. E., Hamagami, F., & Brook, J. S. (2000). Age-related change in personality disorder trait levels between early adolescence and adulthood: A community-based longitudinal investigation. *Acta Psychiatrica Scandinavica*, *102*, 265–275.

Johnson, J. G., Cohen, P., Skodol, A. E., Oldham, J. M., Kasen, S., & Brook, J. (1999). Personality disorders in adolescence and risk of major mental disorders and suicidality during adulthood. *Archives of General Psychiatry*, *56*, 805–811.

Johnson, J. G., Cohen, P., Smailes, E., Kasen, S., Oldham, J. M., Skodol, A. E., et al. (2000). Adolescent personality disorders associated with violence and criminal behavior during adolescence and early adulthood. *American Journal of Psychiatry*, *157*, 1406–1412.

Johnson, J. G., First, M. B., Cohen, P., Skodol, A. E., Kasen, S., & Brook, J. S. (2005). Adverse outcomes associated with personality disorder not otherwise specified (PDNOS) in a community sample. *American Journal of Psychiatry*, *162*, 1926–1932.

Kass, F., Skodol, A. E., Charles, E., Spitzer, R. L., & Williams, J. B. (1985). Scaled ratings of DSM-III personality disorders. *American Journal of Psychiatry*, *142*, 627–630.

Kendell, R., & Jablensky, A. (2003). Distinguishing between the validity and utility of psychiatric diagnoses. *American Journal of Psychiatry*, *160*, 4–12.

Kendler, K. S., Czajkowski, N., Tambs, K., Torgersen, S., Aggen, S. H., Neale, M. C., et al. (2006). Dimensional representations of DSM-IV Cluster A personality disorders in a population-based sample of Norwegian twins: A multivariate study. *Psychological Medicine*, *36*, 1583–1591.

Kupfer, D. J., First, M. B., & Regier, D. E. (Eds.). (2002). *A research agenda for DSM-V*. Washington, DC: American Psychiatric Association.

Lenzenweger, M. F. (1999). Stability and change in personality disorder features. *Archives of General Psychiatry*, *56*, 1009–1015.

Lenzenweger, M. F. (2006). The Longitudinal Study of Personality Disorders: History, design considerations, and initial findings. *Journal of Personality Disorders*, *20*, 645–670.

Lenzenweger, M. R., Johnson, M. D., & Willett, J. B. (2004). Individual growth curve analysis

illuminates stability and change in personality disorder features: The Longitudinal Study of Personality Disorders. *Archives of General Psychiatry*, *61*, 1015–1024.

Linehan, M. M. (1993). *Cognitive-behavioral treatment of borderline personality disorder.* New York: Guilford Press.

Morey, L. C. (1991). *Personality Assessment Inventory: Professional manual.* Odessa, FL: Psychological Assessment Resources.

Morey, L. C., Hopwood, C. J., Gunderson, J. G., Skodol, A. E., Shea, M. T., Yen, S., et al. (2007). Comparison of alternative models for personality disorders. *Psychological Medicine*, *37*, 983–994.

Oldham, J. M., & Skodol, A. E. (2000). Charting the future of Axis II. *Journal of Personality Disorders*, *14*, 17–29.

Oldham, J. M., Skodol, A. E., Kellman, H. D., Hyler, S. E., & Rosnick, L. (1992). Diagnosis of DSM-III-R personality disorders by two structured interviews: Patterns of comorbidity. *American Journal of Psychiatry*, *149*, 213–220.

Ørstavik, R. E., Kendler, K. S., Czajkowski, N., Tambs, K., & Reichborn-Kjennerud, T. (2007a). Genetic and environmental contributions to depressive personality disorder in a population-based sample of Norwegian twins. *Journal of Affective Disorders*, *99*, 181–189.

Ørstavik, R. E., Kendler, K. S., Czajkowski, N., Tambs, K., & Reichborn-Kjennerud, T. (2007b). The relationship between depressive personality disorder and major depressive disorder: A population-based twin study. *American Journal of Psychiatry*, *164*, 1866–1872.

Paris, J. (2007). The nature of borderline personality disorder: Multiple dimensions, multiple symptoms, but one category. *Journal of Personality Disorders*, *21*, 457–473.

Paris, J., Zweig-Frank, H., Ng, F., Schwartz, G., Steiger, H., & Nair, V. (2004). Neurobiological correlates of diagnosis and underlying traits in patients with borderline personality disorder compared with normal controls. *Psychiatry Research*, *121*, 239–252.

Pilkonis, P. A., & Frank, E. (1988). Personality pathology in recurrent depression: Nature, prevalence, and relationship to treatment response. *American Journal of Psychiatry*, *145*, 435–441.

Reichborn-Kjennerud, T., Czajkowski, N., Neale, M. C., Ørstavik, R. E., Torgersen, S., Tambs, K., et al. (2007). Genetic and environmental influences on dimensional representations of DSM-IV Cluster C personality disorders: A population-based multivariate twin study. *Psychological Medicine*, *37*, 645–653.

Reichborn-Kjennerud, T., Czajkowski, N., Torgersen, S., Neale, M. C., Ørstavik, R. E., Tambs, K., et al. (2007). The relationship between avoidant personality disorder and social phobia: A population-based twin study. *American Journal of Psychiatry*, *164*, 1722–1728.

Roberts, B. W., & DelVecchio, W. F. (2000). The rank-order consistency of personality traits from childhood to old age: A quantitative review of longitudinal studies. *Psychological Bulletin*, *126*, 3–25.

Rounsaville, B. J., Alarcon, R. D., Andrews, G., Jackson, J., Kendell, R., & Kendler, K. S. (2002). Basic nomenclature issues for DSM-V. In D. J. Kupfer, M. B. First, & D. E. Regier (Eds.), *A research agenda for DSM-V* (pp. 1–29). Washington, DC: American Psychiatric Association.

Sanislow, C. A., Grilo, C. M., Morey, L. C., Bender, D. S., Skodol, A. E., Gunderson, J. G., et al. (2002). Confirmatory factor analysis of DSM-IV criteria for borderline personality disorder: Findings from the Collaborative Longitudinal Personality Disorders Study. *American Journal of Psychiatry*, *159*, 284–290.

Schurhoff, F., Szoke, A., Chevalier, F., Roy, I., Meary, A., Bellivier, F., et al. (2007). Schizotypal dimensions: An intermediate phenotype associated with the COMT high activity allele. *American Journal of Medical Genetics*, *144*, 64–68.

Shea, M. T., Glass, D. R., Pilkonis, P. A., Watkins, J., & Docherty, J. P. (1987). Frequency and implications of personality disorders in a sample of depressed inpatients. *Journal of Personality Disorders*, *1*, 27–42.

Shea, M. T., Pilkonis, P. A., Beckham, E., Collins, J. F., Elkin, I., Sotsky, S. M., et al. (1990). Personality disorders and treatment outcome in the NIMH Treatment of Depression Collaborative Research Program. *American Journal of Psychiatry*, *147*, 711–718.

Shedler, J., & Westen, D. (2004). Refining personality disorder diagnosis: Integrating science and practice. *American Journal of Psychiatry*, *161*, 1350–1365.

Skodol, A. E. (2008). Longitudinal course and outcome of personality disorders. *Psychiatric Clinics of North America*, *31*, 495–503.

Skodol, A. E., Gunderson, J. G., Shea, M. T., McGlashan, T. H., Morey, L. C., Sanislow, C.

A., et al. (2005). The Collaborative Longitudinal Personality Disorders Study (CLPS): Overview and implications. *Journal of Personality Disorders*, *19*, 487–504.

Skodol, A. E., Oldham, J. M., Bender, D. S., Dyck, I. R., Stout, R. L., Morey, L. C., et al. (2005). Dimensional representations of DSM-IV personality disorders: Relationships to functional impairment. *American Journal of Psychiatry*, *162*, 1919–1925.

Spitzer, R. L., First, M. B., Shedler, J., Westen, D., & Skodol, A. E. (2008). Clinical utility of five dimensional systems for personality diagnosis: A "consumer preference" study. *Journal of Nervous and Mental Disease*, *196*, 356–374.

Trull, T. J. (2001). Structural relations between borderline personality disorder features and putative etiologic correlates. *Journal of Abnormal Psychology*, *110*, 471–481.

Verheul, R., & Widiger, T. A. (2004). A meta-analysis of the prevalence and usage of personality disorder not otherwise specified (PDNOS). *Journal of Personality Disorders*, *18*, 309–319.

Westen, D., & Shedler, J. (1999a). Revising and assessing Axis II: Part I. Developing a clinically and empirically valid assessment method. *American Journal of Psychiatry*, *156*, 258–272.

Westen, D., & Shedler, J. (1999b). Revising and assessing Axis II: Part II. Toward an empirically-based and clinically useful classification of personality disorders. *American Journal of Psychiatry*, 156, 273–285.

Westen, D., Shedler, J., & Bradley, R. (2006). A prototype approach to personality disorder diagnosis. *American Journal of Psychiatry*, *163*, 846–856.

Widiger, T. A., & Simonsen, E. (2005a). Alternative dimensional models of personality disorder: Finding a common ground. *Journal of Personality Disorders*, *19*, 110–130.

Widiger, T. A., & Simonsen, E. (2005b). Introduction to part two of the special section on the research agenda for the development of a dimensional classification of personality disorder. *Journal of Personality Disorders*, *19*, 211.

Widiger, T. A., & Simonsen, E. (2005c). Introduction to the special section: The American Psychiatric Association's research agenda for the DSM-V. *Journal of Personality Disorders*, *19*, 103–109.

Zanarini, M. C. (2003). Zanarini Rating Scale for Borderline Personality Disorder (ZAN-BPD): A continuous measure of DSM-IV borderline psychopathology. *Journal of Personality Disorders*, *17*, 233–242.

Zanarini, M. C., Frankenburg, F. R., & Vujanovic, A. A. (2002). Inter-rater and test-retest reliability of the Revised Diagnostic Interview for Borderlines. *Journal of Personality Disorders*, *16*, 270–276.

Zanarini, M. C., Gunderson, J. G., Frankenburg, F. R., & Chauncey, D. L. (1989). The Revised Diagnostic Interview for Borderlines: Discriminating BPD from other Axis II disorders. *Journal of Personality Disorders*, *3*, 10–18.

Zanarini, M. C., Gunderson, J. G., Frankenburg, F. R., & Chauncey, D. L. (1990). Discriminating borderline personality disorder from other Axis II disorders. *American Journal of Psychiatry*, *147*, 161–167.

Zimmerman, M., Rothchild, L., & Chelminski, I. (2005). The prevalence of DSM-IV personality disorders in psychiatric outpatients. *American Journal of Psychiatry*, *162*, 1911–1918.

CHAPTER 20

An Empirically Based Prototype Diagnostic System for DSM-V and ICD-11

KILE M. ORTIGO
BEKH BRADLEY
DREW WESTEN

Researchers and clinicians from a variety of theoretical and professional perspectives have written much about the strengths and weaknesses of the current polythetic or count/cutoff method of psychiatric diagnosis used in the *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition, and its text revision (DSM-IV-TR; American Psychiatric Association [APA], 2000). Although the changes initiated with DSM-III and DSM-III-R (APA, 1980, 1987) to move toward a more behaviorally descriptive and systematic diagnostic system have proved beneficial in many ways, the diagnostic system has been criticized for a number of shortcomings—including a lack of theoretical and empirical foundation, arbitrary symptom cutoffs and time frames, and unclear clinical utility for many differential diagnoses (see Andersson & Ghaderi, 2006; Beutler & Malik, 2002; Schmidt, Kotov, & Joiner, 2004).

Another problem with DSM-IV(-TR) diagnosis involves mixed, not otherwise specified (NOS), and subthreshold diagnoses. Numerous studies have documented the prevalence of subthreshold diagnoses among the anxiety and mood disorders (e.g., Olfson, Weissman, Leon, Farber, & Sheehan, 1996; Zinbarg et al., 1994), which are unintended by-products of taxonomic refinements since DSM-III. A major advantage of DSM-III and its successors is the increased reliability of diagnosis made possible by operationalizable criteria and structured interviews (Feighner et al., 1972; Spitzer, Endicott, & Robins, 1978). However, refinement of virtually every Axis I category has brought with it identification of "border" cases that require new diagnoses because strict adherence to a set of specific diagnostic algorithms inherently leads to nondiagnosis of subclinical or border syndromes.

A related problem is comorbidity. Along with the more systematic delineation of categories and criteria since DSM-III was published, there has come a virtual explosion of research on comorbidity. When, or to what extent, this research represents incremental knowledge about psychopathology is difficult to discern. The comorbidity of anxiety and mood disorders provides a good example (e.g., Kessler et al., 1996) because it probably reflects in part "the nature

of things" (i.e., the fact that the broad-band personality trait of negative affect is a diathesis for both sets of disorders; see, e.g., Barlow, 2002) as well as criterion overlap. One attempted resolution is to create mixed diagnoses, such as the mixed anxiety–depressive disorder included in a DSM-IV appendix as a diagnosis needing further study. Unfortunately, each similar addition to the DSM creates new and different subthreshold cases, which in turn require further specification (see Zinbarg et al., 1994).

Despite these criticisms of the current diagnostic procedures, the majority of research and work directed at refining successive editions of the DSM has focused not on the process through which disorders are diagnosed, but instead on which diagnostic categories should be included, excluded, or modified (e.g., Lichtenthal, Cruess, & Prigerson, 2004; Mayou, Kirmayer, Simon, Kroenke, & Sharpe, 2005) and/or which specific criteria should be modified or added with respect to any given diagnostic category (e.g., Denton, 2007; Martin, Chung, & Langenbucher, 2008).

One exception has been a body of research established over the last several decades making a case for a dimensionalized approach to psychiatric diagnoses. Specifically, across disorders, researchers are increasingly calling for dimensional diagnosis, either as the primary method of diagnosis or as a secondary way of summarizing diagnostic information (Westen et al., 2002; Widiger & Clark, 1999). Proponents point out that a dimensionalized approach to diagnosis has the potential to address a number of problems inherent in a categorical diagnosis. Calls for dimensional diagnosis of Axis I disorders have extended from mood and anxiety disorders (e.g., Brown, Chorpita, & Barlow, 1998; Krueger & Finger, 2001; Krueger et al., 2002; Widiger & Clark, 1999) to schizophrenia and the other psychotic disorders (e.g., Lenzenweger, 1997; Tsuang, Stone, & Faraone, 2000; Van der Does, Linszen, Dingemans, Nugter, & Scholte, 1993). For example, Appendix B of the DSM-IV outlines a dimensional approach to diagnosis of psychosis, in which clinicians would rate the extent to which the patient has positive symptoms, disorganized symptoms, and negative symptoms, using a 4-point severity scale (from "absent" to "severe").

As another example, most treatment research on depression uses a diagnosis of major depressive disorder as the primary inclusion criterion, but then largely relies on dimensional measures to assess outcome because patients who fall just below the diagnostic threshold may not show clinically significant or lasting change. In classification research, researchers similarly rely almost exclusively on dimensional variables because they provide greater statistical power, tend to be truer to the underlying distributions in the population, and are more useful in data-analytic procedures.

Despite these benefits, dimensional approaches to diagnosis also have drawbacks in practical utility that mitigate their unreserved adoption as a diagnostic approach. As currently conceptualized, dimensionalized approaches to diagnosis lack a quick, parsimonious way to code disorders as present or absent, which limits their utility in medical, clinical, and insurance settings (First, 2005). Moreover, the plurality of dimensional models and lack of consensus hinder the ability to choose how and what should be dimensionalized (Frances, 1993). In this chapter, we propose a prototype-based approach to diagnosis as an alternative approach that combines the strengths of both categorical and dimensional diagnostic systems (Westen & Shedler, 2000; Westen et al., 2002; Westen, Shedler, & Bradley, 2006).

Prototypes and Psychiatric Diagnosis

The development of a clinically effective approach to diagnosis requires an understanding of how people make judgments about the degree of similarity of one case (for our purposes, a psychiatric patient) to an abstract construct (for our purposes, a psychiatric diagnostic category). The current diagnostic system began under the assumption that such decisions are made according to what has been referred to as the "classical" view of decision making (Folstein & Van Petten, 2004; Medin, 1989). The classical view argues that categorizations are made via strict adherence to well-defined rules of membership. For example, in biology, species are classified as mammals if they are vertebrates, have mammary glands, have hair, and give

birth to live young. For psychiatric classifications, what became clear not long after implementation of DSM-III was that this "defining features" approach does not apply well to psychiatric diagnosis, leading to the current "Chinese menu" or polythetic system, in which a patient can meet criteria for a disorder in multiple different ways (e.g., by having four or more of one kind of symptom and three or more of another).

Effective implementation of this approach still depends, however, on clinicians' ability and willingness to use this approach in a regular, reliable fashion. The problem is that people (in this case, diagnosing clinicians) tend to make decisions to categorize complex novel stimuli (in this case, patient presentations) through a decision-making process that uses a "probabilistic" assessment of degree of match to strong "exemplars" in their minds of the category (e.g., patients with "florid" manic symptoms they have seen) or to an abstract category—that is, a "prototype"—rather than through a classical approach to categorization (Folstein & Van Petten, 2004; Medin, 1989). Prototypes are mental models based on characteristics that are common in members of the group ("common features") rather than "defining features" (i.e., necessary prerequisites for category membership) (Rosch & Mervis, 1975).

Numerous philosophers and psychologists (Rosch & Lloyd, 1978; Rosch & Mervis, 1975; Weber, 1949; Wittgenstein, 1953) have observed that most of the objects and concepts we encounter in daily life are not rapidly or easily categorized based on defining features. Rather, they belong to "fuzzy" categories, whose members share many features (likened to "family resemblance") but do not share a set of necessary and sufficient features. Reliance on prototypes is especially likely in the domain of these so-called "fuzzy concepts," within which psychiatric diagnoses and many other psychological constructs, like emotions, currently fall (Rosch, 1978; Rosch & Lloyd, 1978). It should be noted, though, that almost all fields run into "fuzziness"-related classification issues (e.g., the platypus is an animal so difficult to categorize that it was initially believed to be a hoax produced by a taxidermist who had sewn a duck's beak onto a beaver). Other areas of psychology, such as research on social cognition (e.g., Hassebrauck & Aron, 2001; Lane & Gibbons, 2007; Niedenthal & Mordkoff, 1991), have effectively utilized prototype theory to clarify definitional arguments in the field (e.g., Kearns & Fincham, 2004). For example, because the ability to identify another person's emotional state accurately requires quickly processing a large amount of information (e.g., situational information; sociocultural context; demographic information, such as gender and age; facial expressions; etc.), a rule-based approach is much less effective than a prototype approach (Barrett, Mesquita, Ochsner, & Gross, 2007; Burch & Pishkin, 1984; Russell, 2003). Recent research further clarifies the limitations of rule-based categorization: It suggests that people with autism spectrum disorders have a tendency to focus on individual facial features rather than configurations when identifying emotions, and likewise tend to use rule-based rather than template-based approaches to identifying emotions (Rutherford & McIntosh, 2007). Likewise, developmental psychology research notes that as young children develop, they move from a tendency to making categorization judgments based on perceptual similarity (e.g., similar shapes) and shift toward making categorization judgments based on conceptual similarity (e.g., whether something serves a similar function) (Gentner & Namy, 1999).

Thus, given that clinical diagnosis is a specific form of categorization and decision making, it seems that clinicians are more likely inherently to invoke prototypes in their diagnostic decisions. To some degree, this prototype-based approach is acknowledged in the current DSM diagnostic system, as it has incorporated many of its elements. In fact, the DSM developers of the past recognized the utility of prototypes and even pictured DSM-III's polythetic system as an operationalization of a prototype-based diagnostic system (Frances, 1982; Widiger & Frances, 1985). Instead of having a clinician make a decision based on an aggregated prototypical representation, though, the current system breaks the decision down into individual symptom criteria rated as present or absent. The whole is equal to the sum of its parts (plus or minus a few criteria). The current system for diagnosis has led to quantum leaps forward in the understanding of psychopathology. Nevertheless, clinicians do

not appear to make many of the fine-grained distinctions required for valid DSM-IV diagnosis (e.g., whether a patient with severe depression actually has had one of two primary symptoms and at least four additional symptoms for a minimum of 2 weeks). Clinical practice is an imperfect mechanism for assessing clinical utility, but it is likely to be a useful bellwether.

Not surprisingly, given its potential utility, a number of researchers have proposed dimensional modifications to DSM, including various prototype-related approaches, although most of these proposals focus on Axis II diagnosis. The simplest option retains the current or modified criteria and segments an individual's match to the prototype based on the number of criteria he or she meets (Oldham & Skodol, 2000; Widiger & Sanderson, 1995). One resulting system would allow labels of "prototypical" (all criteria met), "moderately present," "threshold," "subthreshold," "trait" (one to three criteria met), and "absent." Millon (Millon, 1969; Millon, Grossman, Millon, Meagher, & Ramnath, 2004; see also Millon, Grossman, & Tringone, Chapter 21, this volume) has also conceptualized personality disorders (PDs) in terms of prototypes and varying levels of severity. In addition, analyses of prototypes have been helpful for modifying criteria for specific disorders (e.g., Gude, Karterud, Pedersen, & Falkum, 2006; Helzer, Bucholz, & Gossop, 2007; Hummelen, Wilberg, Pedersen, & Karterud, 2008). For example in the case of the criteria for dependent PD, analyses testing the prototype in DSM-IV found that the criterion related to difficulties in expressing disagreement was more closely related to avoidant PD (Gude et al., 2006). Likewise, findings for obsessive–compulsive PD suggest that the current diagnosis is an incomplete prototype skewed toward work-related perfectionism, while missing theoretically related criteria about the need for predictability and the impact on relationships (Hummelen et al., 2008). Trait models including the five-factor model of personality (Derefinko & Lynam, 2007; Lynam & Widiger, 2001) have also been recruited for refining prototypes of PDs. Thus, although their application has varied, the utility of prototypes has been recognized by researchers from many theoretical perspectives.

One Approach to Operationalizing a Prototype Approach to Diagnosis

Over the last several years, our research team (Westen & Bradley, 2005; Westen, Heim, Morrison, Patterson, & Campbell, 2002; Westen & Shedler, 2000; Westen, Shedler, & Bradley, 2006) has been working on an alternative prototype-based diagnostic system whose format and brevity resemble DSM-II's paragraph-long, narrative descriptions of disorders; the systematic empirical selection of diagnostic criteria that was the goal of DSM-III through DSM-IV; and a combined dimensional–categorical approach to diagnosis characteristic of neither. A guiding assumption of this approach is that use of the diagnostic manual and reliability of clinical diagnosis are likely to increase if clinicians are not forced to make dichotomous (present–absent) decisions about either diagnoses treated as a whole (DSM-II) or "laundry lists" of conceptually unrelated diagnostic criteria treated individually and then combined via sometimes complex algorithms that vary across diagnoses and hence are difficult to learn (DSM-IV).

In the system we are proposing, the diagnostic task is to examine each diagnostic prototype *taken as a whole* and to gauge the extent to which a patient's symptom picture matches the prototype, resulting in a dimensional diagnosis (see Table 20.1). Once the researcher or clinician completes

TABLE 20.1. Sample Rating Scale for the Proposed Prototype Diagnostic System

Diagnosis	
5	Very good match (patient exemplifies this disorder; *prototypical* case)
4	Good match (patient has this disorder; *diagnosis* applies)
Features	
3	Significant match (patient has *significant features* of this disorder)
2	Slight match (patient has *minor features* of this disorder)
1	Little or no match (description does not apply)

an assessment, he or she rates the individual on a 5-point scale indicating the degree of match with the prototype description. This scale ranges from 1 for "Little or no match (description does not apply)" to 5 for "Very good match (patient exemplifies this disorder; *prototypical* case)." Ratings of 4 and 5 correspond to categorical "caseness," and a rating of 3 indicates "Significant match (patient has *significant features* of this disorder)" (much as physicians measure blood pressure treated as a continuous variable, but by convention refer to values in certain ranges as "borderline" or "high"). Thus a single rating yields both dimensional scores and a categorical score without relying on symptom counting, but instead allowing more complex similarity comparisons to occur. The default value for each diagnosis is 1 ("Little or no match"), so that clinicians only expend their time rating prototypes of disorders warranting a rating of 2 or higher; this allows rapid diagnosis. The ready translation of dimensional into categorical diagnosis (e.g., a 3 translating into "significant features") is, empirically, of particular use for communication among professionals, who are unlikely to find it useful to describe a patient as "3 on major depressive disorder, 2 on panic disorder" (one of the major limitations of potential dimensional approaches to psychiatric diagnosis). Table 20.2 lists the potential benefits of the proposed prototype-based diagnostic system, in view of the limitations of the current diagnostic system (for more discussion of these, see Westen & Bradley, 2005; Westen et al., 2002, 2006; Westen & Shedler, 2000). Comparisons of this model with the current DSM system for both Axis I and Axis II disorders by several research teams have shown that two-thirds to three-fourths of clinicians prefer the prototype-matching system to the count/cutoff method across samples and disorders (Rottman, Ahn, Sanislow, & Kim, 2009; Spitzer, First, Shedler, Westen, & Skodol, 2008; Westen, Shedler, & Bradley, 2006).

In research just completed to test the construct validity of a prototype matching approach to diagnosis, we conducted two studies. In the first, clinicians made both DSM-IV categorical and prototype diagnoses of a patient on several common Axis I diagnoses (mood, anxiety, and eating disorders), and patients self-reported symptoms for the same disorders. In the second, independent interviewers made prototype diagnoses using a systematic clinical interview (see Westen & Muderrisoglu, 2003, 2006) and the SCID-I/P. Patients were seen at outpatient university clinics (in Study 1) and primary care clinics (in Study 2). Clini-

TABLE 20.2. Benefits of Proposed Prototype Diagnostic System in Relation to Limitations of Current Diagnostic System

Current DSM system (*count/cutoff method*)	Proposed prototype diagnostic system (*5-point similarity rating*)
Relatively time-consuming; lack of consistency in format and diagnostic requirements across diagnoses	Takes less time; quick, efficient, and consistent format across diagnoses
Categorical judgments only	Dimensional and categorical diagnoses
Artificial diagnostic comorbidity	Profile of elevations on relevant diagnoses
Relatively high rates of mixed and not otherwise specified (NOS) diagnoses	No need for NOS diagnoses
Method of diagnosis (criterion counting) and efforts to avoid elimination of comorbidity may lead to core diagnostic criteria for a disorder	Potentially more comprehensive and clinically rich descriptions of associated features of each disorder
Discordant with the way the human mind naturally classifies (and thus in current disuse)	Congruent with human cognitive processes and consistently rated by clinicians as superior in clinical utility and user-friendliness

cians' prototype diagnoses in the first study showed moderate to high correlations with self-reports and performed as well or better than categorical DSM-IV diagnoses of the same disorders. Prototype diagnosis in the two independent interviews in the second study (one using a systematic clinical interview similar to interviewing practices by skilled clinical interviewers in everyday clinical practice) correlated $r = .50$ with each other on average and showed substantial incremental validity over categorical DSM-IV diagnoses in predicting adaptive functioning. These data suggested that prototype diagnoses made by one clinically skilled informant correlate with dimensional diagnosis of the same disorder or symptomatology (whether prototype or otherwise) as made by another informant, with both blind to each other's data.

The content of the prototype descriptions can be derived in a number of ways. For example, descriptions can come from the current or modified DSM descriptions of criteria or using empirical methods applied to various assessment devices. In the case of personality diagnosis, we have focused on work using the Shedler–Westen Assessment Procedure (SWAP), a 200-item clinician report measure of personality pathology that requires a ranking of the patient's most important personality characteristics, based on either extensive clinical knowledge of a patient or a systematic clinical interview (e.g., Shedler & Westen, 2004; Westen & Shedler, 2007). Relatively less work has been conducted with respect to establishing prototypical descriptions of Axis I disorders. To date, our work in this area (see below) has begun at the level of using the DSM criteria as the foundation for prototypical descriptions. However, we have also conducted some preliminary work focused on empirically deriving Axis I prototypes for which we also present initial results below. In addition, although our work and the work of others has thus far used the current DSM distinction between Axis I and Axis II disorders, a prototype-based diagnostic system would benefit greatly by rethinking the distinction between Axis I and Axis II disorders, as well as by including a description of healthy functioning that would allow clinicians to code adaptive functioning and strengths (see Westen et al., 2006).

Preliminary Research on the Prototype-Based Approach to Diagnosis

Eating Disorders

One of the first diagnostic classes we have studied using a prototype approach is the DSM-IV eating disorders section, which includes two diagnoses with two subtypes each—anorexia nervosa, with restricting and binge–purging subtypes, and bulimia nervosa, with purging and nonpurging subtypes—and an NOS diagnosis, for a total of five categories. Recently, there has even been discussion of adding a sixth diagnosis, binge-eating disorder, a residual category created by the requirement of both bingeing and purging for a bulimia diagnosis (see Fichter, Quadflieg, & Hedlund, 2008; Pope et al., 2006). Recent research suggests (1) that this system relegates 40–50% of patients with clinically significant eating pathology to a nondescript NOS category; and (2) that patients with both anorexic and bulimic symptoms, who are no more like patients with restricting anorexia than they are like patients with bulimia, are arbitrarily classified as having a subtype of anorexia (Morrison & Westen, 2002).

A prototype system, in contrast, would include only two prototypes, one for anorexia nervosa and one for bulimia nervosa (see Tables 20.3 and 20.4). Rather than counting symptoms and deciding whether a patient meets arbitrary severity and duration requirements (e.g., bingeing and purging at least twice a week for a minimum of 3 months), the clinician would simply rate the extent to which the patient's condition matches each prototype taken as a whole. A score of 4 or 5 on the bulimia prototype would mean that the patient's symptom picture strongly enough matches the diagnostic prototype to warrant a categorical diagnosis. A score of 3 on the anorexia prototype would mean that the patient's symptom picture resembles the prototype, but not enough to warrant a categorical diagnosis. The patient would thus receive a categorical diagnosis of "bulimia nervosa with anorexic features." Preliminary data (unpublished) suggest that these two simple prototypes carry as much or more information than DSM-IV categorical diagnoses of all eating

TABLE 20.3. Prototype Description of Anorexia Nervosa

Anorexia nervosa

Patients who match this prototype refuse to maintain their body weight at or above a minimally normal weight for their age and height. They have an intense fear of gaining weight or becoming fat, even though they are, or are in danger of becoming, substantially underweight. They tend to have a disturbance in the way they experience their body weight or shape, and may deny the seriousness of their low body weight. Their body weight or shape exerts undue influence on their views of and feelings about themselves. Patients who match this prototype may develop amenorrhea (i.e., cessation of menstruation) when their weight is low.

Diagnosis

5 Very good match (patient exemplifies this disorder; *prototypical* case)

4 Good match (patient *has* this disorder; *diagnosis* applies)

Features

3 Moderate match (patient has *significant features* of this disorder)

2 Slight match (patient has *minor features* of this disorder)

1 Little or no match (description does not apply)

TABLE 20.4. Prototype Description of Bulimia Nervosa

Bulimia nervosa

Patients who match this prototype engage in recurrent episodes of binge eating and purging. During binges, they eat, in a discrete period of time, an amount of food that is clearly larger than most people would eat during a similar period of time and under similar circumstances. Binges are typically accompanied by a sense of lack of control (e.g., a feeling that they cannot stop eating or control what or how much they are eating). Patients who match this prototype exhibit recurrent inappropriate compensatory behavior in order to prevent weight gain, such as self-induced vomiting; misuse of laxatives, diuretics, enemas, or other medications; fasting; or excessive exercise. Their views of and feelings about themselves are also unduly influenced by body shape and weight. *Note*: For patients who match features of this prototype but only binge or only purge, score 3.

Diagnosis

5 Very good match (patient exemplifies this disorder; *prototypical* case)

4 Good match (patient has this disorder; *diagnosis* applies)

Features

3 Moderate match (patient has *significant features* of this disorder)

2 Slight match (patient has *minor features* of this disorder)

1 Little or no match (description does not apply)

disorder diagnoses combined and are rated by clinicians as substantially higher in clinical utility and useability.

Posttraumatic Stress Disorder

Both the criteria for and the diagnosis of posttraumatic stress disorder (PTSD) itself have been criticized for problems with comorbidity, specificity of symptoms, and lack of clarity and agreement regarding what constitutes a traumatic experience (Rosen & Lilienfeld, 2008; Rosen, Spitzer, & McHugh, 2008). In regard to the criteria, a number of factor analyses have been conducted on PTSD symptomatology to assess the underlying factor structure; however, little consensus has been reached about whether a three-factor (e.g., intrusion/avoidance, dysphoria, and hyperarousal; Lancaster, Melka, & Rodriguez, 2009) or a four-factor (e.g., re-experiencing, effortful avoidance, emotional numbing, and hyperarousal; King, Leskin, King, & Weathers, 1998) solution is most fitting. High comorbidity rates are also a problem with mood disorders (Campbell et al., 2007; Franklin & Zimmerman, 2001; Oquendo et al., 2005), substance use disorders (Chilcoat & Breslau, 1998; Mills, Teesson, Ross, & Peters, 2006), and other disorders (Abram et al., 2007; Deering, Glover, Ready, Eddleman, & Alarcón, 1996; Kilpatrick et al., 2003; Zlotnick, 1997). The current diagnostic system for PTSD, which requires at least one symptom of reexperiencing, three of avoidance/numbing, and two of hyperarousal, is cumbersome and difficult to remember. In fact, one of us (Bekh Bradley) has been working in the area of PTSD research for several years and still had to refer back

to DSM-IV-TR for the preceding sentence. Moreover, evidence exists that this lack of parsimony does not lead to good clinical work. Levels of PTSD symptoms that would be subthreshold in the current diagnostic system (e.g., one symptom in each of the three symptom categories) are associated with significant problems in adaptive functioning as well as with increased suicidal ideation, even after comorbid depression is controlled for (Marshall et al., 2001).

A prototype approach to diagnosing PTSD addresses the three issues with PTSD raised above. First, the question about factor structure of PTSD symptoms would become irrelevant in a prototype approach because by definition the core clinical features of PTSD are presented together as part of an overall symptom picture rather than being divided into clusters of dubious validity and reliability (although we have experimented with prototype approaches in which clinicians make secondary ratings of severity of symptom clusters within the diagnosis, generally derived empirically through factor analysis). Second, if a prototype approach were used for PTSD as well as for other comorbid disorders, areas of symptom overlap could be attributed to the diagnosis with which they are most likely to be associated. For example, dysphoria related to anhedonia and general hopelessness about the future would most appropriately "belong" to a depression prototype, whereas dysphoria associated with persistent thoughts of a traumatic event would most appropriately "belong" to a PTSD prototype. Finally, a prototype approach allows clinicians and researchers to attend to and accurately code for PTSD-related symptoms that are currently classified as "subthreshold."

Initial data on a prototype approach to PTSD diagnosis were collected as part of a larger study funded by the National Institute of Mental Health (NIMH) and based at Grady Memorial Hospital, a publicly funded, not-for-profit health care system in Atlanta, Georgia. The broader project's purpose has been to examine the correlates of the development of PTSD in a low-income, urban, primarily African American population. Participants were recruited while they were waiting in the primary care and obstetrics–gynecology clinics, and were invited to participate in a research project about stress and coping. Participants completed a series of structured and semistructured assessment interviews over 4 days; these included 1 day's worth of structured interviews, including the Clinician-Administered PTSD Scale (CAPS; Blake et al., 1997) and the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I; Gibbon & Williams, 2002), in addition to a number of self-report instruments. Study methods are described more thoroughly elsewhere (Binder et al., 2008; Schwartz et al., 2006). In addition, the participants were rated on a prototype description of PTSD derived from the current DSM-IV PTSD criteria (see Table 20.5). A total of 169 individuals were both interviewed with the CAPS and rated on the prototype system for PTSD. CAPS-based and prototype-based categorical diagnoses overlapped 91.7% of the time. Both methods correlated positively with self-reported PTSD symptom frequency in general and by symptom cluster, based on the PTSD Symptom Scale (PSS; Foa, Riggs, Dancu, & Rothbaum, 1993). Rates of comorbidity were generally similar, though lower, for categorical DSM-IV and prototype diagnosis.

We also examined the association between PTSD as rated by both diagnostic approaches with exposure to childhood abuse and with overall level of exposure to traumatic events across the lifespan. Prototype ratings had slightly higher correlations for the childhood variables, although diagnoses using both methods positively correlated with childhood emotional, sexual, and physical abuse frequency, as measured by the Early Trauma Inventory (ETI; Bremner, Vermetten, & Mazure, 2000), and with total types of trauma experienced with and without childhood trauma included, as measured by the Traumatic Events Inventory (TEI; Rothbaum & Davidson, n.d.).

We assessed adaptive functioning across multiple areas, including items from the Life Base interview (Keller et al., 1987), the SCID-I (Gibbon & Williams, 2002), and the clinician-rated Clinical Data Form (CDF; Westen & Shedler, 1999; Westen, Shedler, Durrett, Glass, & Martens, 2003).We aggregated standardized adaptive functioning variables from the Life Base (self-reported and interviewer-assessed life satisfaction in past month and best 6-month period of past 2 years), CDF (personality functioning, quality of romantic relationships, friendships, employment functioning, number of close relationships, and physical health),

TABLE 20.5. Prototype Description of Posttraumatic Stress Disorder

Posttraumatic Stress Disorder

Patients who match this prototype have experienced or witnessed a traumatic event—that is, an event that involved actual or threatened death or serious injury to the self or others—that elicited intense feelings of fear, helplessness, or horror. They persistently reexperience the event, which may haunt them in numerous forms: They may have intrusive thoughts, mental images, or dreams related to the trauma; they may feel as if they are reliving the event, through flashbacks, illusions, hallucinatory images, or a sense that the event is occurring again; or they may experience intense psychological distress or physiological arousal when "triggered" by cues that resemble or symbolize the event. Patients who match this prototype try to avoid stimuli, thoughts, feelings, places, people, or conversations that might remind them of the event, and are often unable to recall important aspects of it. They may also "shut down," experiencing an emotional numbing that leaves them with a restricted range of emotion, a sense of a foreshortened future (e.g., not expecting to have a career, marriage, children, or normal lifespan), feelings of detachment or estrangement from others, or diminished interest or participation in significant activities that once excited them. Patients who match this prototype have persistent symptoms of physiological arousal, such as difficulty falling or staying asleep, difficulty concentrating, exaggerated startle response, hypersensitivity to possible signs of danger, or irritability or outbursts of anger.

Diagnosis

5 Very good match (patient exemplifies this disorder; *prototypical* case)

4 Good match (patient has this disorder; *diagnosis* applies)

Features

3 Moderate match (patient has *significant features* of this disorder)

2 Slight match (patient has *minor features* of this disorder)

1 Little or no match (description does not apply)

and interviewer ratings (DSM-based Global Assessment of Functioning [GAF], two interviewer ratings of match to a health prototype; see Westen et al., 2006). (This aggregated variable had high internal consistency, Cronbach's $\alpha = .84$.) Both diagnostic methods correlated with the aggregated adaptive functioning variable to the same degree ($r = -.28$), and hierarchical regressions confirmed that CAPS-based diagnosis did not predict adaptive functioning above prototype diagnosis and vice versa.

In summary, our initial data using a prototype-based approach to PTSD diagnosis indicate that the pattern of PTSD prevalence and distribution of dimensional ratings were comparable between diagnostic methods, as was the pattern of external validity. These findings provide another example of how prototype-based diagnosis can enhance the efficiency of the diagnostic process, while not sacrificing the validity of decisions.

Mood Disorders

In addition to using current DSM criteria as the foundation for deriving prototype descriptions of Axis I disorders, we have begun working on using psychometric instruments designed for use by expert informants (clinically experienced observers) in large clinical samples to develop empirically derived prototypes. As part of our ongoing research on the classification of PDs, a subsample of clinicians ($N = 120$) from a National Institute of Mental Health–funded study completed a 79-item clinician report instrument for assessing mood disorders, the Mood Disorders Questionnaire, to describe a randomly selected patient in their practice. We derived the items for this questionnaire from the items from DSM-III, DSM-III-R, and DSM-IV criteria for mood disorders; relevant research and clinical literature; examination of the item sets from all major interviews and self-report inventories assessing the domain; and our clinical experience with inpatients and outpatients. Each item is scored on a 7-point scale (1, "not true at all"; 4, "somewhat true"; 7, "very true"). Our goal was to see whether we could simplify the mood disorder categories in the DSM-IV by identifying a small number of mood dimensions. As can be seen in Table 20.6, an exploratory factor analysis produced three clear factors (major depression, mania, and dysthymia).

TABLE 20.6. Exploratory Factor Analysis of the Clinician Report Mood Disorders Questionnaire

Items	Factor loadings 1	2	3
Factor 1: Major depression			
Mood is consistently depressed; does not respond to efforts to "cheer him/her up"	.90		
Has trouble enjoying him-herself; derives little pleasure from life	.87		
Is unable to enjoy usual interests and activities, etc.	.85		
Is fatigued, tired, or lacking in energy; everyday activities require enormous effort	.83		
Feels helpless; believes nothing s/he can do will make things all right, better, etc.	.83		
Feels life is not worth living	.82		
Feels hopeless about the future	.78		
Depressed mood has a serious impact on ability to function at work, school, etc.	.74		
Wishes s/he were dead or feels would be better off dead	.72		
Seems slowed down in thought, speech, movement, etc.	.68		
Depression or agitation interferes with ability to concentrate; has trouble reading, sustaining a conversation, etc.	.66		
Has flat or blunted affect; shows little emotion even with matters of import	.62		
Depressed mood seems qualitatively different from prior mood states (even ordinary sadness), as if a cloud or fog has descended	.58		
Is emotionally paralyzed; has trouble making everyday decisions	.58		
Feels lonely or painfully alone	.58		
Has diminished appetite	.56		
Views self as loathsome, evil, contaminating, or totally bad; has global self-hatred for who s/he is	.55		
Is consumed by suicidal thoughts	.55		
Depression is noticeably worse in the morning	.52		
Believes his/her depression is punishment for who s/he is or what s/he has done	.51		
Factor 2: Mania			
Mood cycles rapidly between high, irritable, or manic states and depressed or mixed states over a relatively brief period (e.g., weeks or months)		.80	
Has boundless energy, in a way that differs from his/her usual functioning		.63	
Abnormally elevated, expansive, or irritable mood leads to impairment in usual occupational functioning, social activities, or relationships, or necessitates hospitalization		.62	
Takes undue risks (e.g., financial ventures, reckless driving, illegal activities) with minimal concern for consequences, in a way that differs from his/her usual functioning		.61	
Jumps rapidly from idea to idea in a way that can make communication difficult to follow		.61	
Speech is rapid, nonstop, or pressured, in a way that differs from his/her usual functioning		.57	
Engages in thrill-seeking or otherwise "high-gain," reward-driven behavior (e.g., gambling, spending, indiscriminate sexual encounters), in a way that differs from his/her usual functioning		.52	
Is grandiose or unduly self-confident (e.g., believes s/he can "do anything"), in a way that differs from his/her usual functioning		.50	
Gets angry or irritable more easily than usual; has a "short fuse"		.50	
Experiences thoughts as racing or coming "a mile a minute," in a way that differs from his/her usual functioning; may feel like thoughts come to mind so quickly that s/he cannot keep up with them		.49	
Is hypersexual, in a way that differs from his/her usual functioning		.48	
Is unusually driven or goal-directed (socially, at work or school, etc.), in a way that differs from his/her usual functioning		.47	
Is restless, fidgety, or unable to sit still; has psychomotor agitation		.47	
Is explosive or flies into rages, in a way that differs from his/her usual functioning		.44	

(cont.)

TABLE 20.6. *(cont.)*

Items	Factor loadings 1	2	3
Factor 3: Dysthymia			
Derives sense of self-worth from others' appraisals; needs approval, assurance, etc.			.79
Feels guilty			.73
Tends to blame self for bad things that happen; attributes misfortunes to own enduring psychological traits or attributes			.67
Worries about disappointing significant others			.65
Tends to ruminate over perceived past errors, bad deeds, etc.			.62
Tends to fear rejection or abandonment by significant others			.61
Is self-critical; sets high standards for self and chronically fears s/he is not living up to them			.58
Derives sense of self-worth from achievements and accomplishments			.53
Has low self-esteem			.50
Feels inferior, inadequate, incompetent, or a failure			.49

The major depression factor was marked by items describing discrete episodes of illness strongly resembling DSM-IV criteria, whereas the dysthymia factor was marked by items describing enduring depressive phenomenology. We will soon be exploring whether these three prototypes alone can capture all of the information provided by the multiple mood disorder diagnoses that currently take up dozens of pages in DSM-IV, by capturing spectrum disorders dimensionally rather than categorically.

Cautions and Concerns about Clinical/Human Thought

Although the available theory and research suggest that a prototype approach to psychiatric diagnosis would be beneficial in a number of ways, we also recognize that such an approach is no panacea. The benefits of moving toward the proposed prototype-matching diagnostic system include decreased artificial comorbidity, increased ease of use, and diagnostic descriptions of disorders that are both clinically richer than DSM-IV criteria and empirically derived (as in the mood disorder prototypes just described) (Westen et al., 2006). Nevertheless, such a diagnostic system is not immune to possible errors of human thought that can affect clinical decision making (Garb, 1998). Cognitive heuristics will always play a role in making social judgments, and although these are helpful for the most part, they can also lead to bias. The representativeness and availability heuristics (Tversky & Kahneman, 1974) may be particularly prone to result in errors when clinicians are matching individuals to prototypes because they can be biased if not calibrated to the *right* prototype (or exemplars). For example, a clinician's first (or most memorable) clinical experience of working with a patient diagnosed with any given psychiatric disorder may become the template against which future patients are diagnosed. Other errors that need to be addressed include illusory correlations in the prototype description or a clinician's assessment of a case (Lueger & Petzel, 1979) and the influence of expectations and self-fulfilling prophecies (Harris, 1994).

These problems are inherent to human cognition and thus to any diagnostic system. However, care can be taken in using research to derive the disorders empirically, in creating the prototype descriptions for each resultant diagnosis, and in studying the effects of stereotypes and other biases. Blaming clinical errors on "just being human" is not enough if one can demonstrate that attention to common cognitive and diagnostic mistakes can increase the reliability and validity of diagnosis. Prototype diagnosis will probably prove to work best when these concerns are addressed thoughtfully and empirically.

Conclusion and Future Research

In sum, categorical thinking may be easier and dimensional data may be more precise,

but prototype diagnosis may offer the best of both worlds. Clearly, however, more research needs to be conducted with respect to the relative merits and problems associated with prototype-based diagnosis. The prototype-based rating system we have described can be implemented in combination with the current set of diagnoses (i.e., changing only the method of diagnosis, from symptom counting to prototype matching). However, as described here, we can also apply this approach in combination with empirically refined diagnostic groupings (i.e., changing both the taxonomy and the method of diagnosis). Such an approach could utilize empirically derived prototypes and lead to fewer diagnostic categories, more distinct diagnoses, or a hierarchical system in which broad diagnoses can be further broken down into subgroups (e.g., fragile narcissism as a subtype of narcissistic PD; Russ, Shedler, Bradley, & Westen, 2008). Our data on subtypes of Axis I disorders (Thompson-Brenner, Eddy, Satir, Boisseau, & Westen, 2008) also suggest that an empirically derived prototype classification of psychopathology could rework the current model of personality as diagnostically orthogonal to Axis I disorders.

We have also described here the simplest approach to prototype diagnosis, with a single rating per disorder. As noted earlier, however, it is possible that more complex approaches will prove clinically or empirically useful. For example, patients who receive a rating of 3 or higher (on a 1–5 scale) of PTSD could then receive a series of additional, clinically relevant ratings, such as age of onset, duration of illness, and the extent to which intrusive versus avoidance behaviors characterize the clinical profile. This type of clarification might allow a better match of treatment to patient within diagnosis. For example, we have found that when avoidance is more predominant than intrusive symptoms in PTSD, an early focus on *in vivo* exposure therapy can at times help the patient establish the ability to recover more quickly from other PTSD symptoms (e.g., intrusive memories) outside of treatment. The inclusion of a psychological health prototype could also prove useful in rating global adaptive functioning in a more meaningful manner than the current GAF scale in DSM. An NIMH-funded study is now underway to address many of these questions and to compare the most viable categorical, dimensional, and prototype-based approaches vying for inclusion in Axis II of DSM-V. This multi-informant, longitudinal grant will address the benefits and costs of each approach in convergent, discriminant, and predictive validity.

In conclusion, clinicians, like all other information processors, try to elicit the information they need to solve problems. If clinicians of all theoretical orientations and disciplines gravitate toward diagnostic methods other than those prescribed in the diagnostic manual (e.g., see Westen & Arkowitz-Westen, 1998), it may be that the manual as configured is not optimally serving their purposes. The classification system that laid the foundation for diagnosis in psychiatry since DSM-III was derived directly from a research manual, the *Research Diagnostic Criteria* (Spitzer et al., 1978). Although the goals of clinical and research diagnosis overlap substantially, they diverge in some important respects (Westen, 1997, 1998). The most common explanation for the unreliability of clinical diagnosis since DSM-III (a document explicitly designed to address such unreliability through carefully specified criteria and cutoffs) is that the problem lies with clinicians, who need to follow the procedures in the manual more closely. Although there is little doubt that clinical interviewing in everyday practice should be much more systematic, it is unlikely to become so if the official diagnostic method relies on an approach to decision making different from the ones clinicians find clinically relevant and useful in practice.

Research in cognitive science suggests that in everyday judgment and decision making, people tend to "satisfice" (a cross between "satisfy" and "suffice")—that is, to make a "good enough" assessment for their purposes, and to make more precise determinations based on explicit decision rules if the need arises (Gigerenzer & Goldstein, 1996; Simon, 1978). Rather than expending the time and effort required to diagnose panic disorder formally, for example, a clinician may be content to diagnose that the patient suffers from moderate, clinically significant panic symptoms once or twice a week. In light of the dearth of research on the treatment implications of clinical versus subthreshold symptoms (and in light of data suggesting that subthreshold variants often produce similar or substantial functional

impairments; e.g., Fava, 1999; Fava & Mangelli, 2001; Marshall et al., 2001), satisficing may not be an irrational diagnostic strategy in clinical practice, particularly in initial interviews.

Accurate clinical diagnosis is essential for translation of all research on classification, etiology, and treatment into clinical practice. No matter how carefully researchers make diagnoses in clinical trials, for example, such efforts will fall short of their intended goals if clinicians cannot make reliable diagnostic judgments in everyday practice that allow them to identify cases to which research findings are likely to apply. It is our hope that the approach to diagnosis proposed in this chapter may allow for closer connection between the approach to diagnosis used in clinical settings and the data gathered in research on psychopathology, treatment, risk, and resilience.

Acknowledgments

Research presented in this chapter was funded by the National Institute of Mental Health (Grant Nos. MH071537, MH62377, and MH078100), the Grady Memorial Hospital General Research Center, the National Institutes of Health National Center for Research Resources (Grant No. M01 RR0039), the American Suicide Foundation Young Investigator Grant to Bekh Bradley, and the Emory University Research Committee Grant to Bekh Bradley.

References

Abram, K. M., Washburn, J. J., Teplin, L. A., Emanuel, K. M., Romero, E. G., & McClelland, G. M. (2007). Posttraumatic stress disorder and psychiatric comorbidity among detained youths. *Psychiatric Services*, *58*(10), 1311.

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., rev.). Washington, DC: Author.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.

Andersson, G., & Ghaderi, A. (2006). Overview and analysis of the behaviourist criticism of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM). *Clinical Psychologist*, *10*(2), 67–77.

Barlow, D. (2002). *Anxiety and its disorders* (2nd ed.). New York: Guilford Press.

Barrett, L. F., Mesquita, B., Ochsner, K. N., & Gross, J. J. (2007). The experience of emotion. *Annual Review of Psychology*, *58*, 373–403.

Beutler, L. E., & Malik, M. L. (Eds.). (2002). *Rethinking the DSM: A psychological perspective*. Washington, DC: American Psychological Association.

Binder, E. B., Bradley, R. G., Liu, W., Epstein, M. P., Deveau, T. C., Mercer, K. B., et al. (2008). Association of FKBP5 polymorphisms and childhood abuse with risk of posttraumatic stress disorder symptoms in adults. *Journal of the American Medical Association*, *299*(11), 1291–1305.

Blake, D. D., Weathers, F. W., Nagy, L. M., Kaloupek, D. G., Charney, D. S., & Keane, T. M. (1997). *Clinician-Administered PTSD Scale for DSM-IV (CAPS-DX)—Current and Lifetime Diagnostic Version*. Boston: National Center for Posttraumatic Stress Disorder, Behavioral Science Division, Boston VAMC.

Bremner, J. D., Vermetten, E., & Mazure, C. M. (2000). Development and preliminary psychometric properties of an instrument for the measurement of childhood trauma: The Early Trauma Inventory. *Depression and Anxiety*, *12*(1), 1–12.

Brown, T. A., Chorpita, B. F., & Barlow, D. H. (1998). Structural relationships among dimensions of the DSM-IV anxiety and mood disorders and dimensions of negative affect, positive affect, and autonomic arousal. *Journal of Abnormal Psychology*, *107*, 179–192.

Burch, J. W., & Pishkin, V. (1984). Family resemblance: Category structure of joy and shame. *Journal of Clinical Psychology*, *40*(5), 1136–1143.

Campbell, D. G., Felker, B. L., Liu, C. F., Yano, E. M., Kirchner, J. A. E., Chan, D., et al. (2007). Prevalence of depression–PTSD comorbidity: Implications for clinical practice guidelines and primary care-based interventions. *Journal of General Internal Medicine*, *22*(6), 711–718.

Chilcoat, H. D., & Breslau, N. (1998). Investigations of causal pathways between ptsd and drug use disorders. *Addictive Behaviors*, *23*(6), 827–840.

Clark, L. A. (1993). *SNAP (Schedule for Nonadaptive and Adaptive Personality): Manual*

for administration, scoring and interpretation. Minneapolis: University of Minnesota Press.

Deering, C. G., Glover, S. G., Ready, D., Eddleman, H. C., & Alarcón, R. D. (1996). Unique patterns of comorbidity in posttraumatic stress disorder from different sources of trauma. *Comprehensive Psychiatry*, *37*(5), 336–346.

Denton, W. H. (2007). Issues for DSM-V: Relational diagnosis: An essential component of biopsychosocial assessment [Editorial]. *American Journal of Psychiatry*, *164*(8), 1146–1147.

Derefinko, K. J., & Lynam, D. R. (2007). Using the FFM to conceptualize psychopathy: A test using a drug abusing sample. *Journal of Personality Disorders*, *21*(6), 638–656.

Elklit, A., & Shevlin, M. (2007). The structure of PTSD symptoms: A test of alternative models using confirmatory factor analysis. *British Journal of Clinical Psychology*, *46*(3), 299–313.

Eubanks-Carter, C., & Goldfried, M. R. (2006). The impact of client sexual orientation and sender on clinical judgments and diagnosis of borderline personality disorder. *Journal of Clinical Psychology*, *62*(6), 751–770.

Fava, G. A. (1999). Subclinical symptoms in mood disorders: Pathophysiological and therapeutic implications. *Psychological Medicine*, *29*, 47–61.

Fava, G. A., & Mangelli, L. (2001). Assessment of subclinical symptoms and psychological well-being in depression. *European Archive of Psychiatry and Clinical Neuroscience*, *251*, 47–52.

Feighner, J. P., Robins, E., Guze, S. B., Woodruff, R. A., Winokur, G., & Munoz, R. (1972). Diagnostic criteria for use in psychiatric research. *Archives of General Psychiatry*, *26*, 57–63.

Fichter, M. M., Quadflieg, N., & Hedlund, S. (2008). Long-term course of binge eating disorder and bulimia nervosa: Relevance for nosology and diagnostic criteria. *International Journal of Eating Disorders*, *41*, 577–586.

First, M. B. (2005). Toward a dimensionally based taxonomy of psychopathology: Clinical utility: A prerequisite for the adoption of a dimensional approach in DSM. *Journal of Abnormal Psychology*, *114*(4), 560–564.

Foa, E. B., Riggs, D. S., Dancu, C. V., & Rothbaum, B. O. (1993). Reliability and validity of a brief instrument for assessing post-traumatic stress disorder. *Journal of Traumatic Stress*, *6*(4), 459–473.

Folstein, J. R., & Van Petten, C. (2004). Multidimensional rule, unidimensional rule, and similarity strategies in categorization: Event-related brain potential correlates. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *30*(5), 1026–1044.

Frances, A. (1982). Categorical and dimensional systems of personality diagnosis: A comparison. *Comprehensive Psychiatry*, *23*, 516–527.

Frances, A. (1993). Dimensional diagnosis of personality: Not whether, but when and which. *Psychological Inquiry*, *4*(2), 110–111.

Franklin, C. L., & Zimmerman, M. (2001). Posttraumatic stress disorder and major depressive disorder: Investigating the role of overlapping symptoms in diagnostic comorbidity. *Journal of Nervous and Mental Disease*, *189*(8), 548–551.

Garb, H. N. (1998). *Studying the clinician: Judgment research and psychological assessment*. Washington, DC: American Psychological Association.

Gentner, D., & Namy, L. L. (1999). Comparison in the development of categories. *Cognitive Development*, *14*(4), 487–513.

Gibbon, M., & Williams, J. B. W. (2002). *Structured Clinical Interview for DSM-IV Axis I Disorders, Research Version, Non-patient Edition (SCID-I/NP)*. New York: Biometrics Research, New York State Psychiatric Institute.

Gigerenzer, G., & Goldstein, D. G. (1996). Reasoning the fast and frugal way: Models of bounded rationality. *Psychological Review*, *103*, 650–669.

Gude, T., Karterud, S., Pedersen, G., & Falkum, E. (2006). The quality of the *Diagnostic and Statistical Manual of Mental Disorders*, dependent personality disorder prototype. *Comprehensive Psychiatry*, *47*(6), 456–462.

Harris, M. J. (1994). Self-fulfilling prophecies in the clinical context: Review and implications for clinical practice. *Applied and Preventive Psychology*, *3*, 145–158.

Hassebrauck, M., & Aron, A. (2001). Prototype matching in close relationships. *Personality and Social Psychology Bulletin*, *27*(9), 1111–1122.

Helzer, J. E., Bucholz, K. K., & Gossop, M. (2007). A dimensional option for the diagnosis of substance dependence in DSM-V. *International Journal of Methods in Psychiatric Research*, *16*(Suppl. 1), S24–S33.

Hummelen, B., Wilberg, T., Pedersen, G., & Karterud, S. (2008). The quality of the DSM-IV obsessive–compulsive personality disorder construct as a prototype category. *Journal of Nervous and Mental Disease*, *196*(6), 446–455.

Kearns, J. N., & Fincham, F. D. (2004). A prototype analysis of forgiveness. *Personality and Social Psychology Bulletin*, *30*(7), 838–855.

Keller, M. B., Lavori, P. W., Friedman, B., Nielsen, E., Endicott, J., McDonald-Scott, P., et al. (1987). The Longitudinal Interval Follow-Up Evaluation: A comprehensive method for assessing outcome in prospective longitudinal studies. *Archives of General Psychiatry*, *44*(6), 540–548.

Kessler, R. C., Nelson, C. B., McGonagle, K. A., Liu, J., Swartz, M., & Blazer, D. G. (1996). Comorbidity of DSM-III-R major depressive disorder in the general population: Results from the US National Comorbidity Survey. *British Journal of Psychiatry*, *168*(Suppl. 30), 17–30.

Kilpatrick, D. G., Ruggiero, K. J., Acierno, R., Saunders, B. E., Resnick, H. S., & Best, C. L. (2003). Violence and risk of PTSD, major depression, substance abuse/dependence, and comorbidity: Results from the National Survey of Adolescents. *Journal of Consulting and Clinical Psychology*, *71*(4), 692–700.

King, D. W., Leskin, G. A., King, L. A., & Weathers, F. W. (1998). Confirmatory factor analysis of the Clinician-Administered PTSD Scale: Evidence for the dimensionality of posttraumatic stress disorder. *Psychological Assessment*, *10*, 90–96.

Krueger, R. F., & Finger, M. S. (2001). Using item response theory to understand comorbidity among anxiety and unipolar mood disorders. *Psychological Assessment*, *13*(1), 140–151.

Krueger, R. F., Hicks, B. M., Patrick, C. J., Carlson, S. R., Lacono, W. G., & McGue, M. (2002). Etiologic connections among substance dependence, antisocial behavior, and personality: Modeling the externalizing spectrum. *Journal of Abnormal Psychology*, *111*(3), 411–424.

Lancaster, S. L., Melka, S. E., & Rodriguez, B. F. (2009). A factor analytic comparison of five models of PTSD symptoms. *Journal of Anxiety Disorders*, *23*(2), 269–274.

Lane, D. J., & Gibbons, F. X. (2007). Am I the typical student?: Perceived similarity to student prototypes predicts success. *Personality and Social Psychology Bulletin*, *33*(10), 1380–1391.

Lenzenweger, M. F. (1997). Schizophrenia: Refining the phenotype, resolving the endophenotypes. *Behaviour Research and Therapy*, *37*, 281–295.

Lichtenthal, W. G., Cruess, D. G., & Prigerson, H. G. (2004). A case for establishing complicated grief as a distinct mental disorder in DSM-V. *Clinical Psychology Review*, *24*(6), 637–662.

Lueger, R. J., & Petzel, T. P. (1979). Illusory correlation in clinical judgment: Effects of amount of information to be processed. *Journal of Consulting and Clinical Psychology*, *47*(6), 1120–1121.

Lynam, D. R., & Widiger, T. A. (2001). Using the five-factor model to represent the DSM-IV personality disorders: An expert consensus approach. *Journal of Abnormal Psychology*, *110*(3), 401–412.

Marshall, R. D., Olfson, M., Hellman, F., Blanco, C., Guardino, M., & Struening, E. L. (2001). Comorbidity, impairment, and suicidality in subthreshold PTSD. *American Journal of Psychiatry*, *158*, 1467–1473.

Martin, C. S., Chung, T., & Langenbucher, J. W. (2008). How should we revise diagnostic criteria for substance use disorders in the DSM-V? *Journal of Abnormal Psychology*, *117*(3), 561–575.

Mayou, R., Kirmayer, L. J., Simon, G., Kroenke, K., & Sharpe, M. (2005). Somatoform disorders: Time for a new approach in DSM-V. *American Journal of Psychiatry*, *162*, 847–855.

McDonald, S. D., Beckham, J. C., Morey, R., Marx, C., Tupler, L. A., & Calhoun, P. S. (2008). Factorial invariance of posttraumatic stress disorder symptoms across three veteran samples. *Journal of Traumatic Stress*, *21*(3), 309–317.

Medin, D. L. (1989). Concepts and conceptual structure. *American Psychologist*, *44*, 1469–1481.

Millon, T. (1969). *Modern psychopathology: A biosocial approach to maladaptive learning and functioning*. Philadelphia: Saunders.

Millon, T., Grossman, S., Millon, C. M., Meagher, S., & Ramnath, R. (2004). *Personality disorders in modern life* (2nd ed.). Hoboken, NJ: Wiley.

Mills, K. L., Teesson, M., Ross, J., & Peters, L. (2006). Trauma, PTSD, and substance use disorders: Findings from the Australian National Survey of Mental Health and Well-Being. *American Journal of Psychiatry*, *163*(4), 652–658.

Morrison, C., & Westen, D. (2002). *Dimensional, categorical, and prototypal approaches to diagnosing eating disorders: An empirical examination*. Unpublished manuscript, Emory University.

Namy, L. L., & Gentner, D. (2002). Making a

silk purse out of two sow's ears: Young children's use of comparison in category learning. *Journal of Experimental Psychology General*, *131*(1), 5–15.

Niedenthal, P. M., & Mordkoff, J. T. (1991). Prototype distancing: A strategy for choosing among threatening situations. *Personality and Social Psychology Bulletin*, *17*(5), 483.

Oldham, J. M., & Skodol, A. E. (2000). Charting the future of Axis II: Critical issues in the classification of personality disorder. Part I. *Journal of Personality Disorders*, *14*(1), 17–29.

Olfson, M., Weissman, M. M., Leon, A. C., Farber, L., & Sheehan, D. V. (1996). Psychotic symptoms in primary care. *Journal of Family Practice*, *43*(5), 481–488.

Oquendo, M., Brent, D. A., Birmaher, B., Greenhill, L., Kolko, D., Stanley, B., et al. (2005). Posttraumatic stress disorder comorbid with major depression: Factors mediating the association with suicidal behavior. *American Journal of Psychiatry*, *162*(3), 560–566.

Pope, H. G., Jr., Lalonde, J. K., Pindyck, L. J., Walsh, T., Bulik, C. M., Crow, S. J., et al. (2006). Binge eating disorder: A stable syndrome. *American Journal of Psychiatry*, *163*(12), 2181–2183.

Rosch, E. (1978). Principles of categorization. In E. Rosch & B. Lloyd (Eds.), *Cognition and categorization* (pp. 27–48). Hillsdale, NJ: Erlbaum.

Rosch, E., & Lloyd, B. B. (Eds.). (1978). *Cognition and categorization*. Hillsdale, NJ: Erlbaum.

Rosch, E., & Mervis, C. B. (1975). Family resemblances: Studies in the internal structure of categories. *Cognitive Psychology*, *7*(4), 573–605.

Rosch, E., & Muller, S. (1978). Classification judgments: Restrictive conditions for the explanation of stereotypes. *Zeitschrift für Sozialpsychologie*, *9*, 246–256.

Rosen, G. M., & Lilienfeld, S. O. (2008). Posttraumatic stress disorder: An empirical evaluation of core assumptions. *Clinical Psychology Review*, *28*(5), 837–868.

Rosen, G. M., Spitzer, R. L., & McHugh, P. R. (2008). Problems with the post-traumatic stress disorder diagnosis and its future in DSM-V. *British Journal of Psychiatry*, *192*(1), 3–4.

Rothbaum, B. O., & Davidson, J. R. (n.d.). *The Traumatic Events Inventory manual*. Unpublished manual.

Rottman, B., Ahn, W., Sanislow, C., & Kim, N. (2009). Can clinicians recognize DSM-IV personality disorders from five-factor model descriptions of patient cases? *American Journal of Psychiatry, 166*, 1–7.

Russ, E., Shedler, J., Bradley, R., & Westen, D. (2008). Refining the construct of narcissistic personality disorder: Diagnostic criteria and subtypes. *American Journal of Psychiatry*, *165*(11), 1473–1481.

Russell, J. A. (2003). Core affect and the psychological construction of emotion. *Psychological Review*, *110*(1), 145–172.

Rutherford, M. D., & McIntosh, D. N. (2007). Rules versus prototype matching: Strategies of perception of emotional facial expressions in the autism spectrum. *Journal of Autism and Developmental Disorders*, *37*(2), 187–196.

Schmidt, N. B., Kotov, R., & Joiner, T. E. (2004). *Taxometrics toward a new diagnostic scheme for psychopathology*. Washington, DC: American Psychological Association.

Schwartz, A. C., Bradley, R., Penza, K. M., Sexton, M., Jay, D., Haggard, P. J., et al. (2006). Pain medication use among patients with posttraumatic stress disorder. *Psychosomatics*, *47*, 136–142.

Shedler, J., & Westen, D. (2004). Dimensions of personality pathology: An alternative to the five-factor model. *American Journal of Psychiatry*, *161*(10), 1743–1754.

Shedler, J., & Westen, D. (2007). The Shedler–Westen Assessment Procedure (SWAP): Making personality diagnosis clinically meaningful. *Journal of Personality Assessment*, *89*(1), 41–55.

Simon, H. A. (1978). Information-processing theory of human problem solving. In W. K. Estes (Ed.), *Handbook of learning and cognitive processes* (pp. 271–295). Hillsdale, NJ: Erlbaum.

Spitzer, R. L., Endicott, J., & Robins, E. (1978). Research Diagnostic Criteria: Rationale and reliability. *Archives of General Psychiatry*, *35*, 773–782.

Spitzer, R. L., First, M. B., Shedler, J., Westen, D., & Skodol, A. E. (2008). Clinical utility of 5-dimensional systems for personality diagnosis: A "consumer preference" study. *Journal of Nervous and Mental Disease*, *196*(5), 356–374.

Tellegen, A., & Waller, N. G. (1992). *Exploring personality through test construction: Development of the Multidimensional Personality Questionnaire (MPQ)*. Unpublished manuscript, University of Minnesota.

Thompson-Brenner, H., Eddy, K. T., Satir, D. A., Boisseau, C. L., & Westen, D. (2008). Personality subtypes in adolescents with eating dis-

orders: Validation of a classification approach. *Journal of Child Psychology and Psychiatry*, *49*(2), 170–180.

Tsuang, M. T., Stone, W. S., & Faraone, S. V. (2000). Towards reformulating the diagnosis of schizophrenia. *American Journal of Psychiatry*, *157*, 1041–1050.

Tversky, A., & Kahneman, D. (1974). Judgment under uncertainty: Heuristics and biases. *Science*, *185*, 1124–1131.

Van der Does, A. J. W., Linszen, D. H., Dingemans, P. M., Nugter, M. A., & Scholte, W. (1993). A dimensional and categorical approach to the symptomatology of recent-onset schizophrenia. *Journal of Nervous and Mental Disease*, *181*(12), 744–749.

Weber, M. (1949). *The methodology of the social sciences*. Glencoe, IL: Free Press.

Westen, D. (1997). Divergences between clinical and research methods for assessing personality disorders: Implications for research and the evolution of Axis II. *American Journal of Psychiatry*, *154*, 895–903.

Westen, D. (1998). Case formulation and personality diagnosis: Two processes or one? In J. Barron (Ed.), *Making diagnosis meaningful* (pp. 111–138). Washington, DC: American Psychological Association.

Westen, D., & Arkowitz-Westen, L. (1998). Limitations of Axis II in diagnosing personality pathology in clinical practice. *American Journal of Psychiatry*, *155*, 1767–1771.

Westen, D., & Bradley, R. (2005). Prototype diagnosis of personality. In S. Strack (Ed.), *Handbook of personology and psychopathology* (pp. 238–256). Hoboken, NJ: Wiley.

Westen, D., & Harnden-Fischer, J. (2001). Personality profiles in eating disorders: Rethinking the distinction between Axis I and Axis II. *American Journal of Psychiatry*, *165*, 547–562.

Westen, D., Heim, A., Morrison, K., Patterson, M., & Campbell, L. (2002). Simplifying diagnosis using a prototype-matching approach: Implications for the next edition of the DSM. In L. E. Beutler & M. L. Malik (Eds.), *Rethinking the DSM: A psychological perspective* (pp. 221–250). Washington, DC: American Psychological Association.

Westen, D., & Muderrisoglu, S. (2003). Reliability and validity of personality disorder assessment using a systematic clinical interview: Evaluating an alternative to structured interviews. *Journal of Personality Disorders*, *17*, 350–368.

Westen, D., & Muderrisoglu, S. (2006). Clinical assessment of pathological personality traits. *American Journal of Psychiatry*, *163*, 1285–1297.

Westen, D., & Shedler, J. (1999). Revising and assessing Axis II, Part 1: Developing a clinically and empirically valid assessment method. *American Journal of Psychiatry*, *156*, 258–272.

Westen, D., & Shedler, J. (2000). A prototype matching approach to diagnosing personality disorders: Toward DSM-V. *Journal of Personality Disorders*, *14*(2), 109–126.

Westen, D., Shedler, J., & Bradley, R. (2006). A prototype approach to personality diagnosis. *American Journal of Psychiatry*, *163*, 846–856.

Westen, D., Shedler, J., Durrett, C., Glass, S., & Martens, A. (2003). Personality diagnosis in adolescence: DSM-IV Axis II diagnoses and an empirically derived alternative. *American Journal of Psychiatry*, *160*, 952–966.

Widiger, T. A. (2007). Alternatives to DSM-IV: Axis II. In W. T. O'Donohue, K. A. Fowler, & S. O. Lilienfeld (Eds.), *Personality disorders: Toward the DSM-V* (pp. 21–40). Thousand Oaks, CA: Sage.

Widiger, T. A., & Clark, L. A. (1999). Toward DSM-V and the classification of psychopathology. *Psychological Bulletin*, *126*, 947–963.

Widiger, T. A., & Frances, A. (1985). The DSM-III personality disorders: Perspectives from psychology. *Archives of General Psychiatry*, *42*, 615–623.

Widiger, T. A., & Sanderson, C. J. (1995). Toward a dimensional model of personality disorders. In W. J. Livesley (Ed.), *The DSM-IV personality disorders* (pp. 433–458). New York: Guilford Press.

Wittgenstein, L. (1953). *Philosophical investigations*. Oxford, UK: Blackwell.

Zinbarg, R. E., Barlow, D. H., Liebowitz, M., Street, L., Broadhead, E., Katon, W., et al. (1994). The DSM-IV field trial for mixed anxiety–depression. *American Journal of Psychiatry*, *151*(8), 1153–1162.

Zlotnick, C. (1997). Posttraumatic stress disorder (PTSD), PTSD comorbidity, and childhood abuse among incarcerated women. *Journal of Nervous and Mental Disease*, *185*(12), 761–763.

CHAPTER 21

The Millon Personality Spectrometer

A Tool for Personality Spectrum Analyses, Diagnoses, and Treatments

THEODORE MILLON
SETH GROSSMAN
ROBERT TRINGONE

Efforts have been made to shift diagnostic procedures from the neo-Kraepelinian categorical structural model to one that may capture a more quantitative and dimensional picture of a person. Popular five-factor models (e.g., Costa & McCrae, 1992, 1995) address this issue by describing personality traits from an inductive statistical process (Davis & Millon, 1993); by contrast, we turn in this chapter to a well-researched prototypal evolutionary model (Millon, 1990, 1996) and its functional and structural attributes. There is a need to access these clinical domains efficiently and directly from the perspective of the treating clinician. This chapter begins with a brief review of developments that have led to the creation of the Millon Personality Spectrometer (MPS).

Historical Perspective

There has been a renewed interest in recent decades in the area of personology, as well as in using assessment for therapy planning. The most significant catalyst was the development of DSM-III (American Psychiatric Association, 1980), with its multiaxial format, the separate placement of the personality disorders (PDs) on their own axis, and the specification of diagnostic criteria. These changes marked a "paradigm shift" in the approach to classification and psychodiagnosis (Klerman, 1986; Millon, 1986a). Whereas DSM-I and DSM-II employed a "classic" approach to psychodiagnosis, DSM-III utilized a prototypal typology that recognized the diagnostic syndromes' intrinsic heterogeneity, due to the probabilistic nature and varied character of their diagnostic features.

Wittgenstein (1953) is credited with recognizing the inherent ambiguity and multidimensionality involved in categorization. Researchers extrapolated from his notions of the multiplicity of "language games" and applied them to the PDs. DSM-III (American Psychiatric Association, 1980) represented substantial progress toward this end in its development of a prototypal typology (Frances, 1980; Millon, 1990). However, this advance was not complete (Frances, 1982; Frances & Widiger, 1986). For example, some of the DSM-III Axis II PDs were conceptualized within the prototypal typology, while others required the presence of all their diagnostic criteria within a multiple-choice format. Still other PD categories represented DSM-III "holdouts"; that is, under the clas-

sic monothetic format, all diagnostic criteria had to be present to meet a diagnosis. In DSM-III-R (American Psychiatric Association, 1987), DSM-IV (American Psychiatric Association, 1994), and DSM-IV-TR (American Psychiatric Association, 2000), all Axis II PDs have been conceptualized in line with a polythetic, prototypal typology.

Debates have raged and continue to abound concerning the shift from the classic model to a prototypal model, and now to the current consideration of dimensional models (Widiger & Trull, 2007). Historically, psychology and psychiatry have tended to align their views of diagnosis with those of medicine and biology (Garfield, 1986), in which categories possess clear demarcations indicative of distinct entities. Membership is determined by the presence of "necessary and sufficient" characteristics that differentiate and place persons into homogeneous categories. Advantages of the classic perspective include the ease and convenience with which pertinent information can be communicated. Well-established categories are highlighted by a set of the most salient characteristics, and they provide a standard reference for clinicians (Millon, 1991). The failure to meet the assumptions of monothetic criteria and homogeneous group membership, however, argues against this position (Cantor, Smith, French, & Mezzich, 1980). Also, no available studies have empirically determined the thresholds or cutoff points that make a clear distinction between the presence and absence of a PD (Widiger, 1992). In addition, Frances and Widiger (1986) noted that attempts to delineate restrictive diagnostic criteria in an effort to increase the sameness of members in a category have led to an increase in the number of "wastebasket" categories. Ironically, the DSM-III-R Axis II revisions led to dramatic increases in the prevalence rates of the PDs as well as in their comorbidity (Morey, 1988).

The prototypal model allows that instances within categories may display quantitative as well as qualitative differences. The critical contribution of the prototypal model is the assumption of probabilistic features, which demands greater flexibility in determining diagnostic categorization. There is a systematic deemphasis on the presence of "necessary and sufficient" characteristics; hence instances are viewed more along a continuum of similarity to prototypes, or "goodness of fit." Attributes serve as correlated indicators of disorders and carry varying degrees of diagnostic efficiency and validity (Clarkin, Widiger, Frances, Hurt, & Gilmore, 1983; Widiger, Hurt, Frances, Clarkin, & Gilmore, 1984; Widiger & Trull, 2007). Within the prototypal model, category members are not likely to meet all inclusive and exclusive criteria of a category. Therefore, one anticipates heterogeneity within a disorder, numerous ambiguous or "atypical" cases, and varying degrees of similarity to the standard of comparison (e.g., a DSM-IV[-TR] PD). Other than the issue of within-group heterogeneity, the prototypal model allows for the overlap of categories. Although it has been argued that categories may then no longer be very distinctive, the very nature of personality is one of overlap and covariation between personalities. Research utilizing early forms of the MPS, to be reviewed shortly, has provided considerable support for the prototypal model (Millon & Tringone, 1989; Tringone, 1990).

Two variants were proposed within a prototypal typology: summary prototypes and exemplar prototypes (Cantor & Genero, 1986). The most relevant examples of summary prototypes are the DSM-IV(-TR) diagnostic criteria sets, whereas exemplar prototypes emphasize the use of multiple examples for any category. The difference lies in the latter's reliance on known instances or "exemplars" of a category, rather than on the abstract image of a cognitive structure (Cantor & Genero, 1986). Millon's writings on PD subtypes (e.g., Millon, 1996) provide theoretically derived exemplars within each PD category. For example, a person who has primary narcissistic PD may also possess salient features of histrionic PD (amorous narcissism), antisocial PD (unprincipled narcissism), or paranoid PD (elitist narcissism).

A common counterargument to the classic position states that persons cannot be divided into homogeneous, discrete units (Frances, 1980). In an effort to alleviate the qualitative problems of the classic model, numerous dimensional perspectives have recently been proposed and have generated considerable interest. Viewing characteristics and disorders along a continuum of severity from normal to pathological, a spectrum model emphasizes quantitative gradations

rather than qualitative, all-or-none distinctions (Skinner, 1986; Millon, 1987b, 1991). Such latitude allows an individual to possess certain features indicative of various disorders in matters of degree. Though they are perceived to be in opposition, classic and dimensional approaches complement one another (Frances, 1982; Millon, 1987b, 1991; Millon & Bloom, 2008). Whereas dimensions account for quantitative differences for criteria, personality constructs can be perceived as qualitative sets of attributes. Dimensional scores can also be translated into categorical diagnoses through the use of cutoff points.

Classification systems that employ dimensions are viewed as more flexible and informative than the classic perspective (Widiger, 1982; Widiger & Trull, 2007). Also, they are better able to classify ambiguous cases than is a "forced-choice" paradigm. Their utility may be lessened, however, when descriptions become too complex and unwieldy (Frances, 1982). Other noted problems involve defining the core dimensions, agreeing on the number of core dimensions needed to represent the personality disorders, and identifying the meaningfulness of increments within the chosen dimensions (Millon, 1991).

With the shift from the classic perspective to the prototypal view, there has been a dramatic increase in the number of studies investigating the PDs. While we are progressing in our empirical understanding of these disorders, advances must still be made in our theoretical understanding of their origins, their self-perpetuating tendencies, and the ways clinicians must intervene and treat them (Millon, 1969, 1981, 1990, 1991, 1996).

The Millon Personality Diagnostic Checklist

Before we introduce the MPS, it will be useful to examine its forerunners, in which much of its methodology was developed. The Millon Personality Diagnostic Checklist (MPDC; Millon & Tringone, 1989; Tringone, 1990, 1997) was constructed initially to assist in the validation of the Millon Clinical Multiaxial Inventory–II (MCMI-II; Millon, 1987a). This was a time of transition in psychological diagnosis and categorization. No "gold standard" was available as a comparison point for the PDs; clinicians were therefore asked to complete the MPDC and to provide up to three PD diagnoses, listed in order of salience, while their patients completed the MCMI-II research form. Clinician assessments helped set diagnostic prevalence rates, as well as the presence and prominence levels of the MCMI-II personality scales. Concordance rates were then generated between the clinicians' diagnostic impressions and the self-report inventory. As part of this process, the diagnostic efficiency of each MPDC item across all the PDs was then calculated to investigate the MPDC's strength and utility in the diagnostic process, as well as its potential as a stand-alone clinician's checklist (Millon & Tringone, 1989; Tringone, 1990).

Like that of all Millon inventories, the development of the MPDC has followed the validation sequence proposed by Loevinger (1957) and Jackson (1970). In her classic monograph, Loevinger delineated three components of construct validity: substantive, structural, and external. She suggested that these components are construction and validation stages that can be followed sequentially. Her schema incorporates the conceptualizations of Cronbach and Meehl (1955) on construct validity, and of Campbell and Fiske (1959) on convergent and discriminant validity. The intent of following this model was to enhance the MPDC's reliability and validity, and to maximize its efficiency in assessing personality characteristics. A brief review of the first stage of development is offered here.

For the first, substantive stage (relabeled "theoretical/substantive"), Millon's (1969, 1981, 1986a, 1986b) biosocial learning theory served as the underlying theoretical model for the MPDC's conceptualization of the DSM-III-R and DSM-IV PDs. This model proposed the relationships of the different constructs to one another. Furthermore, it proposed prototypal functional and structural features of each PD within common domains. Two steps were involved in compiling and developing items to meet this requirement with the MPDC: (1) creating an initial item pool based on theoretical and empirical grounds, and (2) reducing the initial item pool on empirical and rational grounds. These items, representing the 13

proposed DSM-III-R PDs, were ultimately derived from Millon's theory as well as from DSM-III diagnostic criteria, both of which were viewed as having theoretical and empirical support for defining PDs.

Content validity for a classification system is achieved when all of its categories are defined across the full range of clinically relevant domains. The criteria sets for the DSM-III-R and DSM-IV PDs were deemed both "noncomprehensive" and "noncomparable" (Millon, 1996). Some of the constructs' criteria are narrow and restricted to behavior-oriented features, while other constructs' criteria are redundant and reiterate a single theme across multiple criteria. Shea (1992) also noted that the criteria sets vary in the number of underlying dimensions they address and the level of inference required to assess the criteria, with the latter issue amplified in regard to whether or not the underlying motivations of the manifest behaviors have been made explicit. This point is especially important because similar behaviors can have different determinants, and different behaviors can have similar determinants (Stricker & Gold, 1988).

By the development period of the MPDC, Millon (1986b, 1990) had outlined defining features for each PD across eight domain areas. Those features manifested between a person and his or her environment were labeled "functional domains." These domains represented the "behaviors, interpersonal conduct, cognitive processes, and unconscious mechanisms which manage, adjust, transform, coordinate, balance, discharge, and control the give and take of inner and outer life" (Millon, 1990, p. 136). Three functional domains were incorporated into the MPDC: Expressive Acts, Interpersonal Conduct, and Cognitive Style. A second group of clinically relevant characteristics were labeled "structural domains," which represented "a deeply embedded and relatively enduring template of imprinted memories, attitudes, needs, fears, conflicts, and so on, which guide experience and transform the nature of ongoing life events" (Millon, 1990, p. 147). The structural domains assessed with the MPDC were Self-Image and Mood/Temperament. These five domains were selected because they were considered to be generally more objective and to require less inference on the part of clinicians than the three remaining domains, recently relabeled Intrapsychic Dynamics, Intrapsychic Content, and Intrapsychic Structure.

Further stages of development of the MPDC indicated that the instrument exhibited reasonably strong internal/structural reliability as well as external validity when correlated with Millon's theoretical constructs (Tringone, 1997). It was proven to be of great value in assisting the development of the MCMI-II; it was further thought, owing to the continuity of constructs between the MCMI-II and MCMI-III (Millon, 2006b), that its general structure and content woubd continue to serve well as a clinician's checklist companion to the more recent MCMI-III. It was with this background—that of providing an additional, coordinated source of data in assessment—that Millon and his colleagues began development of a new version of the MPDC.

The Millon–Grossman Personality Domains Checklist

Clinicians and researchers need multiple sources of data for accurate assessments of individuals. These sources range from incidental to well-structured observations, casual to highly systematic interviews, and cursory to formal analyses of biographical history; also employed are various laboratory tests, self-report inventories, and performance-based or projective techniques. All of these have proven to be useful grounds for diagnostic study. In this context, several key questions come to light, most notably the following: How do we put these diverse data sources together to systematize and quantify the information we have gathered? It was toward the end of organizing and maximizing the therapeutic utility of our personality findings that the Millon–Grossman Personality Domains Checklist (MG-PDC) was developed. (Also at this time, Robert Tringone, who had participated actively in the development of the MPDC, moved forward in his career; he was replaced by Seth Grossman as Millon's primary research associate on the checklist project, helping to give shape and direction to what was to be renamed the MG-PDC.)

Whether assessment tools are based on empirical investigations, epidemiological research, mathematical analyses, or theo-

retical deductions, they often fail to characterize persons in the language and concepts traditionally employed by therapy-oriented psychopathologists. Although many instruments have proven of value in numerous research studies, such as demonstrating reasonable intercorrelations or a correspondence with established diagnostic systems (e.g., DSM), many an astute therapist has questioned whether these tools yield anything beyond the reliability of surface impressions. Some (Westen & Weinberger, 2004) doubt whether self-report instruments, for example, successfully tap into or unravel the diverse, complex, and hidden relationships among difficult-to-fathom processes. Other critics have contended that patient-generated responses may contain *no* clinically relevant information beyond the judgments of nonscientists employing the vocabulary of a layperson's lexicon.

Data obtained from patient-based self-judgments may be contrasted with the sophisticated clinical appraisals of therapy-oriented professionals. We must ask whether clinical language, concepts, and instruments encoded in the evolving professional language of the past 100 years or so generate information incremental to the naive descriptions of an ordinary person's everyday lexicon. We know that therapeutic languages differ from laypersons' languages because they serve different and more sophisticated purposes (Livesley, Jackson, & Schroeder, 1989). Indeed, therapy concepts reflect the experienced contributions of numerous historical schools of thought (Millon, 2004). All of these therapy-oriented schools (e.g., psychodynamic, cognitive, interpersonal) have identified a multitude of diverse and complex psychic processes that operate in our mental lives. Surely the concepts of these historical professional lexicons are not reducible to the superficial factors drawn from the everyday vocabulary of nontherapists.

In large part, the accurate representation and integration of the insights and concepts of the several major schools of thought led first to the formulation of the MPDC, a domain-based, clinician-rated assessment (Millon, 1969, 1981, 1984, 1986a, 1990, 1996; Millon & Tringone, 1989; Tringone, 1990, 1997); they then led to the development, following numerous empirical and theoretical refinements, of the MG-PDC. In contrast with the five-factor method, popular among research-oriented psychologists, the MG-PDC continued basing its primary measures on the contributions of five major therapeutic traditions: the behavioral, the interpersonal, the self, the cognitive, and the biological. As in the MPDC, three domains were also included in the MG-PDC to reflect the psychoanalytic tradition; however, because the use of these domains was diminishing, they were made optional (not required) components of the new instrument.

Several criteria were used to select and develop the therapeutic domains included in the MG-PDC:

1. The domains were broad-based and varied in the features they embodied; that is, they were not limited to biological temperaments or cognitive processes, but instead encompassed a full range of personality characteristics based on frequently used therapeutic terms and concepts.
2. They corresponded to the major therapeutic modalities employed by contemporary mental health professionals to treat their patients (e.g., cognitive techniques for altering dysfunctional beliefs, group procedures for modifying interpersonal conduct), and hence could be readily employed by practicing therapeutic clinicians.
3. They were coordinated with and reflected the official PD prototypes established by the *International Classification of Diseases*, 10th revision (ICD-10; World Health Organization, 1992) and DSM-IV(-TR), and thereby could be understood by insurance and other management professionals.
4. A distinctive psychological trait could be identified and operationalized in each of the clinical trait domains for each personality prototype, assuring thereby both scope and comparability among personological criteria.
5. They lent themselves to the appraisal of domain characteristics for both normal and abnormal personalities, and hence would promote further advances in the field of normality—one of growing interest in the psychological literature.
6. They could constitute an educational therapeutic tool to sensitize mental health workers in training (psychologists, psy-

chiatrists, clinical social workers, etc.) to the many distinctions, subtleties, and domain interactions that are worth considering in appraising personality attributes.

The *integrative perspective* that the MG-PDC encouraged views personalities as a multidetermined and multireferential construct. One (albeit problematic) approach taken by some clinical researchers to dealing with the conceptual alternatives that characterize personality study is to oversimplify the task. They choose to assess each patient in accord with a single conceptual orientation, eliminating thereby the integration of divergent perspectives by an act of regressive dogmatism. A truly effective assessment, however—one that is logically consonant with the modern integrative character of personality, both as a construct and as a reality—requires that the individual be assessed systematically across multiple characterological domains. This approach ensures that the assessment is comprehensive, useful to a broad range of therapists, and more likely to be valid. In assessing with the MG-PDC, therapists would refrain, therefore, from regarding each domain as an independent entity and thereby falling into a naive, single-minded treatment approach. Each of the domains was a legitimate but highly contextualized part of a unified or integrated whole, a necessary composite ensuring that the full integrity of the person would be represented. It was with these ideas in mind also that the MG-PDC (and in turn the MPS) would be employed as a supplement to the Millon Adolescent Clinical Inventory (MACI; Millon, 2006a) and MCMI-III (Millon, 2006b); in these inventories, the domains were conceived as scale subcomponents in constructing the Grossman Facet Scales.

As noted previously in regard to the MPDC, the domains of the MG-PDC could be organized in a manner similar to distinctions drawn in the biological realm; that is, they could be divided into and characterized as "structural" and "functional" attributes. The functional domains of the instrument represented dynamic processes transpiring between an individual and his or her psychosocial environment. Such transactions take place through what we have termed the person's "modes of regulatory action"—that is, his or her demeanor, social relations, and thought processes, each of which serve to manage, adjust, transform, coordinate, and control the give-and-take of inner and outer life. Several functional domains relevant to each personality were included among the major components of the MG-PDC.

In contrast to the functional characteristics, the structural domains of the MG-PDC represented templates of deeply embedded affect dispositions and imprinted memories, attitudes, needs, and conflicts that guide experience and orient ongoing life events. These domains could be conceived of as quasi-permanent substrates for identity and temperament. Such residues of the past and relatively enduring affects effectively constrain and even close off innovative learnings, and limit new possibilities to already established habits and dispositions. Their persistent and preemptive character perpetuates the maladaptive behavior and vicious circles of a patient's extant personality pathology.

Of course, individuals differ with respect to the domains they enact most frequently. People vary not only in the degree to which they approximate each personality prototype, but also in the extent to which each domain dominates their behavior. In conceptualizing personality as a system, we must recognize that different parts of the system will be dominant in different individuals, even when those individuals are patients who share the same prototypal diagnosis. It was the goal of the MG-PDC to differentiate, operationalize, and measure quantitatively those domain features that would be primary in contributing to a therapist's planning. It was thus hoped that the instrument would guide the clinical therapist's efforts to modify the person's problematic features (e.g., interpersonal conduct, cognitive beliefs), and thereby enable the patient to acquire a greater variety of adaptive behaviors in his or her life circumstances.

The Millon Personality Spectrometer

In this section, we introduce the MPS—the third instrument developed in our test construction series, and the first one to emerge from spectrum elaborations of the theory. First, we summarize the bipolar components

of the evolutionary theory undergirding the MPS. Second, we summarize the 15 personality spectra, each spectrum extending from the normal personality type to the pathological PD. Then we present fully the eight clinical domains (interpersonal, cognitive), each differentiated into the 15 trait dimensions of which the domains are composed; each dimensional trait corresponds to one of the 15 personality spectra.

The three polarities derived from evolutionary theory are described briefly below (and in greater detail in Millon, Chapter 23, this volume). They serve in various combinations as the basis for the 15 spectra.

Existential Aims: The Pleasure–Pain Polarity

The most basic of all evolutionary objectives, that of existence, has a twofold aspect. The first is the enhancement or enrichment of life—that is, creating or strengthening ecologically survivable organisms. The second is the preservation of life—that is, creating survivability and security by avoiding events that might terminate it. Among humans, the former may be seen in life-enhancing acts that enrich existence by what are experientially recorded as "pleasurable" events (positive reinforcers); the latter may be seen in life-preserving behaviors oriented to achieve security by repelling or avoiding events that are experientially characterized as "painful" (negative reinforcers). The pleasure–pain bipolarity not only places sensations, motivations, feelings, emotions, moods, and affects on two contrasting dimensions, but recognizes that each possesses separate and independent quantitative gradations (Watson & Tellegen, 1985). In other words, events that are attractive, gratifying, rewarding, or positively reinforcing may be experienced as either weak or strong, as can those that are aversive, distressful, sad, or negatively reinforcing .

Adaptive Modes: The Passive–Active Polarity

The second basic polar distinction relates to what we have termed the modes of adaptation; it is also framed as a two-part polarity. One may best be characterized as the mode of "ecological accommodation." This signifies inclinations to passively "fit in," to locate and remain securely anchored in a niche, subject to the vagaries and unpredictabilities of the environment—all of which are acceded to with one crucial proviso: that the elements of the surroundings will furnish both the nourishment and the protection needed to sustain existence. Though the concept is based on a somewhat simplistic bifurcation among adaptive strategies, this passive, accommodating mode is one of the two fundamental methods that living organisms have evolved as a means of survival. It represents the core process employed in the evolution of what has come to be designated as the plant kingdom—a stationary, rooted, yet essentially pliant and dependent survival mode. By contrast, the other of the two major modes of adaptation is seen in the lifestyle of the animal kingdom. Here we observe a primary inclination toward "ecological modification." We define this as an *active* tendency to change or rearrange the elements constituting the larger milieu, to intrude upon otherwise quiescent settings—a versatility in shifting from one niche to another as unpredictability arises. It is a mobile and interventional mode that actively stirs, maneuvers, yields, and, at the human level, substantially transforms the environment to meet its own survival aims (Rapaport, 1953).

Both modes—passive and active—have proven impressively capable of nourishing and preserving life. Whether the polarity sketched is phrased in terms of accommodating versus modifying, passivity versus activity, or plant versus animal, it represents, at the most basic level, the two fundamental modes that complex organisms have evolved to sustain their existence. The second, accommodating–modifying polarity differs from the first, enhancing–preserving polarity (that concerned with what may be called existential "becoming"), in that it characterizes modes of "being"—in other words, how what has become endures.

Broadening the active–passive polarity model to encompass human experience, we find that the vast range of human behaviors may fundamentally be grouped in terms of whether initiative is taken in altering and shaping life's events, or whether behaviors are reactive to and accommodate those events. Both approaches are useful at times; humans are sometimes actors and at other

times reactors. Moreover, it is our contention that significant individual differences of personality significance are to be found along this passivity–activity dimension. No individual is one or the other, but both in varying proportions—a difference relevant to the classification of personality styles.

Replicatory Strategies: The Self–Other Polarity

The third polarity is less profound than the first polarity, which represents the enhancement of order (existence/life/pleasure) and the prevention of disorder (nonexistence/death/pain), or the second polarity, which differentiates the adaptive mode of accommodation (plant/passive) from that of modification (animal/active). Nevertheless, this third polarity, based on distinctions in reproductive strategies (gene replication), is no less fundamental: It contrasts the maximization of reproductive propagation (male/self) with the maximization of reproductive nurturance (female/other) (Millon, 1990; Trivers, 1974).

What we provide in the following paragraphs is a précis of how the three bipolarities combine and interact in producing the several spectra of normal and abnormal personality (Maser & Akiskal, 2002). These paragraphs briefly describe the 15 different personality spectrum prototypes generated by the theory. The first descriptor for each spectrum indicates the "normal" personality type; the second one indicates the "abnormal" personality, or PD.

A. *The apathetic–schizoid personality spectrum*. On what basis can disturbances in the level or capacity of either the pain and pleasure polarities be seen as relevant to personality types or PDs? Several possibilities present themselves. Persons in the apathetic–schizoid spectrum are those in whom both pleasure and pain polarity objectives are deficient; that is, they lack the capacity, relatively speaking, to experience life's events as either painful or pleasurable. Without these motivations, they are likely to sit passively as life goes by.

B. *The schizotypal–schizophrenic personality spectrum*. The schizotypal–schizophrenic personality spectrum also represents a markedly deficient orientation in the pleasure–pain polarity schema. Individuals with these personalities experience minimal pleasure; they have difficulty in differentiating consistently between self- and other-oriented strategies, as well as utilizing effectively either active or passive modes of adaptive functioning. Many regress into eccentricity and social isolation, with minimal personal attachments and obligations.

C. *The withdrawn–avoidant personality spectrum*. Another clinically meaningful combination deriving from problems in the pleasure–pain polarity describes patients with a diminished ability to experience pleasure, while at the same time possessing an unusual sensitivity and responsiveness to psychic pain. To them, life is vexatious, possessing few rewards and much anguish. Hence they are hyperalert and actively seek to avoid the anticipation of pain.

D. *The attached–dependent personality spectrum*. Those in the attached–dependent spectrum have learned that feeling good, secure, and confident—that is, having feelings associated with pleasure and the avoidance of pain—calls for a passive reliance on the goodwill of others. These persons become strongly bonded and display a strong need for interpersonal support and attention. Should they be deprived of social affection and nurturance, they are likely to experience marked discomfort, perhaps even sadness and anxiety.

E. *The exuberant–turbulent personality spectrum*. Individuals in this group are unusual by virtue of the central role they give to their active pursuit of the pleasurable side of the pain–pleasure polarity. Typically energetic and buoyant in manner, they may become overly animated, scattered, and manic.

F. *The sociable–histrionic personality spectrum*. Turning to others as their primary strategy, as do those with attached–dependent personalities, individuals with sociable–histrionic personalities take an active dependency stance. They achieve their goal of maximizing protection, nurturance, and reproductive success by engaging busily in a series of manipulative, seductive, gregarious, and attention-getting maneuvers with others.

G. *The confident–narcissistic personality spectrum*. Patients falling into the confident–narcissistic personality spectrum exhibit a

primary reliance on self rather than others. They have learned that maximum pleasure and minimum pain is achieved by minimizing the significance of others and turning passively to a naive but high validation of self.

H. *The paranoid–paraphrenic personality spectrum*. Here are seen a vigilant mistrust of others and an edgy defensiveness against anticipated criticism and deception. Driven by a high sensitivity to pain (rejection/humiliation), and oriented strongly to the self polarity, these patients exhibit a touchy irritability. They need to assert themselves, not necessarily in action, but in an inner world of self-determined beliefs and assumptions.

I. *The nonconforming–antisocial personality spectrum*. Individuals in this spectrum, especially toward the abnormal end, exhibit the outlook, temperament, and socially unacceptable behaviors characteristic of DSM-defined antisocial PD. They act to counter the expectation of pain at the hands of others; this is done by actively engaging in duplicitous or illegal behaviors in which they seek to exploit others for self-gain. Skeptical regarding the motives of others, they desire autonomy, and wish revenge for what are felt as past injustices.

J. *The assertive–sadistic personality spectrum*. There are patients in whom the usual properties associated with pain and pleasure are conflicted or reversed. Like those in the aggrieved–masochistic spectrum, to be described shortly, patients in the assertive–sadistic spectrum not only create objectively painful events, but experience them as pleasurable. This variant of pleasure–pain reversal considers pain (stress, fear, cruelty) rather than pleasure to be the preferred mode of relating actively to others.

K. *The doleful–melancholic personality spectrum*. Chronic feelings of sadness and depression are typical of these persons, who persist, despite periods of objective good fortune, in being downhearted and gloomy. Oriented to the *pain* polarity, they characteristically behave in a passive, "giving-up" manner.

L. *The aggrieved–masochistic personality spectrum*. Like the assertive–sadistic spectrum, this troubled spectrum stems largely from a reversal of the pleasure–pain polarity. These patients engage in relationships that are at variance with this normal polarity balance. To those in this personality spectrum, pain may have become a preferred experience, passively accepted if not encouraged in intimate relationships.

M. *The resentful–negativistic personality spectrum*. Persons in the resentful–negativistic spectrum are oriented toward both self and others, but there is an intense conflict between the two. A number of these patients (originally represented in DSM as having passive–aggressive PD) vacillate between giving primacy one time to others and then to self the next, behaving obediently one time and reacting defiantly the next. Unable to resolve their ambivalence, they weave an actively erratic course.

N. *The borderline–cyclophrenic personality spectrum*. This personality spectrum corresponds to the theory's emotionally dysfunctional and maladaptively ambivalent polarity orientation. Conflicts exist across the board, in all three polarities—pleasure and pain, active and passive, self and other. They seem unable to take a consistent, neutral, or balanced position between the extremes of any polarity, tending to fluctuate from one end to the other. These persons experience intense endogenous moods, with recurring periods of dejection and apathy, often interspersed with spells of anger, anxiety, or euphoria.

O. *The compliant–compulsive personality spectrum*. The compliant–compulsive personality spectrum displays a picture of distinct other-directedness, a consistency in social compliance and interpersonal respect. Their histories usually indicate having been subjected to constraint and discipline, parental strictures, and high expectations. Beneath an overtly passive veneer, they experience intense desires to rebel and assert an underlying and covert self-oriented feeling and impulse. Trapped in their ambivalence, they are often unable to make decisions or act.

Using the MPS

In Tables 21.2–21.9 below, you, our readers, will see 15 descriptive trait choices each. Start with Table 21.2 (for the Expressive Behavior domain), and locate the descriptive choice that appears to you to *fit best* in

characterizing a patient you may be thinking about. Circle that choice in the "1st best fit" column. Then, because most people can be characterized by more than one set of expressive behavior traits, locate a second-best-fit descriptive characteristic—one not as applicable to this person as the first best fit you have selected, but notable nonetheless. Circle that choice in the "2nd best fit" column. Should there be any other set of descriptive trait features applicable to this person, but less so than the set selected as second best, circle that choice (or choices) in the "3rd best fit" column. You may circle up to three choices in this column. (Note that only one trait description each may be marked in the "1st best fit" and "2nd best fit" columns.).

Consider the following points as you proceed. The 15 descriptive traits for each domain were written to characterize patients. Furthermore, each trait is illustrated with several clinical characteristics and examples. Note that the person you are rating need not display precisely the characteristics that are listed; they need only be the best-fitting of the listed sets of features. It is important to note also that for rating persons without a clinical disorder (i.e., normal individuals who display only minor or mild aspects of a trait), you should nevertheless fully mark the best-fit columns (even though a description may be more seriously clinical than suits the person). In short, *do not* leave any of the best-fit columns blank. Fill them in, in rank order of best fit, even when the features of a trait are only marginally present. After completing ratings for the Expressive Behavior domain (Table 21.2), continue to fill in your choices for the next seven domains, one at a time, using the same procedure for each one.

Before you complete Tables 21.2–21.9, of course, you need to know which personality spectrum corresponds to each of the letters that precede the descriptors. For example, in Table 21.2, note that the letter A precedes the first descriptor, "Impassive." The letter A signifies that this descriptor characterizes the apathetic–schizoid spectrum prototype. Table 21.1 summarizes the spectra and their associated letters (A, B, C, etc.). These letters have been used in our earlier descriptions of the spectra, and they are used consistently in the tables that follow. Again, Tables 21.2 through 21.9 list the 15 trait characteristics for each of the eight different clinical domains.

Now, on the basis of your knowledge of the person you have evaluated, use the domain categories described in the preceding section and listed in Table 21.1 to summarize your judgments by making overall first-, second-, and third-best-fit personality spectrum diagnoses in Table 21.10. If you wish, before you proceed, you may want to go back to review your best choices for the eight domains and *double-circle* the three that you judge most important to be therapeutically modified.

Finally, we would like you to further evaluate the person you have just rated. In Table 21.11, please assess his or her current overall level of social and occupational functioning. Make your judgment on the 7-point continuum given there, which ranges from "Excellent" to "Markedly impaired." Focus your rating on the individual's present mental state and social competencies, overlooking (where possible) physical impairments or socioeconomic considerations. Circle the number in the second column that most closely approximates your judgment.

(text resumes on page 410)

TABLE 21.1. Summary of the 15 MPS Spectra and Their Letters

A. Apathetic–Schizoid	I. Nonconforming–Antisocial
B. Schizotypal–Schizophrenic	J. Assertive–Sadistic
C. Withdrawn–Avoidant	K. Doleful–Melancholic
D. Attached–Dependent	L. Aggrieved–Masochistic
E. Exuberant–Turbulent	M. Resentful–Negativistic
F. Sociable–Histrionic	N. Borderline–Cyclophrenic
G. Confident–Narcissistic	O. Compliant–Compulsive
H. Paranoid–Paraphrenic	

TABLE 21.2. MPS: I. Expressive Behavior Domain

These attributes relate to observables at the *behavioral level* of emotion and are usually recorded by noting how the patient acts. Through inference, observations of overt behavior enable us to deduce what the patient unknowingly reveals about his or her emotions or, often conversely, what he or she wants others to think about him or her. The range and character of expressive actions are wide and diverse and they convey distinctive and worthwhile clinical information, from communicating a sense of personal incompetence to exhibiting emotional defensiveness to demonstrating disciplined self-control, and so on.

1st best fit	2nd best fit	3rd best fit	Characteristic behavior
1	2	3	*A. Impassive:* Is colorless, sluggish, displaying deficits in activation and emotional expressiveness; appears to be in a persistent state of low energy and lack of vitality (e.g., phlegmatic and lacking in spontaneity).
1	2	3	*B. Peculiar:* Is perceived by others as eccentric, disposed to behave in an unobtrusively aloof, curious, or bizarre manner; exhibits socially gauche habits and aberrant mannerisms (e.g., manifestly odd or eccentric).
1	2	3	*C. Fretful:* Fearfully scans environment for social derogation; overreacts to innocuous events and judges them to signify personal derision and mockery (e.g., anxiously anticipates ridicule/humiliation).
1	2	3	*D. Incompetent:* Ill-equipped to assume mature and independent roles; is passive and lacking functional competencies, avoiding self-assertion and withdrawing from adult responsibilities (e.g., has difficulty doing things on his or her own).
1	2	3	*E. Impetuous:* Is forcefully energetic and driven, emotionally excitable and overzealous; often worked up, unrestrained, rash, and hot-headed (e.g., is restless and socially intrusive).
1	2	3	*F. Dramatic:* Is histrionically overreactive and stimulus-seeking, resulting in unreflected and theatrical responsiveness; describes penchant for sensational situations and short-sighted hedonism (e.g., overly emotional and artificially affected).
1	2	3	*G. Haughty:* Manifests an air of being above conventional rules of shared social living, viewing them as naive or inapplicable to self; reveals an egocentric indifference to the needs of others (e.g., acts arrogantly self-assured and confident).
1	2	3	*H. Defensive:* Is vigilantly guarded, hyperalert to ward off anticipated deception and malice; is tenaciously resistant to sources of external influence (e.g., disposed to be wary, envious, and jealous).
1	2	3	*I. Impulsive:* Since adolescence, acts thoughtlessly and irresponsibly in social matters; is short-sighted, heedless, incautious, and imprudent, failing to plan ahead or consider legal consequences (e.g., conduct disorder evident before age 15).
1	2	3	*J. Precipitate:* Is stormy and unpredictably abrupt, reckless, thick-skinned, and unflinching, seemingly undeterred by pain; is attracted to challenge, as well as undaunted by punishment (e.g., attracted to risk, danger, and harm).
1	2	3	*K. Disconsolate:* Appearance and posture convey an irrelievably forlorn, heavy-hearted, if not grief-stricken quality; markedly dispirited and discouraged (e.g., somberly seeks others to be protective).
1	2	3	*L. Abstinent:* Presents self as nonindulgent, frugal, and chaste, refraining from exhibiting signs of pleasure or attractiveness; acts in an unpresuming and self-effacing manner, placing self in an inferior light (e.g., undermines own good fortune).
1	2	3	*M. Resentful:* Exhibits inefficiency, erratic, contrary, and irksome behaviors; reveals gratification in undermining the pleasures and expectations of others (e.g., uncooperative, contrary, and stubborn).
1	2	3	*N. Spasmodic:* Displays a desultory energy level with sudden, unexpected self-punitive outbursts; endogenous shifts in emotional state; places behavioral equilibrium in constant jeopardy (e.g., does impulsive, self-damaging acts).
1	2	3	*O. Disciplined:* Maintains a regulated, emotionally restrained, and highly organized life; often insists that others adhere to personally established rules and methods (e.g., meticulous and perfectionistic).

TABLE 21.3. MPS: II. Interpersonal Conduct Domain

A patient's style of relating to others may be captured in a number of ways, such as how his or her actions affect others, intended or otherwise; the attitudes that underlie, prompt, and give shape to these actions; the methods by which he or she engages others to meet his or her needs; and his or her way of coping with social tensions and conflicts. Extrapolating from these observations, the clinician may construct an image of how the patient functions in relation to others.

1st best fit	2nd best fit	3rd best fit	Characteristic conduct
1	2	3	*A. Unengaged:* Is indifferent to the actions or feelings of others, possessing minimal "human" interests; ends up with few close relationships and a limited role in work and family settings (e.g., has few desires or interests).
1	2	3	*B. Secretive:* Strives for privacy, with limited personal attachments and obligations; drifts into increasingly remote and clandestine social activities (e.g., is enigmatic and withdrawn).
1	2	3	*C. Aversive:* Reports extensive history of social anxiety and isolation; seeks social acceptance, but maintains careful distance to avoid anticipated humiliation and derogation (e.g., is socially pan-anxious and fearfully guarded).
1	2	3	*D. Submissive:* Subordinates needs to a stronger and nurturing person, without whom will feel alone and anxiously helpless; is compliant, conciliatory, and self-sacrificing (e.g., generally docile, deferential, and placating).
1	2	3	*E. High-Spirited:* Is unremittingly full of life and socially buoyant; attempts to engage others in an animated, vivacious, and lively manner; often seen by others, however, as intrusive and needlessly insistent (e.g., is persistently overbearing).
1	2	3	*F. Attention-Seeking:* Is self-dramatizing, and actively solicits praise in a showy manner to gain desired attention and approval; manipulates others and is emotionally demanding (e.g., seductively flirtatious and exhibitionistic).
1	2	3	*G. Exploitive:* Acts entitled, self-centered, vain, and unempathic; expects special favors without assuming reciprocal responsibilities; shamelessly takes others for granted and uses them to enhance self and indulge desires (e.g., egocentric and socially inconsiderate).
1	2	3	*H. Provocative:* Displays a quarrelsome, fractious, and distrustful attitude; bears serious grudges and precipitates exasperation by a testing of loyalties and a searching preoccupation with hidden motives (e.g., unjustly questions fidelity of spouse/friend).
1	2	3	*I. Irresponsible:* Is socially untrustworthy and unreliable, intentionally or carelessly failing to meet personal obligations of a marital, parental, employment, or financial nature; actively violates established civil codes through duplicitous or illegal behaviors (e.g., shows active disregard for rights of others).
1	2	3	*J. Abrasive:* Reveals satisfaction in competing with, dominating, and humiliating others; regularly expresses verbally abusive and derisive social commentary, as well as exhibiting harsh, if not physically brutal behavior (e.g., intimidates, coerces, and demeans others).
1	2	3	*K. Defenseless:* Feels and acts vulnerable and guilt-ridden; fears emotional abandonment and seeks public assurances of affection and devotion (e.g., needs supportive relationships to bolster hopeless outlook).
1	2	3	*L. Deferential:* Relates to others in a self-sacrificing, servile, and obsequious manner, allowing, if not encouraging others to exploit or take advantage; is self-abasing, accepting undeserved blame and unjust criticism (e.g., courts others to be exploitive and mistreating).
1	2	3	*M. Contrary:* Assumes conflicting roles in social relationships, shifting from dependent acquiescence to assertive independence; is obstructive toward others, behaving either negatively or erratically (e.g., sulky and argumentative in response to requests).
1	2	3	*N. Paradoxical:* Needing extreme attention and affection, but acts unpredictably and manipulatively and is volatile, frequently eliciting rejection rather than support; reacts to fears of separation and isolation in angry, mercurial, and often self-damaging ways (e.g., is emotionally needy, but interpersonally erratic).
1	2	3	*O. Respectful:* Exhibits unusual adherence to social conventions and proprieties; prefers polite, formal, and "correct" personal relationships (e.g., interpersonally proper and dutiful).

TABLE 21.4. MPS: III. Cognitive Style Domain

How the patient focuses and allocates attention, encodes and processes information, organizes thoughts, makes attributions, and communicates reactions and ideas to others represent key cognitive functions of clinical value. These characteristics are among the most useful indices of the patient's distinctive way of thinking. By synthesizing his or her beliefs and attitudes, it may be possible to identify indications of problematic cognitive functions and assumptions.

1st best fit	2nd best fit	3rd best fit	Characteristic cognitive style
1	2	3	*A. Impoverished:* Seems deficient in human spheres of knowledge and evidences vague thought processes about everyday matters that are below intellectual level; social communications are easily derailed or conveyed via a circuitous logic (e.g., lacks awareness of human relations).
1	2	3	*B. Autistic:* Intrudes social communications with personal irrelevancies; there are notable circumstantial speech, ideas of reference, and metaphorical asides; is ruminative, appears self-absorbed and lost in occasional magical thinking; there is a marked blurring of fantasy and reality (e.g., exhibits peculiar ideas and superstitious beliefs).
1	2	3	*C. Distracted:* Is bothered by disruptive and often distressing inner thoughts; the upsurge from within of irrelevant and digressive ideation upsets thought continuity and interferes with social communications (e.g., withdraws into reveries to fulfill needs).
1	2	3	*D. Naive:* Is easily persuaded, unsuspicious, and gullible; reveals a Pollyanna attitude toward interpersonal difficulties, watering down objective problems and smoothing over troubling events (e.g., childlike thinking and reasoning).
1	2	3	*E. Scattered:* Thoughts are momentary and scrambled in an untidy disarray with minimal focus to them, resulting in a chaotic hodgepodge of miscellaneous and haphazard beliefs expressed randomly with no logic or purpose (e.g., intense and transient emotions disorganize thoughts).
1	2	3	*F. Flighty:* Avoids introspective thought and is overly attentive to trivial and fleeting external events; integrates experiences poorly, resulting in shallow learning and thoughtless judgments (e.g., faddish and responsive to superficialities).
1	2	3	*G. Expansive:* Has an undisciplined imagination and exhibits a preoccupation with illusory fantasies of success, beauty, or love; is minimally constrained by objective reality; takes liberties with facts and seeks to redeem boastful beliefs (e.g., indulges fantasies of repute/power).
1	2	3	*H. Mistrustful:* Is suspicious of the motives of others, construing innocuous events as signifying conspiratorial intent; magnifies tangential or minor social difficulties into proofs of duplicity, malice, and treachery (e.g., wary and distrustful).
1	2	3	*I. Deviant:* Construes ordinary events and personal relationships in accord with socially unorthodox beliefs and morals; is disdainful of traditional ideals and conventional rules (e.g., shows contempt for social ethics and morals).
1	2	3	*J. Dogmatic:* Is strongly opinionated, as well as unbending and obstinate in holding to his or her preconceptions; exhibits a broad social intolerance and prejudice (e.g., closed-minded and bigoted).
1	2	3	*K. Fatalistic:* Sees things in their blackest form and invariably expects the worst; gives the gloomiest interpretation of current events, believing that things will never improve (e.g., conceives life events in persistent pessimistic terms).
1	2	3	*L. Diffident:* Is hesitant to voice his or her views; often expresses attitudes contrary to inner beliefs; experiences contrasting and conflicting thoughts toward self and others (e.g., demeans own convictions and opinions).
1	2	3	*M. Cynical:* Skeptical and untrusting, approaching current events with disbelief and future possibilities with trepidation; has a misanthropic view of life, expressing disdain and caustic comments toward those who experience good fortune (e.g., envious or disdainful of those more fortunate).
1	2	3	*N. Vacillating:* Experiences rapidly changing, fluctuating, and antithetical perceptions or thoughts concerning passing events; contradictory reactions are evoked in others by virtue of his or her behaviors, creating, in turn, conflicting and confusing social feedback (e.g., erratic and contrite over own beliefs and attitudes).
1	2	3	*O. Constricted:* Constructs world in terms of rules, regulations, time schedules, and social hierarchies; is unimaginative, indecisive, and notably upset by unfamiliar or novel ideas and customs (e.g., preoccupied with lists, details, rules, etc.).

TABLE 21.5. MPS: IV. Self-Image Domain

As the inner world of symbols is mastered through development, one major configuration emerges to impose a measure of sameness on an otherwise fluid environment: the perception of self-as-object, a distinct, ever-present identity. Self-image is significant in that it serves as a guidepost and lends continuity to changing experience. Most patients have an implicit sense of who they are, but differ greatly in the clarity, accuracy, and complexity of their introspection of the psychic elements that make up this image.

1st best fit	2nd best fit	3rd best fit	Characteristic self-image
1	2	3	*A. Complacent:* Reveals minimal introspection and awareness of self; seems impervious to the emotional and personal implications of his or her role in everyday social life (e.g., minimal interest in own personal life).
1	2	3	*B. Estranged:* Possesses permeable ego boundaries, exhibiting acute social perplexities and illusions as well as experiences of depersonalization, derealization, and dissociation; sees self as "different," with repetitive thoughts of life's confusions and meaninglessness (e.g., self-perceptions are haphazard and fragmented).
1	2	3	*C. Alienated:* Sees self as a socially isolated person, one rejected by others; devalues self-achievements and reports feelings of aloneness and undesirability (e.g., feels injured and unwanted by others).
1	2	3	*D. Inept:* Views self as weak, fragile, and inadequate; exhibits lack of self-confidence by belittling own aptitudes and competencies (e.g., sees self as childlike and/or fragile).
1	2	3	*E. Energetic:* Sees self as full of vim and vigor, a dynamic force, invariably hardy and robust, a tireless and enterprising person whose ever-present energy galvanizes others (e.g., proud to be active and animated).
1	2	3	*F. Gregarious:* Views self as socially stimulating and charming; enjoys the image of attracting acquaintances and pursuing a busy and pleasure-oriented social life (e.g., perceived as appealing and attractive, but shallow).
1	2	3	*G. Admirable:* Confidently exhibits self, acts in a self-assured manner, and publicly displays achievements, despite being seen by others as egotistic, inconsiderate, and arrogant (e.g., has a sense of high self-worth).
1	2	3	*H. Inviolable:* Is highly insular, experiencing intense fears of losing identity, status, or powers of self-determination; nevertheless, has persistent ideas of self-reference, asserting as personally derogatory and scurrilous entirely innocuous actions and events (e.g., sees ordinary life events as invariably referring to self).
1	2	3	*I. Autonomous:* Values the sense of being free, unencumbered, and unconfined by persons, places, obligations, or routines; sees self as unfettered by the restrictions of social customs and the restraints of personal loyalties (e.g., values being independent of social responsibilities).
1	2	3	*J. Combative:* Values aspects of self that present tough, domineering, and power-oriented image; is proud to characterize self as unsympathetic and unsentimental (e.g., proud to be stern and feared by others).
1	2	3	*K. Worthless:* Sees self as valueless, of no account, a person who should be overlooked, owing to having no praiseworthy traits or achievements (e.g., sees self as insignificant or inconsequential).
1	2	3	*L. Undeserving:* Focuses on and amplifies the very worst features of self; judges self as worthy of being shamed, humbled, and debased; has failed to live up to the expectations of others, and hence should be reproached and demeaned (e.g., sees self as deserving to suffer).
1	2	3	*M. Discontented:* Sees self as unjustly misunderstood and unappreciated; recognizes that he or she is characteristically resentful, disgruntled, and disillusioned with life (e.g., sees self as unfairly treated).
1	2	3	*N. Uncertain:* Experiences the marked confusions of a nebulous or wavering sense of identity and self-worth; seeks to redeem erratic actions and changing self-presentations with expressions of contrition and self-punitive behaviors (e.g., has persistent identity disturbances).
1	2	3	*O. Reliable:* Sees self as industrious, meticulous, and efficient; fearful of error or misjudgment, and hence overvalues aspects of self that exhibit discipline, perfection, prudence, and loyalty (e.g., sees self as reliable and conscientious).

TABLE 21.6. MPS: V. Mood/Affect Domain

Few observables are more clinically relevant than the predominant character of an individual's affect and the intensity and frequency with which he or she expresses it. The meaning of extreme emotions is easy to decode. This is not so with the more subtle moods and feelings that insidiously and repetitively pervade the patient's ongoing relationships and experiences. The expressive features of mood/affect may be revealed, albeit indirectly, in activity level, speech quality, and physical appearance.

1st best fit	2nd best fit	3rd best fit	Characteristic mood/affect
1	2	3	*A. Apathetic:* Is emotionally impassive, exhibiting an intrinsic unfeeling, cold, and stark quality; reports weak affectionate or erotic needs, rarely displaying warm or intense feelings, and apparently unable also to experience either sadness or anger (e.g., unable to experience pleasure in depth).
1	2	3	*B. Distraught or insentient: Either* reports being apprehensive and ill at ease, particularly in social encounters; anxiously watchful, distrustful of others, and wary of their motives; *or* manifests drab, sluggish, joyless, and spiritless appearance; reveals marked deficiencies in emotional expression and personal encounters (e.g., highly agitated and/or affectively flat).
1	2	3	*C. Anguished:* Vacillates between desire for affection, fear of rebuff, and numbness of feeling; describes constant and confusing undercurrents of tension, sadness, and anger (e.g., unusually fearful of new social experiences).
1	2	3	*D. Pacific:* Quietly and passively avoids social tension and interpersonal conflicts; is typically pleasant, warm, tender, and noncompetitive (e.g., characteristically timid and uncompetitive).
1	2	3	*E. Mercurial:* Volatile and quicksilverish, at times unduly ebullient, charged up, and irrepressible; at other times flighty and erratic emotionally, blowing hot and cold (e.g., has marked penchant for momentary excitements).
1	2	3	*F. Fickle:* Displays short-lived and superficial emotions; is dramatically overreactive and exhibits tendencies to be easily enthused and as easily bored (e.g., impetuously pursues pleasure-oriented social life).
1	2	3	*G. Insouciant:* Manifests a general air of nonchalance and indifference; appears coolly unimpressionable or calmly optimistic, except when self-centered confidence is shaken, at which time either rage, shame, or emptiness is briefly displayed (e.g., generally appears imperturbable and composed).
1	2	3	*H. Irascible:* Displays a sullen, churlish, and humorless demeanor; attempts to appear unemotional and objective, but is edgy, touchy, surly, quick to react angrily (e.g., ready to take personal offense).
1	2	3	*I. Callous:* Exhibits a coarse incivility, as well as a ruthless indifference to the welfare of others; is unempathic, as expressed in wide-ranging deficits in social charitableness, human compassion, or personal remorse (e.g., experiences minimal guilt or contrition for socially repugnant actions).
1	2	3	*J. Hostile:* Has an overtly rough and pugnacious temper, which flares periodically into contentious argument and physical belligerence; is fractious, willing to do harm, even persecute others to get own way (e.g., easily embroiled in brawls).
1	2	3	*K. Woeful:* Is typically mournful, tearful, joyless, and morose; characteristically worrisome and brooding; low spirits rarely remit (e.g., frequently feels dejected or guilty).
1	2	3	*L. Dysphoric:* Intentionally displays a plaintive and gloomy appearance, occasionally to induce guilt and discomfort in others (e.g., drawn to relationships in which he or she will suffer).
1	2	3	*M. Irritable:* Is often petulant, reporting being easily annoyed or frustrated by others; typically obstinate and resentful, followed in turn by sulky and grumpy withdrawal (e.g., impatient and easily provoked into oppositional behavior).
1	2	3	*N. Labile:* Fails to accord unstable moods with external reality; has marked shifts from normality to depression to excitement, or has extended periods of dejection and apathy, interspersed with brief spells of anger, anxiety, or euphoria (e.g., mood changes erratically from sadness to bitterness to torpor).
1	2	3	*O. Solemn:* Is unrelaxed, tense, joyless, and grim; restrains overtly warm or covertly antagonistic feelings, keeping most emotions under tight control (e.g., affect is constricted and confined).

TABLE 21.7. MPS: VI. Intrapsychic Dynamics Domain

Although mechanisms of self-protection, need gratification, and conflict resolution are consciously recognized at times, they represent data derived primarily at the intrapsychic level. Because the ego or defense mechanisms are internal regulatory processes, they are more difficult to discern and describe than processes that are anchored closer to the observable world. As such, they are not directly amenable to assessment by self-reflective appraisal in their pure form, but only as derivatives that are potentially many levels removed from their core conflicts and their dynamic resolution. Despite the methodological problems they present, the task of identifying which mechanisms are most characteristic of a patient and the extent to which they are employed is extremely useful in a comprehensive clinical assessment.

1st best fit	2nd best fit	3rd best fit	Characteristic intrapsychic dynamics
1	2	3	*A. Intellectualization:* Describes interpersonal and affective experiences in a matter-of-fact, abstract, impersonal, or mechanical manner; pays primary attention to formal and objective aspects of social and emotional events.
1	2	3	*B. Undoing:* Bizarre mannerisms and idiosyncratic thoughts appear to reflect a retraction or reversal of previous acts or ideas that have stirred feelings of anxiety, conflict, or guilt; ritualistic or "magical" behaviors serve to repent for or nullify assumed misdeeds or "evil" thoughts.
1	2	3	*C. Fantasy:* Depends excessively on imagination to achieve need gratification and conflict resolution; withdraws into reveries as a means of safely discharging affectionate as well as aggressive impulses.
1	2	3	*D. Introjection:* Is firmly devoted to another to strengthen the belief that an inseparable bond exists between them; jettisons any independent views in favor of those of another to preclude conflicts and threats to the relationship.
1	2	3	*E. Magnification:* Engages in hyperbole, overstating and overemphasizing ordinary matters so as to elevate their importance, especially features that enhance not only his or her own virtues but those of others who are valued.
1	2	3	*F. Dissociation:* Regularly alters self-presentations to create a succession of socially attractive but changing facades; engages in self-distracting activities to avoid reflecting on/integrating unpleasant thoughts/emotions.
1	2	3	*G. Rationalization:* Is self-deceptive and facile in devising plausible reasons to justify self-centered and socially inconsiderate behaviors; offers alibis to place self in the best possible light, despite evident shortcomings or failures.
1	2	3	*H. Projection:* Actively disowns undesirable personal traits and motives and attributes them to others; remains blind to own unattractive behaviors and characteristics, yet is overalert to and hypercritical of the defects of others.
1	2	3	*I. Acting out:* Inner tensions that might accrue by postponing the expression of offensive thoughts and malevolent actions are rarely constrained; socially repugnant impulses are not refashioned in sublimated forms, but are discharged directly in precipitous ways, usually without guilt.
1	2	3	*J. Isolation:* Can be cold-blooded and remarkably detached from an awareness of the impact of his or her destructive acts; views objects of violation impersonally, often as symbols of devalued groups devoid of human sensibilities.
1	2	3	*K. Asceticism:* Engages in acts of self-denial, self-tormenting, and self-punishment, believing that one should exhibit penance and not be rewarded with life's bounties; not only is there a repudiation of pleasures, but there are harsh self-judgments and minor self-destructive acts.
1	2	3	*L. Exaggeration:* Repetitively recalls past injustices and seeks out future disappointments as a means of raising distress to troubled homeostatic levels; misconstrues if not sabotages personal good fortunes to enhance or maintain preferred suffering and pain.
1	2	3	*M. Displacement:* Discharges anger and other troublesome emotions either indirectly or by shifting them from their true objective to settings or persons of lesser peril; expresses resentments by substitute or passive means, such as acting inept or perplexed, or behaving in a forgetful or indolent manner.
1	2	3	*N. Regression:* Retreats under stress to developmentally earlier levels of anxiety tolerance, impulse control, and social adaptation; is unable or disinclined to cope with responsible tasks and adult issues, as evident in immature if not increasingly childlike behaviors.
1	2	3	*O. Reaction formation:* Repeatedly presents positive thoughts and socially commendable behaviors that are diametrically opposite to his or her deeper, contrary, and forbidden feelings; displays reasonableness and maturity when faced with circumstances that normally evoke anger or dismay in most persons.

TABLE 21.8. MPS: VII. Intrapsychic Content Domain

Significant experiences from the past leave an inner imprint, a structural residue composed of memories, attitudes, and affects that serve as a substrate of dispositions for perceiving and reacting to life's events. Analogous to the various organ systems in the body, both the character and the substance of these internalized representations of significant figures and relationships from the past can be differentiated and analyzed for clinical purposes. Variations in the nature and content of this inner world, or what are often called "object relations," can be identified with one or another personality and lead us to employ the following descriptive terms to represent them.

1st best fit	2nd best fit	3rd best fit	Characteristic intrapsychic content
1	2	3	*A. Meager:* Inner representations are few in number and minimally articulated, largely devoid of the manifold percepts and memories, or the dynamic interplay among drives and conflicts, that typify even well-adjusted persons.
1	2	3	*B. Chaotic:* Inner representations consist of a jumble of miscellaneous memories and percepts, random drives and impulses, and uncoordinated channels of regulation that are only fitfully competent for binding tensions, accommodating needs, and mediating conflicts.
1	2	3	*C. Vexatious:* Inner representations are composed of readily reactivated, intense, and anxiety-ridden memories; limited avenues of gratification; and few mechanisms to channel needs, bind impulses, resolve conflicts, or deflect external stressors.
1	2	3	*D. Immature:* Inner representations are composed of unsophisticated ideas and incomplete memories, rudimentary drives and childlike impulses, as well as minimal competencies to manage and resolve stressors.
1	2	3	*E. Piecemeal:* Inner representations are disorganized and dissipated, a jumble of diluted and muddled recollections that are recalled by fits and starts, serving only as momentary guideposts for dealing with everyday tensions and conflicts.
1	2	3	*F. Shallow:* Inner representations are composed largely of superficial yet emotionally intense affects, memories, and conflicts, as well as facile drives and insubstantial mechanisms.
1	2	3	*G. Contrived:* Inner representations are composed far more than usual of illusory ideas and memories, synthetic drives and conflicts, and pretentious if not simulated percepts and attitudes, all of which are readily refashioned as the need arises.
1	2	3	*H. Unalterable:* Inner representations are arranged in an unusual configuration of rigidly held attitudes, unyielding percepts, and implacable drives, which are aligned in a semidelusional hierarchy of tenacious memories, immutable cognitions, and irrevocable beliefs.
1	2	3	*I. Debased:* Inner representations are a mix of revengeful attitudes and impulses oriented to subvert established cultural ideals and mores, as well as to debase personal sentiments and conventional societal attainments.
1	2	3	*J. Pernicious:* Inner representations are distinguished by the presence of aggressive energies and malicious attitudes, as well as by a contrasting paucity of sentimental memories, tender affects, internal conflicts, shame, or guilt feelings.
1	2	3	*K. Forsaken:* Inner representations have been depleted or devitalized, either drained of their richness and joyful elements or withdrawn from memory, leaving the person to feel abandoned, bereft, and discarded.
1	2	3	*L. Discredited:* Inner representations are composed of disparaged past memories and discredited achievements, of positive feelings and erotic drives transposed onto their least attractive opposites, of internal conflicts intentionally aggravated, of mechanisms of anxiety reduction subverted by processes that intensify discomforts.
1	2	3	*M. Fluctuating:* Inner representations compose a complex of opposing inclinations and incompatible memories that are driven by impulses designed to nullify his or her own achievements and/or the pleasures and expectations of others.
1	2	3	*N. Incompatible:* Rudimentary and expediently devised, but repetitively aborted, inner representations have led to perplexing memories, enigmatic attitudes, contradictory needs, antithetical emotions, erratic impulses, and opposing strategies for conflict reduction.
1	2	3	*O. Concealed:* Only those inner affects, attitudes, and actions that are socially approved are allowed conscious awareness or behavioral expression, resulting in gratification being highly regulated, forbidden impulses sequestered and tightly bound, personal and social conflicts defensively denied and kept from awareness, all maintained under stringent control.

TABLE 21.9. MPS: VIII. Intrapsychic Structure Domain

The overall architecture that serves as a framework for an individual's psychic interior may display weakness in its structural cohesion, exhibit deficient coordination among its components, and possess few mechanisms to maintain balance and harmony, regulate internal conflicts, or mediate external pressures. The concept of intrapsychic structure refers to a personality's organizational strength, interior congruity, and functional efficacy. Psychoanalytic usage tends to be limited to quantitative degrees of integrative pathology, not to *qualitative variations* in integrative configuration.

1st best fit	2nd best fit	3rd best fit	Characteristic intrapsychic structure
1	2	3	*A. Undifferentiated:* Given an inner barrenness, a feeble drive to fulfill needs, and minimal pressures to defend against or resolve internal conflicts, or to cope with external demands, internal structures are best characterized by limited coordination and deficient organization.
1	2	3	*B. Fragmented:* Coping and defensive operations are haphazardly organized in a fragile assemblage, leading to spasmodic and desultory actions in which primitive thoughts and affects are directly discharged, with few reality-based sublimations, leading to significant further structural disintegrations.
1	2	3	*C. Fragile:* Tortuous emotions depend almost exclusively on a single modality for their resolution and discharge, that of avoidance, escape, and fantasy; hence, when faced with unanticipated stress, there are few resources available to deploy and few positions to revert to, short of a regressive decompensation.
1	2	3	*D. Inchoate:* Owing to entrusting others with the responsibility to fulfill needs and to cope with adult tasks, there is both a deficit and a lack of diversity in internal structures and controls, leaving a miscellany of relatively undeveloped and immature adaptive abilities and elementary systems for independent functioning.
1	2	3	*E. Fleeting:* Structures are highly transient, existing in momentary forms that are cluttered and disarranged, making effective coping efforts temporary at best. Affect and action are unconstrained, owing to the paucity of established controls and purposeful goals.
1	2	3	*F. Disjointed:* A loosely knit structural conglomerate exists in which processes of internal regulation and control are scattered and unintegrated, with few methods for restraining impulses, coordinating defenses, and resolving conflicts, leading to broad and sweeping mechanisms to maintain psychic cohesion and stability that, when employed, only further disarrange thoughts, feelings, and actions.
1	2	3	*G. Spurious:* Coping and defensive strategies are flimsy and transparent, only appearing substantial and dynamically orchestrated, regulating impulses only marginally, channeling needs with minimal restraint, and creating an egocentric inner world in which conflicts are dismissed, failures are quickly redeemed, and self-pride is effortlessly reasserted.
1	2	3	*H. Inelastic:* A markedly constricted and inflexible pattern of coping and defensive methods exists, as well as rigidly fixed channels of conflict mediation and need gratification; these create an overstrung and taut frame that is so uncompromising in its accommodation to changing circumstances that unanticipated stressors are likely to precipitate either explosive outbursts or inner shatterings.
1	2	3	*I. Unruly:* Inner defensive operations are noted by their paucity, as are efforts to curb irresponsible drives and attitudes, leading to easily transgressed social controls, low thresholds for impulse discharge, few subliminatory channels, unfettered self-expression, and a marked intolerance of delay or frustration.
1	2	3	*J. Eruptive:* Despite a generally cohesive structure of routinely modulating controls and expressive channels, surging, powerful, and explosive energies of an aggressive and sexual nature produce precipitous outbursts that periodically overwhelm and overrun otherwise reasonable restraints.
1	2	3	*K. Depleted:* The scaffold for structures is markedly weakened, with coping methods enervated and defensive strategies impoverished and devoid of vigor and focus, resulting in a diminished if not exhausted capacity to initiate action and regulate affect.

(cont.)

TABLE 21.9. *(cont.)*

1st best fit	2nd best fit	3rd best fit	Characteristic intrapsychic structure
1	2	3	*L. Inverted:* Structures have a dual quality, one more or less conventional, the other its obverse—resulting in a repetitive undoing of affect and intention, of a transposing of channels of need gratification with those leading to their frustration, and of actions that produce antithetical if not self-sabotaging consequences.
1	2	3	*M. Divergent:* There is a clear division in the pattern of internal elements, such that coping and defensive maneuvers are often directed toward incompatible goals, leaving major conflicts unresolved and psychic cohesion impossible, as fulfillment of one drive or need inevitably nullifies or reverses another.
1	2	3	*N. Split:* Inner cohesion constitutes a sharply segmented and conflictful configuration with a marked lack of consistency among elements; levels of consciousness occasionally blur; a rapid shift occurs across boundaries separating unrelated memories/affects, resulting in schisms upsetting limited extant psychic order.
1	2	3	*O. Compartmentalized:* Psychic structures are rigidly organized in a tightly consolidated system that is clearly partitioned into numerous distinct and segregated constellations of drive, memory, and cognition, with few open channels to permit any interplay among these components.

TABLE 21.10. Spectra That Best Characterize the Person

1st best fit	2nd best fit	3rd best fit	Normal-to-abnormal personality spectrum
1	2	3	Apathetic–schizoid
1	2	3	Schizotypal–schizophrenic
1	2	3	Withdrawn–avoidant
1	2	3	Attached–dependent
1	2	3	Exuberant–turbulent
1	2	3	Sociable–histrionic
1	2	3	Confident–narcissistic
1	2	3	Paranoid–paraphrenic
1	2	3	Nonconforming–antisocial
1	2	3	Assertive–sadistic
1	2	3	Doleful–melancholic
1	2	3	Aggrieved–masochistic
1	2	3	Resentful–negativistic
1	2	3	Borderline–cyclophrenic
1	2	3	Compliant–compulsive

TABLE 21.11. Overall Level of Social and Occupational Functioning

Judgment	Rating number	Description
Excellent	1	Clearly manifests an effective if not superior level of functioning in relating to family and social peers, even to helping others in resolving their difficulties, as well as demonstrating high occupational performance and success.
Very good	2	Exhibits considerable social and occupational skills on a reasonably consistent basis, evidencing few if any major areas of interpersonal stress or occupational difficulty.
Good	3	Displays a higher-than-average level of social and occupational competence in ordinary matters of everyday life. He or she does experience intermittent difficulties in interpersonal relationships and in efforts to achieve work satisfaction.
Fair	4	Functions about average for a typical patient seen in outpatient clinical work. Although able to meet everyday family, social, and occupational responsibilities adequately, there remain problematic or extended periods of occupational stress and/or interpersonal conflict.
Poor	5	Able to be maintained on an outpatient basis, but often precipitates severe conflicts with others that upset his or her equanimity in either or both interpersonal relationships and occupational settings.
Very poor	6	There is an inability to function competently in most social and occupational settings. Difficulties are precipitated by the patient, destabilizing job performance and upsetting relationships with significant others. Inpatient hospitalization may be necessary to manage periodic severe psychic disruptions.
Markedly impaired	7	A chronic and marked disintegration is present across most psychic functions. The loss of physical and behavioral controls necessitate extended stays in residential or hospital settings, requiring both sustained care and self-protection.

Domain/Tactical Therapeutic Options

An obvious benefit of an instrument such as the MPS is that it facilitates the coordination of domain-based tactical therapeutic strategies, in keeping with an integrative approach to personalized (i.e., personality-guided) treatment planning (e.g., Millon, 1999; Millon & Grossman, 2007a, 2007b, 2007c). The MPS specifies *what* techniques and modalities are likely to benefit a patient. For example, we can address dysfunctions in the realm of interpersonal conduct by employing interpersonal, family, or group therapeutic methods. Brief dynamic modalities may be especially suited to the realm of intrapsychic content. The phenomenological schools subsuming cognitive, existential, and humanistic therapies may be well chosen to modify difficulties of cognitive style and self-image.

Therapeutic modalities vary in their degree of specificity and strategic goals; this is not often merely an accident of history, but can be tied back to assumptions latent in the therapies themselves. However, the progression over time has been toward both greater specificity and clearer goals; modern therapeutic delivery systems demand this, and the larger society—with greater time demands, less luxury in longer-term treatments, and higher volumes of psychotherapy—enforces it. Some approaches to psychotherapy, such as the cognitive-behavioral, put into place highly detailed elements (e.g., agreed-upon goals, termination criteria, and ongoing assessments) in which therapy itself becomes a

self-regulating system. Ongoing assessments ensure the existence of a feedback process that is open to inspection and negotiation by both therapist and patient. The mode is one of action rather than talk; the action itself is interactive and transactive; and therapy is forward-looking and concentrates on realizing present possibilities as a means of creating or opening up new possibilities. Persons are often changed more through exposure and action than through focusing on and unraveling the problems of the past. Insight may be catalytic, even necessary, but is ultimately limited for its own purposes.

In an early book, Millon (1981) likened personality to an immune system for the psyche, such that stability, constancy, and internal equilibrium become the goals of a personality. This, of course, may run directly in opposition to the explicit goal of therapy, which is change. If (or, usually, when) a particular therapy patient feels threatened, his or her personality system functions as a form of passive resistance, albeit one that may be experienced as a positive force (or trait) by the therapist. In fact, the structural groundings of a patient's self-image are so preemptive and confirmation-seeking that the true meaning of the therapist's comments may never reach the level of conscious processing. Alternatively, even if a patient's equilibrium is initially up-ended by a particular interpretation, his or her intrapsychic defenses may kick in to ensure that a therapist's comments are somehow distorted, misunderstood, interpreted in a less threatening manner, or even ignored. The first is a passive form of resistance; the second is an active form. No wonder, then, that effective therapy is often considered anxiety-provoking, for it is in situations where the patient really has no effective response—where the functioning of the psychic immune system is temporarily suppressed—that the scope of his or her response repertoire is most likely to be broadened. Personality goes with what it knows, and it is with the unknown that learning is most possible.

A coordinated schema of strategic goals and tactical modalities for treatment seeks to accomplish change in an effective and efficient psychotherapy (Millon & Grossman, 2007a, 2007b, 2007c). In coordinating approaches mirroring the synchronized composition of the individual's complex clinical syndrome and personality system, an effort should be made to select domain-focused tactics that will fulfill the strategic goals of treatment. Interventions of an unfocused, rambling, and diffuse nature lead only to minor progressions and passive resistance to change via habitual characteristics already intrinsic to the individual's personality system. Ultimately, something must happen that cannot be readily fielded by habitual processes—in other words, something that targets and disrupts the homeostatic domain functions identified by the MPS.

The purpose of a domain focus, or knowing clearly what to do in therapy and why to do it, is to keep the therapeutic enterprise from becoming too diffused. The person-focused systems model runs counter to the deterministic universe-as-machine model of the late 19th century, which features slow but incremental gains. In a focused, "punctuated," personalized model, therapeutic advances may clearly be spelled out through combinations and/or progressions of tactics such as those Millon (1997) has termed "potentiated pairings" and "catalytic sequences." Tactical specificity, then, is required in part because the psychic level at which therapy is practiced is fairly explicit. The therapeutic relationship is largely dominated by a discussion of specific domain behaviors, specific domain feelings, and specific domain cognitions, not by an abstract discussion of personality style or clinical syndromes.

As Millon (1997) has noted previously, there are "strategic goals" of therapy (i.e., goals that endure across numerous sessions and against which progress is measured), and there are specific "domain modality tactics" by which these goals are pursued. Ideally, strategies and tactics should be integrated, with the tactics chosen to accomplish strategic goals, and the strategies chosen on the basis of what tactics might actually achieve. To illustrate, intrapsychic therapies are highly strategic but tactically impoverished; pure behavioral therapies are highly tactical but strategically narrow and inflexible. There are in fact many different ways that strategies may be operationalized. Just as diagnostic criteria are neither necessary nor sufficient for membership in a given class, it is likely that no technique is an inevitable consequence of a given clinical strategy. The ingenuity of individual therapists to invent

techniques ad hoc will assure an almost infinite number of ways to operationalize or put into action a given clinical strategy.

The following vignette describes a recent case in which such a domain-based, personalized case conceptualization was utilized, and outlines the MPS assessment procedure. Many aspects of this individual's case have been fictionalized to protect the person's privacy; however, efforts have also been made to preserve potential for therapeutic process and domain analysis consistency.

Case Illustration: Yulya V.

Background Information

Yulya, a 32-year-old woman born in Kiev, Ukraine, to Jewish parents, presented for treatment 8 months after returning from an extended tour of duty in both Iraq and Afghanistan. Her family had emigrated to the United States when she was 7 years old, and, owing to their feelings of persecution and personal limitations at the time of their departure from what was then the Soviet Union, "constantly were overprotective and insistent on keeping me on a short leash." As she recalled, her first years in her new country were marked by shyness and uncertainty, with constant yearning for her parents when they were not immediately present. In her early adolescence, however, she took a very different turn: "I suddenly started resenting the hell out of it and for a while I did anything and everything I wanted." She reported periods of sexual promiscuity, heavy substance use, and consistently oppositional behaviors. "Basically, if my parents told me to do something, I'd do the opposite. They eventually started to try the opposite—encouraging me to do stupid things—and I'd be a model daughter until they noticed; then I'd go bad. It was when they stopped caring that I didn't know what to do." In her early 20s, Yulya was working as an exotic dancer, and allowed herself to get "picked up" by a club patron who then nearly beat her to death. "Ever since, I've been thankful for that awful experience and everything I do. I've made it a point to ask, 'Is this what's best for me, my family, and my world?' "

Hoping to study medicine eventually via subsequent benefits, Yulya enlisted in the U.S. Army as a medical services specialist shortly before the events of 9/11, after which time she was deployed to Afghanistan and subsequently to Iraq. "Of course now I question the whole thing, but then I just thought I was acting for a noble cause and would do absolutely anything to support the cause." In the service, she met her future husband, a war contractor, and became pregnant; shortly thereafter, the couple returned to the United States and got married.

Presenting Picture

Approximately 1 year later, Yulya noted that she started experiencing "really scary moments," wherein she felt as if she was visualizing and reliving episodes from medical traumas and could not recall any medical training. This sinking, hopeless feeling appeared to lead to her feeling "distanced" from most aspects of her life, and her husband noticed her becoming less engaged, acting more apathetic, and behaving "like a catatonic zombie." At first Yulya argued that "if you'd been through what I've been through, you'd not want to feel everything either," and "of course, I'm going to remember what I've been through; it's no big deal." However, when her husband noted that he felt she might become a "cold mother" to her newborn son, she decided to address it.

Assessment Procedures

Initial assessment via clinical interview and the MCMI-III with the Grossman Facet Scales, as well as other instruments capturing both broad-band and symptom-specific features, indicated an overall personality constellation of primarily resentful–negativistic and compliant–compulsive features not meeting full criteria for an Axis II diagnosis, but nevertheless indicating significant patterns that would markedly influence treatment. Also present were some very mild but still influential nonconforming–antisocial characteristics. This pattern was strongly marked by an excessive and troubling conflict between meeting the needs of the self and responding to the expectations of others, as well as difficulties negotiating decisions and actions that would stand to modify her environment versus adjusting her own expectations and sense of self to "fit in" with her given circumstances. Layered on this were mild, agitated depressive symptomatology and a fairly clear, moderately severe diagnosis of posttraumatic stress disorder (PTSD).

The clinician also completed the MPS in order to capture more molecular facets of this person and her presentation, and to facilitate selection and implementation of specific thera-

peutic techniques to be coordinated into a tactical treatment plan addressing overt symptomatology and characterological domain traits. The following significant domains were identified:

I. *Expressive Behavior domain*. Yulya was notably disciplined in her overt behavioral tendencies, showing a forthrightness and sense of duty in most all of her actions, although she rarely seemed passionate while engaged in a task. She did, however, display occasional resentful acts, though these seemed more resentful by description than in action.

II. *Interpersonal Conduct domain*. In this area, Yulya showed only respectfulness, without regard to whom she was interacting with. Once again, there was a sense that this was more a role she was playing than how she really felt about the interaction; this did not seem to be an instance of falseness, but more of an affective "removal."

III. *Cognitive Style domain*. Clearly here, Yulya demonstrated a certain *constriction*, as though thinking or acting in a manner that would violate established norms would be totally unacceptable. However, she could identify, with some insight, that this was inconsistent with her earlier life beliefs, which were actually more in line with the nonconforming–antisocial spectrum's deviant stylings.

IV. *Self-Image domain*. Yulya presented herself as both autonomous and reliable, and this combination typically functioned well when she did not have imperatives from others. However, she frequently did have to answer others' needs, and a certain amount of discontentment then tended to appear.

V. *Mood/Affect domain*. Clearly, Yulya demonstrated solemnness in her affect, keeping tight controls over her expressed emotion; this occasionally appeared to gravitate toward apathy, but it would become clear, with very little engagement on the part of the clinician, that this distancing was more a product of control than of uninterest.

VI. *Intrapsychic Dynamics domain*. Displacement characterized this first, most observable intrapsychic domain: Yulya would, on specific stressful occasions (and largely out of overall character), "forget" important responsibilities or fail to acknowledge something. She would blame her "closet ADD," as she termed it, for this.

VII. *Intrapsychic Content domain*. Intrapsychic objects seemed to be concealed with Yulya, as she reported regularly that her feelings regarding important people, events, and relationships would only be recalled or reframed in terms of their positive qualities, and her views of past conflicts or conflictual content had a consistent quality of being "for the best."

VIII. *Intrapsychic Structure domain*. Yulya indicated, through vague inferences, that her inner world often seemed divergent, setting up win–lose scenarios in terms of one drive's or motivation's effectively nullifying another. At the same time, she cloaked this with a rather rigid, tenuous, compartmentalized structure.

In examining these personological domains, we can see that the etiology and presentation of Yulya's PTSD (the identified catalyst for treatment) can be viewed in a much more specific and individualized manner than in its Axis I symptomatic description. Perhaps in this case, the intrapsychic turmoil between keeping order and resisting expectation gave Yulya a context for the expression of guilt over not feeling able to do what was expected in times of crisis (the content of her symptomatic flashbacks). Perhaps she felt limited by rules and expectations, but her belief system did not allow her to express unique ideas; this impasse might be compounded by her experience as an exotic dancer, in which she took it upon herself to find a less socially acceptable means of survival and ended up nearly dead. This in turn ran counter to her already conflicted view of self. A tactical treatment plan, then, might begin with acknowledgment (in both the Self-Image and Cognitive Style domains) of places where Yulya might feel she had "no choice" but to meet expectations, especially *because* she was so capable. This might allow her, then, to become aware of the resultant resentfulness that not only percolated beneath the surface of a dutiful front (the expressive behavior realm), but also permeated other domains of her life. This would lay the groundwork for Yulya to begin the process of working through her discontentments and developing less constricted and more flexible coping measures than those driven only by the expectations of others. This, thematically, would open the doors for a more contextual processing of her "relived experiences."

Conclusion

The MPS is the most recent endeavor in addressing personological spectra and do-

mains for purposes of coordinated, tactical treatment planning such as that found in our recent intervention-oriented writings (e.g., Millon & Grossman, 2007a, 2007b, 2007c). The MPS represents a continuation of the MPDC and MG-PDC, updated to reflect innovations in theory and therapeutic approaches, and modified to be well suited to time-limited assessment and intervention paradigms. In focusing on the domain level of personality description represented by the instrument, with its perspective on comprehensiveness and the comparability of functional and structural domains, a clinician can emphasize and operationalize a pragmatic, dimensional approach to personality-oriented treatment. Further work is necessary and encouraged in order to evaluate the clinical utility and acceptance of the MPS, beyond its concordance with an explicated and comprehensive theoretical perspective.

Acknowledgment

Portions of this chapter have been adapted and updated from Grossman, Tringone, and Millon (2008). Copyright 2008 by The Guilford Press. Adapted by permission.

References

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., rev.). Washington, DC: Author.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.

Campbell, D. T., & Fiske, D. W. (1959). Convergent and discriminant validation by the multitrait–multimethod matrix. *Psychological Bulletin*, *56*, 81–105.

Cantor, N., & Genero, N. (1986). Psychiatric diagnosis and natural categorization: A close analogy. In T. Millon & G. L. Klerman (Eds.), *Contemporary directions in psychopathology: Toward the DSM-IV* (pp. 233–256). New York: Guilford Press.

Cantor, N., Smith, E. E., French, R., & Mezzich, J. (1980). Psychiatric diagnosis as prototype categorization. *Journal of Abnormal Psychiatry*, *89*, 181–193.

Clarkin, J. F., Widiger, T. A., Frances, A., Hurt, S. W., & Gilmore, M. (1983). Prototypic typology and the borderline personality disorder. *Journal of Abnormal Psychology*, *92*, 263–275.

Costa, P. T., Jr., & McCrae, R. R. (1992). The five-factor model of personality and its relevance to personality disorders. *Journal of Personality Disorders*, *6*, 343–359.

Costa, P. T., Jr., & McCrae, R. R. (1995). Domains and facets: Hierarchical personality assessment using the Revised NEO Personality Inventory. *Journal of Personality Assessment*, *64*, 21–50.

Cronbach, L. J., & Meehl, P. E. (1955). Construct validity in psychological tests. *Psychological Bulletin*, *52*, 281–302.

Davis, R. D., & Millon, T. (1993). The five-factor model for personality disorders: Apt or misguided? *Psychological Inquiry*, *4*, 104–109.

Frances, A. (1980). The DSM-III personality disorders section: A commentary. *American Journal of Psychiatry*, *137*, 1050–1054.

Frances, A. (1982). Categorical and dimensional systems of personality diagnosis: A comparison. *Comprehensive Psychiatry*, *23*, 516–527.

Frances, A., & Widiger, T. A. (1986). Methodological issues in personality disorder diagnosis. In T. Millon & G. L. Klerman (Eds.), *Contemporary directions in psychopathology: Toward the DSM-IV* (pp. 381–400). New York: Guilford Press.

Garfield, S. (1986). Problems in diagnostic classification. In T. Millon & G. L. Klerman (Eds.), *Contemporary directions in psychopathology: Toward the DSM-IV* (pp. 99–114). New York: Guilford Press.

Grossman, S., Tringone, R. F., & Millon, T. 92008). Using the Millon–Grossman Personality Domains Checklist (MG-PDC) to integrate diverse clinical data. In T. Millon & C. Bloom (Eds.), *The Millon inventories: A practitioner's guide to personalized clinical assessment* (2nd ed., pp. 580–606). New York: Guilford Press.

Jackson, D. N. (1970). A sequential system for personality scale development. In C. D. Spielberger (Ed.), *Current topics in clinical and*

community psychology (Vol. 2, pp. 61–92). New York: Academic Press.

Klerman, G. L. (1986). Historical perspective on contemporary schools of psychopathology. In T. Millon & G. L. Klerman (Eds.), *Contemporary directions in psychopathology: Toward the DSM-IV* (pp. 3–28). New York: Guilford Press.

Livesley, W. J., Jackson, D. N., & Schroeder, M. L. (1989). A study of the factorial structure of personality pathology. *Journal of Personality Disorders*, *3*, 292–306.

Loevinger, J. (1957). Objective tests as instruments of psychological theory. *Psychological Reports*, *3*, 635–694.

Maser, J. D., & Akiskal, H. (Eds.). (2002). Spectrum concepts in major mental disorders. *Psychiatric Clinics of North America*, *25*, xi–xiii.

Millon, T. (1969). *Modern psychopathology: A biosocial approach to maladaptive learning and functioning*. Philadelphia: Saunders.

Millon, T. (1981). *Disorders of personality: DSM-III, Axis II*. New York: Wiley.

Millon, T. (1986a). On the past and future of the DSM-III: Personal recollections and projections. In T. Millon & G. L. Klerman (Eds.), *Contemporary directions in psychopathology: Toward the DSM-IV* (pp. 29–70). New York: Guilford Press.

Millon, T. (1986b). Personality prototypes and their diagnostic criteria. In T. Millon & G. L. Klerman (Eds.), *Contemporary directions in psychopathology: Toward the DSM-IV* (pp. 671–712). New York: Guilford Press.

Millon, T. (1987a). *Millon Clinical Multiaxial Inventory–II manual*. Minneapolis, MN: National Computer Systems.

Millon, T. (1987b). On the nature of taxonomy in psychopathology. In M. Hersen & C. Last (Eds.), *Issues in diagnostic research* (pp. 3–85). New York: Plenum Press.

Millon, T. (1990). *Toward a new personology: An evolutionary model*. New York: Wiley.

Millon, T. (1991). Classification in psychopathology: Rationale, alternatives, and standards. *Journal of Abnormal Psychology*, *100*, 245–261.

Millon, T. (1999). *Personality-guided therapy*. New York: Wiley.

Millon, T. (2004). *Masters of the mind*. Hoboken, NJ: Wiley.

Millon, T., & Bloom, C. (Eds.). (2008). *The Millon inventories: A practitioner's guide to personalized clinical assessment* (2nd ed.). New York: Guilford Press.

Millon, T. (with Davis, R. D.). (1996). *Disorders of personality: DSM-IV and beyond* (2nd ed.). New York: Wiley.

Millon, T., & Grossman, S. (2007a). *Moderating severe personality disorders: A personalized psychotherapy approach*. Hoboken, NJ: Wiley.

Millon, T., & Grossman, S. (2007b). *Overcoming resistant personality disorders: A personalized psychotherapy approach*. Hoboken, NJ: Wiley.

Millon, T., & Grossman, S. (2007c). *Resolving difficult clinical syndromes: A personalized psychotherapy approach*. Hoboken, NJ: Wiley.

Millon, T. (with Millon, C., Davis, R., & Grossman, S.). (2006a). *The Millon Adolescent Clinical Inventory (MACI) manual* (2nd ed.). Minneapolis, MN: Pearson Assessments.

Millon, T. (with Millon, C., Davis, R., & Grossman, S.). (2006b). *The Millon Clinical Multiaxial Inventory–III (MCMI-III) manual* (3rd ed.). Minneapolis, MN: Pearson Assessments.

Millon, T., & Tringone, R. (1989). Co-occurrence and diagnostic efficiency statistics. Unpublished raw data.

Morey, L. C. (1988). Personality disorders in DSM-III and DSM-III-R: Convergence, coverage, and internal consistency. *American Journal of Psychiatry*, *145*, 573–577.

Rapaport, D. (1953). Some metapsychological considerations concerning activity and passivity. In M. M. Gill (Ed.), *The collected papers of David Rapaport*. New York: Basic Books.

Shea, M. T. (1992). Some characteristics of the Axis II criteria sets and their implications for assessment of personality disorders. *Journal of Personality Disorders*, *6*, 377–381.

Skinner, H. A. (1986). Construct validation approach to psychiatric classification. In T. Millon & G. L. Klerman (Eds.), *Contemporary directions in psychopathology: Toward the DSM-IV* (pp. 307–330). New York: Guilford Press.

Stricker, G., & Gold, J. R. (1988). A psychodynamic approach to the personality disorders. *Journal of Personality Disorders*, *2*, 350–359.

Tringone, R. (1990). *Construction of the Millon Personality Diagnostic Checklist–III-R and personality prototypes*. Unpublished doctoral dissertation, University of Miami.

Tringone, R. (1997). The MPDC: Composition and clinical applications. In T. Millon (Ed.), *The Millon inventories* (pp. 449–474). New York: Guilford Press.

Trivers, R. L. (1974). Parental investment and sexual selection. In B. Campbell (Ed.), *Sexual selection and the descent of man 1871–1971*. Chicago: Aldine.

Watson, D., & Tellegen, A. (1985). Toward a consensual structure of mood. *Psychological Bulletin*, *98*, 219–235.

Westen, D., & Weinberger, J. (2004). When clinical description becomes statistical prediction. *American Psychologist*, *59*, 595–613.

Widiger, T. A. (1982). Prototypic typology and borderline diagnoses. *Clinical Psychology Review*, *2*, 115–135.

Widiger, T. (1992). Categorical versus dimensional classification. *Journal of Personality Disorders*, *6*, 287–300.

Widiger, T. A., Hurt, S. W., Frances, A., Clarkin, J. F., & Gilmore, M. (1984). Diagnostic efficiency and DSM-III. *Archives of General Psychiatry*, *41*, 1005–1012.

Widiger, T. A., & Trull, T. J. (2007). Plate tectonics in the classification of personality disorder: Shifting to a dimensional model. *American Psychologist*, *62*, 71–83.

Wittgenstein, L. (1953). *Philosophical investigations*. Oxford, UK: Blackwell.

World Health Organization. (1992). *International statistical classification of diseases, injuries, and causes of death* (10th rev.). Geneva: Author.

PART IV

INNOVATIVE THEORETICAL AND EMPIRICAL PROPOSALS

CHAPTER 22

Neuroscientific Foundations of Psychopathology

Christopher J. Patrick
Edward M. Bernat

Prominent scholars in the mental health field have called for efforts to integrate neurobiological concepts and findings directly into systems for diagnosing psychopathological disorders (Hyman, 2007), in order to improve the effectiveness of assessment, prevention, and treatment of such disorders (Insel & Scolnick, 2006). However, a number of challenges exist to understanding traditional mental disorders in neuroscientific terms. One of the most significant is that mental disorder syndromes represent complex targets for neurobiological study: They manifest themselves in diverse ways clinically (phenotypically), and they show frequent overlap (comorbidity) rather than occurring in isolation from one another. A further challenge is the essential measurement gap that exists between diagnostic phenotypes (operationalized in the domain of interview or self-report) and neurobiological systems/processes (operationalized in the domain of brain or other physiological activity). Yet another has to do with the psychometric limitations of single-session/single-task neuroscience procedures as a basis for *individual differences* assessment (Vul, Harris, Winkielman, & Pashler, 2009).

Here we propose that neuroscientific conceptualization and understanding of mental disorders can be advanced by focusing programmatic efforts on *neurobehavioral trait* constructs—that is, individual difference constructs with direct referents in neurobiology as well as behavior (Depue & Iacono, 1989). As concrete examples, we highlight fear–fearlessness and inhibitory control as two neurobehavioral constructs of relevance to differing forms of psychopathology. Variations in fear and fearlessness are posited to reflect individual differences in the sensitivity of the brain's defensive motivational system. Variations in inhibitory control are posited to reflect individual differences in the functioning of brain systems that modulate affective and behavioral response in the service of distal goals. We propose that these constructs, because they provide a concrete basis for linking neurobiological systems to measurable deviations in behavior, can serve as important initial referents for a "psychoneurometric" approach to the

assessment of individual differences relevant to psychopathology.

Psychometric and Experimental Approaches to the Study of Psychopathology

Two approaches to the neurobiological study of mental disorders have predominated for many years up to the present. One is the psychometric–dimensional approach, which relies on correlational analytic methods; the other is the experimental–diagnostic approach, which relies on statistical comparisons of groups. The former—exemplified by the work of such writers as Eysenck (1967), Tellegen (1985), and Cloninger (1987)—entails efforts to identify psychopathology-related individual difference dimensions on the basis of quantitative/psychometric methods and link them to neurobiological systems and processes. The latter—exemplified by the work of early experimental psychopathologists like Hare (1978), Lykken (1957), Maher (1968), and McGhie and Chapman (1961), as well as that of many contemporary clinical neuroscientists (e.g., Barch et al., 2001; Blair, 2006; Gotlib et al., 2005; Heller, Nitschke, Etienne, & Miller, 1997)—entails efforts to identify neurobiological processing or reactivity differences between participant groups classified on the basis of the presence versus absence of some diagnostic condition.

Although each of these approaches has contributed importantly to our understanding of neurobiological factors in psychopathology, notable limitations are associated with each. Research in the psychometric–dimensional tradition has for the most part focused on personality trait constructs defined on the basis of self-report. This approach is advantageous in that it makes use of specialized quantitative methods (including item analysis, structural analysis, and varying types of reliability analysis) to optimize precision and consistency of measurement of target individual difference dimensions. In addition, it provides for efficient data collection with high numbers of participants. As a function of these advantages, research employing the psychometric–dimensional approach has yielded robust and replicable findings in large participant samples. However, the trait constructs targeted in work of this sort, although associated empirically with psychopathological syndromes, do not converge clearly with specific diagnostic conditions (i.e., they correlate to varying degrees, and moderately at best, with multiple disorders).

In contrast, experimental psychopathology research has focused predominantly on diagnostic conditions of interest, including those defined within the current version (fourth edition, text revision) of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV-TR; American Psychiatric Association, 2000). The power of this approach derives from the elegance of experimental task paradigms that can be brought to bear on the study of psychopathology. Contemporary clinical neuroscience research, for example, combines the methodological sophistication of cognitive science with the precise anatomical measurement afforded by techniques such as dense-array electroencephalography (EEG), magnetoencephalography (MEG), and magnetic resonance imaging (MRI) to elucidate brain-based processing differences associated with psychopathological syndromes. However, a common limitation of studies of this type, because of the greater time and costs associated with recruitment and experimental testing of patients, is that sample sizes tend to be small and effects tend to be of varying stability. A further, more fundamental limitation to this approach is that it is in fact *quasi*-experimental by nature (cf. Campbell & Stanley, 1966), rather than truly experimental. That is, groups selected to differ in the presence versus absence of a designated diagnostic condition can easily differ in ways that are either distinct from, or not specific to, that condition—such that group differences on experimental measures may reflect variables other than the presence of the condition per se. A common means of controlling for condition-irrelevant confounds is to match groups on such variables as gender, age, and patient status. Along similar lines, the problem of diagnostic effect specificity is commonly dealt with by selection of "pure cases"—that is, individuals possessing only the disorder of interest without co-occurring psychopathology. However, truly "pure" cases (i.e., lacking even subthreshold comorbid conditions) are rare in patient populations and thus unrep-

resentative of cases that come to the attention of clinicians. Related to this, pure cases commonly represent weaker variants of the condition of interest, in terms of severity and chronicity of symptoms—variants in which core neurobiological diatheses may play a lesser etiological role.

A further crucial limitation of both the psychometric–dimensional and the experimental–diagnostic approaches to the neurobiology of psychopathology is that the individual difference (phenotypic) constructs of interest in each case are not directly biological. In the case of the psychometric–dimensional approach, systematic efforts are made to precisely operationalize psychologically oriented individual difference constructs on the basis of self-report items, followed by efforts to map their neurobiological correlates. In some work, individual difference constructs are selected that have neurobiological as well as psychological referents (e.g., Cloninger, 1987; Depue, Luciana, Arbisi, Collins, & Leon, 1994; Gray, 1991); even in work of this kind, however, target phenotypes are operationalized on the basis of nonbiological, self-report-based indicators. Observed correlations between specific psychometric phenotypes and relevant physiological indicators in large samples, although robust, are typically small in magnitude (.30 or lower; e.g., Benning, Patrick, & Iacono, 2005; Hall, Bernat, & Patrick, 2007). This is true for at least three essential reasons. The first is that self-report assessment and physiological response assessment reflect different domains of measurement. As noted many years ago by Campbell and Fiske (1959), indicators of the *same construct* derived from differing measurement domains are expected to correlate only moderately (.30–.60), at best. A second reason is that physiological variables in studies of this type are typically linked too loosely to psychometric trait variables of interest to be considered indicators of the same construct. This further constrains the observed relations between variables across the two domains. Third, whereas self-report trait measures are normally developed through an iterative process of item generation, administration, evaluation, and refinement that results in a final set of indicators with demonstrable reliability for indexing a specified target construct, no such process is typically employed with physiological measures in order to establish their reliability as individual difference measures per se—much less their reliability as indicators of *specified* psychological trait constructs.

In the experimental–diagnostic approach, on the other hand, systematic effort is devoted to precisely operationalizing psychologically meaningful processes on the basis of carefully designed behavioral tasks, followed by application of these tasks to identify processing differences associated with nonbiologically defined phenotypic categories (i.e., diagnostic groups). In clinical neuroscience studies, dependent variables of interest consist of direct brain response measures, including measures such as blood-oxygen-level-dependent (BOLD) MRI that yield precise information regarding anatomical sites of neural activity. However, a number of factors limit the sensitivity and specificity of physiological measures (including direct brain response measures) within isolated tasks as indicators of neural processes relevant to particular diagnostic conditions. Some of these factors mirror those described above in relation to neurobiological studies of self-report trait constructs: Diagnostic and physiological assessments reflect differing measurement domains; constructs tapped by brain response indices in experimental tasks do not directly match constructs tapped by diagnostic symptom indicators; and experimental task paradigms are normally developed for the purpose of operationalizing *normative* psychological processes of interest, without systematic effort devoted to ensuring their effectiveness as *individual difference* measures.

In addition, the experimental–diagnostic approach has some distinctive limitations as a method for elucidating neurobiological factors in psychopathological conditions. One is that diagnostic classification is a dichotomous approach to phenotyping that does not take into account variations in symptom expression or severity. Participants categorized as meeting criteria for a diagnosis can exhibit varying numbers and types of symptoms, with severity of particular symptoms varying from one participant to another, and individuals classified as not meeting criteria for a diagnosis can nonetheless vary in degree of (subthreshold) symptomatology. Furthermore, the pervasive phenomenon of

diagnostic comorbidity ensures that participants meeting criteria for a particular clinical disorder will routinely exhibit symptoms of other disorders as well as features specific to the disorder of interest. In addition, much of this comorbidity is systematic rather than random (i.e., particular disorders co-occur more frequently with disorders of certain types than with others; cf. Krueger, 1999b). As discussed below, the implication is that various disorders share underlying processing deviations in common. As a function of this, group differences in brain (or other physiological) reactivity observed in experimental–diagnostic studies may reflect processes common to disorders of differing types more than they do processes specific to the target disorder of interest.

In summary, both the psychometric–dimensional and the experimental–diagnostic approaches—while informative, respectively, about individual difference dimensions affiliated with varying forms of psychopathology, and underlying processes implicated in disorders of particular types—are nonetheless limited as methods for mapping deviations in the function of particular neurobiological systems onto disorder-relevant phenotypes. In particular, with each of these approaches, a substantial measurement gap exists between phenotypes of interest (either trait constructs or diagnostic categories) and neurobiological systems/processes of interest. As a complement to these existing approaches, directed toward bridging the gap between psychopathological phenotypes and biological systems, we highlight in this chapter a third potential approach we term the "psychoneurometric" approach. Psychoneurometrics can be defined as the systematic development of neurobiologically based trait measures, using psychological (psychometric) phenotypes as referents.

As applied to the study of mental disorders, the goal of this approach is to establish direct *neurophysiological* measures of individual difference constructs relevant to psychopathology that have optimal psychometric properties. Rather than targeting trait constructs from particular models of personality, or discrete diagnostic entities (e.g., as defined in the DSM), the psychoneurometric approach targets relevant neurobehavioral trait constructs (i.e., trait constructs with direct referents in neurobiology as well as behavior). Established psychometric measures of these target constructs serve as initial referents for the identification of reliable indicators in the physiological domain. As illustrated and discussed later (see "Toward a Psychoneurometrics of Psychopathology," below), observed convergences among differing neurophysiological indicators can provide insights into the nature of brain variations relevant to individual difference constructs of interest. This information in turn can be used to refine psychological conceptualizations (and psychometric operationalizations) of these target constructs—and psychopathological conditions with which they are associated.

This chapter focuses on two neurobehavioral constructs in particular: defensive reactivity and inhibitory control. We focus on these constructs because of their demonstrable relevance to differing forms of psychopathology in DSM (Axis II personality syndromes as well as Axis I clinical disorders), and because empirical demonstrations are available of how these constructs can be indexed physiologically as well as behaviorally/psychometrically. The next section below highlights the phenomenon of comorbidity and describes integrative hierarchical models that have been developed to account for this phenomenon in terms of broad factors that disorders share, while at the same time positing distinct lower-order factors that account for unique features of individual disorders. Considering the emphasis assigned to abnormal emotional response and deficient impulse control in the definitions of many different mental disorders, our view is that constructs of defensive reactivity and inhibitory control are particularly relevant to an understanding of processing deviations that differing disorders have in common. The third major section below discusses psychological conceptualizations of defensive reactivity and inhibitory control, highlighting specialized psychometric measures of dispositional fear/fearlessness and disinhibitory (externalizing) tendencies. The fourth section reviews neurobiological conceptualizations of defensive reactivity and inhibitory control (i.e., brain systems relevant to these individual difference constructs) and summarizes evidence regarding neurophysiological correlates of these constructs. The

fifth section describes empirical data linking the constructs of defensive reactivity and inhibitory control, operationalized as dispositional fear and externalizing proneness, to differing forms of psychopathology (Axis II as well as Axis I) within DSM. The sixth section provides an illustration of the psychoneurometric approach, drawing on multiple known electrocortical indicators of externalizing tendencies. The chapter ends with a brief discussion of implications for future research.

Accounting for Comorbidity among Mental Disorders: Hierarchial Models

The classic medical perspective on psychopathology, which served as the foundation for the DSM nosological system, is that individual diagnostic syndromes represent discrete phenotypic entities with distinctive etiological underpinnings. However, a significant challenge to operationalizing mental disorders in this fashion is the well-documented phenomenon of diagnostic comorbidity (e.g., Clark, Watson, & Reynolds, 1995; Kendler, Prescott, Myers, & Neale, 2003; Krueger, 1999b; Vollebergh et al., 2001; Zuckerman, 1999). That is, mental disorders tend not to occur in isolation from one another; rather, they occur more typically in overlapping fashion within the same individual. For example, individuals diagnosed with major depression often exhibit co-occurring anxiety disorders (e.g., social phobia, panic disorder), as well as fearful/anxious personality disorders (e.g., avoidant, dependent). As noted earlier, one reason why the phenomenon of comorbidity poses a significant challenge to neurobiological research on the mechanisms of psychopathology is that in research on particular disorders of interest, it is possible that the etiological process under investigation may be generally characteristic of that disorder as well as others that reliably co-occur with it, rather than specific to the disorder of interest. A second reason is that comorbidity is so prevalent that "pure-case" research (i.e., research focusing on non-comorbid cases of a specific disorder) is likely to be unrepresentative of clinical cases encountered in practice and thus limited in generalizability.

A valuable approach to accommodating the phenomenon of diagnostic comorbidity has been to develop hierarchical models encompassing particular families (spectra) of interrelated mental disorders. These models account for the comorbidity among differing syndromes in terms of a broad factor reflecting their shared variance (overlapping symptomatology), along with specific factors reflecting the unique variance (distinct symptomatology) of particular disorders. From an etiological standpoint, the broad factor can be viewed as reflecting common etiological influences that contribute to all disorders within a spectrum, whereas specific factors reflect narrower etiological influences that determine the unique symptomatic expression of particular disorders. Models of this sort have been developed for anxiety and mood ("internalizing") disorders, and for impulse control ("externalizing") disorders.

In the domain of internalizing psychopathology, Mineka, Watson, and Clark (1998) proposed a hierarchical model in which unipolar depression and various anxiety-related disorders share a common broad factor of "negative affect" (NA), reflecting general distress, susceptibility to negative mood states, and hypervigilance to threat. In addition, Mineka et al. postulated that each individual internalizing disorder has a specific etiological factor accounting for its uniqueness. For example, although depression (like the various anxiety disorders) is associated with heightened NA, it is distinguished by a reduced capacity for pleasurable mood states. Among the anxiety disorders, panic disorder is distinguished from the others by the presence of physiological hyperreactivity ("anxious arousal"), manifested in the form of acute panic attacks.

In a revision of this model, Watson (2005) proposed a nosological distinction between "distress" disorders (comprising major depression, dysthymic disorder, generalized anxiety disorder, and posttraumatic stress disorder [PTSD]) and "fear" disorders (encompassing specific phobia, social phobia, panic disorder, and agoraphobia). This distinction was inspired importantly by Krueger's (1999b) demonstration of separable "anxious misery" versus "fear" disorder subcategories within the internalizing spectrum. In Watson's revised model, these two

subcategories have in common an overarching NA (general subjective distress) factor, but (1) this broad factor accounts for substantially more variance in the distress disorders than in the fear disorders; and (2) the fear disorders are distinguished by the presence of salient physiological hyperarousal (uncued negative activation, in the case of panic disorder; situationally bound negative activation, in the case of the phobic disorders). Relevant to this conceptualization, Sellbom, Ben-Porath, and Bagby (2008) parsed the broad construct of NA into distinctive (albeit correlated) "demoralization" and "dysfunctional negative emotion" components, reflecting general dysphoria/dissatisfaction/helplessness and negative emotional activation, respectively. They demonstrated that demoralization was more strongly characteristic of distress disorders, whereas high negative activation was more strongly characteristic of fear disorders. These authors also reported distinctive associations of low positive affect with major depression (within the distress disorder category) and social phobia (within the fear disorder category).

In the domain of externalizing psychopathology, Krueger and colleagues (2002) demonstrated, in a sample of twins, the existence of a general "externalizing" factor accounting for the shared variance among diverse impulse control disorders within DSM (i.e., child conduct disorder, adult antisocial behavior, alcohol dependence, and drug dependence), along with scores on a self-report measure of disinhibitory personality. These authors estimated that over 80% of the variance in this common externalizing factor was attributable to additive genetic influence (see also Kendler et al., 2003; Young, Stallings, Corley, Krauter, & Hewitt, 2000). In contrast, the residual variance in each of the diagnostic variables as well as the personality variable not accounted for by the broad externalizing factor was accounted for mainly by nonshared environmental influence (with shared environment also contributing specifically to conduct disorder). Krueger and colleagues proposed a hierarchical model based on these findings, in which the general externalizing factor represents a predominantly heritable vulnerability that contributes to the development of diverse traits and problem behaviors, with the precise phenotypic expression of this vulnerability (i.e., as subclinical disinhibitory tendencies, antisocial deviance of different sorts, or alcohol or drug problems) determined by other, more specific etiological influences. Krueger, Markon, Patrick, Benning, and Kramer (2007) extended this work by developing a comprehensive quantitative–hierarchical model to accommodate a broad spectrum of impulse control problems and traits, operationalized as coherent lower-order constructs in the domain of self-report. As described further in the next section below, this work corroborated the existence of an overarching externalizing factor accounting for substantial variance in all traits and problems within this spectrum, and also revealed evidence of distinct subordinate factors (callous aggression, addiction proneness) accounting for residual variance in particular subsets of traits/problems.

These hierarchical models are valuable because they point to a novel two-part strategy for investigating etiological contributions to mental disorders. One part entails studying the nature and bases of broader individual difference factors that contribute to varying disorders within a spectrum (i.e., generalized distress, physiological hyperreactivity, and diminished positive affect in the case of internalizing disorders; general externalizing tendencies, callousness, and addiction proneness in the case of externalizing disorders). This component is essential for dealing with the phenomenon of diagnostic comorbidity—in particular, for differentiating processes that are common to varying disorders from those that are unique to individual disorders. The other part involves studying the aspects of each individual disorder that distinguish it from affiliated disorders within a spectrum. This component is essential to understanding unique influences contributing to the development and maintenance of particular disorders. For example, panic disorder can be understood in part through investigation of factors contributing to generalized distress (which is common to all internalizing disorders) and to physiological hyperreactivity (which is more specific to fear disorders), but also through investigation of unique aspects of panic disorder (e.g., its lack of cue specificity). Studies along these lines can be conducted by using quantitative/statistical methods (e.g., structural modeling) to partition individual

disorders into their broad versus distinctive facets, or by using a case-based strategy in which groups are selected to exemplify one or the other another facet (e.g., individuals with symptoms of differing impulse control disorders can be selected for studies of the etiology of the general externalizing factor; individuals meeting criteria for a single disorder but low on the general externalizing factor can be selected for studies of unique etiological influences contributing to that disorder).

Notably, these hierarchical models have been developed and refined primarily on the basis of diagnostic symptom data and self-report questionnaire measures. Although the broad factors described in these models are presumed to have neurobiological referents (linked to temperament dispositions; see e.g., Clark & Watson, 1999; Patrick & Bernat, 2006), these referents have yet to be elucidated. A primary aim of the current chapter is to summarize empirical evidence linking differing forms of psychopathology (and, in particular, the factors they share) to two individual difference constructs with direct neurobiological as well as behavioral referents: defensive reactivity and inhibitory control. Individual differences in defensive (fear) reactivity are conceptualized as reflecting variations in the sensitivity of the brain's defensive motivational system. The psychometric–dimensional phenotype corresponding to defensive reactivity has been labeled "dispositional fear–fearlessness" or "trait fear" (Kramer, Patrick, Krueger, & Bayevsky, 2010; Patrick & Bernat, 2009b; Vaidyanathan, Patrick, & Bernat, 2009). Individual differences in inhibitory control are posited to reflect variations in the functioning of brain systems that operate to guide and inhibit behavior and to regulate affective response in the service of distal goals. The psychometric–dimensional phenotype corresponding to this dispositional construct has been labeled "disinhibition" (Patrick, Fowles, & Krueger, 2009; Patterson & Newman, 1993; Sher & Trull, 1994) or "externalizing" (Achenbach & Edelbrock, 1978; Krueger et al., 2002; Krueger, Markon, et al., 2007). The sections that follow describe how the individual difference constructs of defensive reactivity and inhibitory control can be conceptualized in psychological and neurobiological terms, and how these constructs relate to varying forms of psychopathology.

Psychological Conceptualizations of Defensive Reactivity and Inhibitory Control

Defensive (Fear) Reactivity

The emotional state of fear has been conceptualized in terms of reactivity of the brain's defensive motivational system, which functions to prime evasive action in the presence of threat cues (Davis, 1992; Fanselow, 1994; Lang, 1995; LeDoux, 1995). The idea of biologically based differences in general fearfulness is plausible from a biological–evolutionary perspective, insofar as tendencies toward greater versus lesser defensive reactivity have differing adaptive value across varying environmental contexts (owing to such factors as resource availability and prevalence of dangers; cf. Lykken, 1995). Individual differences in fear have been featured prominently in theories of temperament and personality. Goldsmith and Campos (1982) posited fearfulness as one of five basic dimensions of temperament, and Buss and Plomin (1984) identified fear as one of two basic trait expressions of negative emotional reactivity (the other being anger) that emerge within the first year of life. A scale assessing proneness to fear (Distress to Novelty) is included in Rothbart's (1981) widely used Infant Behavior Questionnaire (IBQ); Goldsmith, Lemery, Buss, and Campos (1999) examined etiological contributions to IBQ scale scores, and reported a prominent additive genetic contribution to this fear scale. Kochanska (1997) has emphasized variations in dispositional fear as an important moderator of conscience development in children. Timidity in novel situations is also central to Kagan's (1994) concept of inhibited temperament in children, which he views as a trait risk factor for the development of anxiety-related problems.

In the adult personality literature, a trait construct of "harm avoidance," reflecting avoidance of dangerous and unfamiliar situations, is represented in Tellegen's (1982) Multidimensional Personality Questionnaire (MPQ) and in Cloninger's (1987) Tridimensional Personality Questionnaire (TPQ).[1]

Trait fearlessness has also been addressed in the literatures on temperament and personality. For example, the counterpart to the inhibited child in Kagan's theory is the uninhibited or "low-reactive" child, described as nonfearful, venturous in novel situations, and socially assertive (Kagan, 1994; Kagan & Snidman, 1999). Fearlessness is also represented in Zuckerman's (1979) well-known Sensation Seeking Scale (SSS), in its Thrill and Adventure Seeking (TAS) subscale.

To refine conceptualization and psychometric measurement of defensive (fear) reactivity as a dispositional construct, and to examine its etiological foundations, Kramer and colleagues (2010) collected data for the following established fear and fearlessness scales in a large, mixed-gender sample (N = 2,572) of monozygotic and dizygotic twins recruited from the community: the Fear Survey Schedule (FSS; Arrindell, Emmelkamp, & van der Ende, 1984); the Fearfulness subscale of the Emotionality–Activity–Sociability Temperament Inventory (EAS-Fear; Buss & Plomin, 1984); the four subscales (Fear of Uncertainty, Shyness with Strangers, Anticipatory Worry, Fatigability) constituting the Harmavoidance (HA) scale of Cloninger's (1987) TPQ; the TAS subscale of Zuckerman's (1979) SSS; and the three subscales (Fearlessness, Stress Immunity, Social Potency) composing the Fearless Dominance (FD; Benning, Patrick, Hicks, Blonigen, & Krueger, 2003) factor of the Psychopathic Personality Inventory (PPI; Lilienfeld & Andrews, 1996). Confirmatory factor analyses of these various measures revealed the best fit for a model in which all scales loaded substantially on a general, overarching factor (labeled "trait fear"). Fearfulness measures (FSS, EAS-Fear, the four TPQ-HA subscales) loaded positively (M loading = +.69) on this factor, whereas fearlessness measures (the three PPI-FD scales, SSS-TAS) loaded negatively (M = –.58). Given the twin composition of the sample, it was possible to estimate genetic and environmental contributions to scores on this trait fear factor. Its estimated heritability (i.e., percentage of variance in scores attributable to genetic influence; Falconer, 1989) was 74%, with the remaining 26% attributable to nonshared environment. Thus the general disposition toward fear versus fearlessness indexed by these differing psychometric measures (like the broad externalizing factor identified by Krueger et al., 2002) constitutes a highly heritable phenotype.

Inhibitory Control

Psychological theorists since the earliest days of the discipline have recognized a broad dimension of human variation encompassing tendencies toward behavioral restraint versus disinhibition. In his classic *Principles of Psychology*, William James (1890/1983) noted that "there is a type of character in which impulses seem to discharge so promptly into movements that inhibitions get no time to arise" (p. 1144). Along these lines, contemporary theorists in the domains of personality and psychopathology have identified individual difference constructs ranging from "ego control" (Block & Block, 1980) to "constraint" (Tellegen, 1985) to "novelty seeking" (Cloninger, 1987) to "syndromes of disinhibition" (Gorenstein & Newman, 1980). The dimension of behavioral restraint versus impulsivity is also featured prominently in developmental theories of temperament (e.g., Buss & Plomin, 1975; Kochanska, 1997; Rothbart & Ahadi, 1994).

With regard to the psychological bases of impulse control problems, Patterson and Newman (1993) proposed a four-stage model of inhibitory processing to account for the impulsive behavior of disinhibited individuals. These authors posited that the processing deviation most germane to general disinhibitory tendencies (also known as "general proneness to externalizing"; Krueger et al., 2002; Krueger, Markon, et al., 2007) entails impairments at stages 3 and 4 of this model. Stage 3 represents the stage at which the occurrence of a conflictual event normally prompts a shift from an ongoing, goal-oriented response set to a passive, information-gathering set. According to Patterson and Newman, impairments at this processing stage have implications both for inhibition of immediate ongoing behavior at stage 3, and for the formation or strengthening of associative representations crucial to prospective reflection (i.e., inclination to anticipate potential consequences of one's actions) at stage 4. Patterson and Newman posited that this mechanism is crucial to an understanding of disinhibited behav-

ior associated with a variety of syndromes, including antisocial/psychopathic behavior, substance dependence (i.e., early-onset alcoholism), and attention-deficit/hyperactivity disorder (ADHD).

In an effort to refine psychometric measurement and conceptualization of inhibitory control as a dispositional construct, Krueger, Markon, and colleagues (2007) used traditional as well as more contemporary item-analytic methods (including item response modeling, exploratory factor analysis, and hierarchical cluster analysis) to develop a new self-report-based instrument, the Externalizing Spectrum Inventory (ESI), for comprehensively assessing the domain of externalizing problems and traits in terms of coherent lower-order constructs. The ESI includes 23 unidimensional scales developed to measure distinctive constructs, including varying forms of impulsiveness; differing types of aggression (physical, relational, and destructive); irresponsibility; rebelliousness; excitement seeking; blame externalization; and alcohol, drug, and marijuana use/problems. Confirmatory factor analyses of these 23 scales yielded evidence of a overarching factor (externalizing) on which all subscales loaded substantially (.45 or higher), and two subordinate factors (callous aggression, addictions) that accounted for residual variance in particular subscales. As noted earlier, these findings provide further support for the idea that a common dispositional factor (externalizing) contributes to a broad array of impulse control problems and affiliated traits. In addition, they suggest that separate dispositional factors shape the expression of externalizing tendencies toward callous aggression on the one hand, and addictive behaviors on the other.

Neurobiological Bases and Physiological Correlates

Defensive (Fear) Reactivity

As noted, the emotional state of fear is presumed to reflect activation of the brain's defensive motivational system. The amygdala in particular has been described as a core component of the defensive (fear) system in mammals (Davis, 1992; Fanselow, 1994; LeDoux, 1995). Research with adult human participants has demonstrated a role for genetic factors in individual differences in fear conditioning (Hettema, Anna, Neale, Kendler, & Fredrikson, 2003) and has revealed associations between specific gene alleles and variations in reactivity of the amygdala to fear stimuli (e.g., Harari et al., 2002). Young children exhibiting what Kagan has described as disinhibited temperament show reduced amygdala reactivity to novel human faces, when tested as adults, compared with individuals classified as inhibited (Schwartz, Wright, Shin, Kagan, & Rauch, 2003).

However, it is important to note that the amygdala represents only one element of the circuitry involved in defensive motivational processing and activation. For example, the bed nucleus of the stria terminalis shares close connections with the amygdala and has been hypothesized to form part of an extended amygdala system that governs more enduring (tonic) activation in relation to strong or persistent stressors (Davis, Walker, & Lee, 1997). The amygdala also interacts with higher brain regions that govern such processes as directed attention, declarative memory, and response inhibition (Davidson, Putnam, & Larson, 2000; LeDoux, 1995). Thus abnormal levels of negative emotional reactivity can reflect deviations in the functioning of other brain structures besides the amygdala (cf. Curtin, Patrick, Lang, Cacioppo, & Birbaumer, 2001; Patrick & Lang, 1999). Furthermore, the amygdala does not appear to function strictly as a fear activation system. There is evidence for its involvement in detecting unfamiliar stimuli more generally, in prioritizing attention to stimuli in the environment, and in activating positive as well as negative emotion (Lang, Bradley, & Cuthbert, 1997). Thus deviations in amygdala functioning may be associated with abnormalities in other types of processing aside from fear.

One methodology that has proven effective as an index of defensive reactivity to aversive stimuli is potentiation of the startle reflex to an intervening noise probe, measured via the eyeblink response in humans or via the whole-body "jump" reaction in animals. Davis and colleagues (e.g., Davis, 1989; Davis, Falls, Campeau, & Kim, 1993) mapped the neural circuitry of fear-potentiated startle in animals, establishing that the mechanism for this effect is a path-

way from the central nucleus of the amygdala to the nucleus reticularis pontis caudalis, the brainstem node of the basic startle circuit. In humans, the startle blink response to sudden noise is reliably enhanced during viewing of aversive pictures compared with neutral pictures (Lang, 1995; Lang, Bradley, & Cuthbert, 1990). Blink potentiation is strongest for directly threatening images (e.g., aimed weapons, menacing attackers), although it also occurs less reliably for vicarious aversive scenes involving physical injury or aggression (Bernat, Patrick, Benning, & Tellegen, 2006; Bradley, Codispoti, Cuthbert, & Lang, 2001; Levenston, Patrick, Bradley, & Lang, 2000). This effect in humans is blocked by diazepam (Patrick, Berthot, & Moore, 1996), a drug that inhibits activity in the amygdala, and that has also been shown to block fear-potentiated startle in animals (Davis, 1979).

There is also evidence for the specificity of aversive startle potentiation as an index of fear. Davis and colleagues (1997) presented evidence that fear-potentiated startle, associated with phasic (time-limited) increases in defensive activation tied to an explicit aversive cue, is mediated by the central nucleus of the amygdala, whereas startle reflex sensitization, associated with more tonic (prolonged) states of negative emotional activation, is mediated by the bed nucleus of the stria terminalis (BNST). From this standpoint, startle potentiation during discrete aversive cuing holds potential as a physiological indicator of individual differences in fear reactivity in humans. In this regard, increased startle potentiation during viewing of fear-relevant scenes has been demonstrated in individuals with phobic disorders (e.g., Hamm, Cuthbert, Globisch, & Vaitl, 1997; Vrana, Constantine, & Westman, 1992); and deficient fear-potentiated startle is reliably observed in incarcerated offenders diagnosed with psychopathy (cf. Patrick & Bernat, 2009b), a condition theorized to entail a deficiency in fear. In contrast with results for individuals with phobic fears, patients diagnosed with distress disorders such as depression and PTSD show normal fear-potentiated startle in relation to discrete aversive cues, but enhanced startle sensitization under conditions of prolonged stress or uncertainty (Grillon & Baas, 2003). The implication is that increased startle potentiation reflects heightened cue-specific defensive reactivity in individuals with phobic fear disorders, whereas enhanced startle sensitization reflects a more pervasive anxiety process (perhaps akin to high generalized NA; Watson, 2005) in individuals with distress disorders (cf. Davis et al., 1997; Rosen & Schulkin, 1998). Findings for panic disorder are more mixed: Although the general trend of evidence points to enhanced startle sensitization in clinic patients diagnosed with panic, rather than enhanced cue potentiation, the moderating role of comorbid distress disorders (e.g., depression, generalized anxiety) needs to be considered in studies employing patients seeking treatment for panic disorder. In this regard, a recent study by Melzig, Weike, Zimmermann, and Hamm (2007) found that patients with panic disorder but *without* comorbid depression, in relation to healthy controls, showed enhanced potentiation of startle during exposure to a threat cue; in contrast, patients with panic disorder *and* comorbid depression showed no such augmentation of threat-potentiated startle. These authors concluded that patients with panic but not depression respond like individuals with other fear disorders (e.g., specific or social phobia), whereas patients with panic and comorbid depression respond more like individuals with distress disorders.

Individual differences in startle reflex potentiation during aversive cuing have also been reported in relation to scores on differing psychometric scale measures of fear, fearlessness, and psychopathy—including the FSS (cf. Cook, 1999), the TPA-HA scale (Corr et al., 1995; Corr, Kumari, Wilson, Checkley, & Gray, 1997), the SSS-TA scale (Lissek & Powers, 2003), and the FD factor of the PPI (Benning, Patrick, Blonigen, Hicks, & Iacono, 2005). As noted earlier, these scale measures function as high- and low-pole indicators, respectively, of a common "trait fear" dimension (Kramer et al., 2010). Based on the known bivariate relations of these varying scale indicators with magnitude of aversive startle potentiation, Vaidyanathan and colleagues (2009) tested the hypothesis that aversive startle potentiation represents a continuous *physiological* indicator of this underlying trait fear dimension. Participants in this study were college men and women (N = 88) who were

administered the FSS, the EAS-Fear and SSS-TAS scales, and the subscales constituting the TPQ-HA and PPI-FD. Participants were tested in an affect–startle procedure that included differing categories of aversive (threat, physical injury, other-attack) and pleasant picture stimuli (erotic, action, nurturant) along with neutral pictures. Consistent with the findings of Kramer and colleagues (2010), a principal-components analysis of these various trait scales in this test sample yielded evidence of a dominant first factor on which all scales loaded substantially. An omnibus index of trait fear was computed for each participant, consisting of scores on the first component from this analysis. A modest but statistically robust linear relationship was found (in the sample as a whole, and for male and female subgroups separately) between trait fear and startle modulation for threat pictures in particular—the picture category, as noted earlier, that is most directly fear-relevant and yields the most reliable startle potentiation effects (see Figure 22.1). The findings of this study confirm that aversive startle potentiation represents a physiological indicator of the psychometric trait fear dimension, and lend support to the idea that these two variables (startle potentiation, trait fear) represent indices of a common neurobehavioral trait construct (i.e., reactivity of the defensive motivational system).

As discussed further in the final section below, multiple physiological indicators of trait fear will be required to establish a direct physiological index of individual differences in defensive reactivity with effective psychometric properties. Thus the demonstration of an association between aversive startle potentiation and trait fear represents only an initial step in this direction. To proceed further, systematic research will need to be undertaken to identify additional physiological indicators of trait fear. In this regard, the available literature suggests a variety of potential candidates. For example, another method that has been used to index

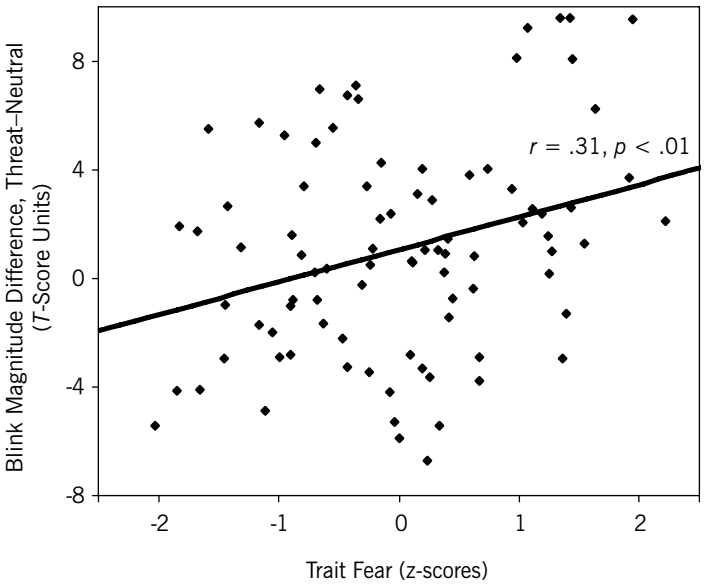


FIGURE 22.1. Scatterplot of the association, within a sample of male and female college students (N = 88), between trait fear scores and startle potentiation scores, defined as average magnitude of blink response to noise probes during viewing of direct-threat pictures (aimed weapons, menacing attackers) minus average magnitude of blink response during viewing of neutral pictures. Solid line is best-fitting regression line. Trait fear scores consist of scores on the first principal component derived from a principal-components analysis of varying self-report measures of fear and fearlessness. From Vaidyanathan, Patrick, and Bernat (2009). Copyright 2009 by Wiley-Blackwell. Reprinted by permission.

individual differences in fear and fearlessness involves measurement of responses to affective face stimuli. There is evidence that emotional facial expressions (fearful expressions in particular) reliably activate the amygdala in humans (e.g., Morris et al., 1996; Whalen, 1998), and that individuals high in NA show increased amygdala reactivity to fear faces (e.g., Bishop, Duncan, & Lawrence, 2004). There is also evidence that individuals low in dispositional fear show diminished reactivity to fearful expressions. For example, individuals high in psychopathy show reduced behavioral and brain response to emotional face stimuli, particularly fearful faces (cf. Blair, 2006). Notably, research on youth with conduct problems has demonstrated reduced behavioral (Blair, Colledge, Murray, & Mitchell, 2001) and amygdala (Marsh et al., 2008) reactivity to fear faces, specifically in children with callous–unemotional traits—akin to the emotional and interpersonal features of psychopathy in adulthood, which have been linked to deficits in fear-potentiated startle (cf. Patrick & Bernat, 2009b; see next major section below). However, it is important to note that amygdala damage does not invariably result in impaired recognition of fear faces, or impairments limited to processing of fearful expressions (Adolphs et al., 1999). Furthermore, as noted earlier, there is compelling evidence that the amygdala plays a role in the processing of positive as well as negative emotional events. Thus it remains to be determined whether impairments in facial affect processing in psychopathy reflect deficits in fear tied specifically to the amygdala, or broader impairments in emotional sensitivity extending beyond the amygdala.

Related to the use of visual stimuli such as fearful faces to index individual differences in reactivity of the core defensive system, a key issue to consider is the nature of the processing task used to assess brain reactivity differences. For example, standard picture-viewing tasks in which pictorial stimuli are presented individually for durations of several seconds are likely to elicit activation in diverse regions of the brain associated with processing at varying levels (e.g., emotional, attentional, memorial–imaginal, etc.). Tasks in which stimuli are presented fleetingly (e.g., Junghoefer, Bradley, Elbert, & Lang, 2001) or under conditions that preclude higher elaborative processing (e.g., Öhman & Soares, 1994) may be particularly useful for evaluating individual differences in affective sensitivity at the primary subcortical (amygdala) level. In this regard, an intriguing new methodology for investigating low-level affective processing is the interocular suppression paradigm, a variant of binocular rivalry in which stimuli are presented "invisibly" to one eye by presenting a more salient, imperative visual stimulus concurrently to the other eye. Using functional MRI, Jiang and He (2006) demonstrated that, in contrast with results from a normal (visible) viewing condition in which fearful faces were seen to activate the amygdala by way of the fusiform face area, under conditions of interocular suppression fearful faces activated the amygdala via the superior temporal sulcus—indicating that processing in this condition was limited to more primitive pathways. Jiang and colleagues (2009) have replicated and extended these findings, using event-related potential (ERP) measures. Although this technique has not been used to date in the study of individual differences in affect, it holds clear potential in this regard.

Other task procedures in which physiological measures have been used to index individual differences in defensive reactivity include aversive conditioning tasks (e.g., Flor, Birbaumer, Hermann, Ziegler, & Patrick, 2002), procedures involving anticipation of an impending stressor (cf. Hare, 1978), and dual-attention tasks in which fear cues are presented incidentally in conjunction with primary task cues (e.g., Curtin et al., 2001; Dvorak-Bertsch, Curtin, Rubinstein, & Newman, 2007). Existing tasks such as these provide further avenues for identifying physiological indicators of trait fear. It seems likely that some candidate physiological measures of defensive reactivity will ultimately prove more effective as indicators of general distress (reflecting hyperreactivity of the extended amygdala system) than of trait fear. For example, as noted above, there is evidence that enhanced sensitization of the startle response under conditions of prolonged stress or uncertainty operates as an index of general anxiety/distress. Other physiological indices that may emerge as indicators of general distress as opposed to trait fear include persistence of startle potentiation following the offset of a discrete

aversive stimulus (Jackson et al., 2003) and right frontal cerebral hemispheric asymmetry (Davidson, Pizzigalli, Nitschke, & Putnam, 2002; Heller & Nitschke, 1998).

Inhibitory Control

What brain systems/mechanisms underlie individual differences in the capacity to regulate affective and behavioral expression and to constrain impulses? Several lines of evidence point to anterior brain structures, including the prefrontal cortex (PFC) and anterior cingulate cortex (ACC), as playing crucial roles in this processing domain. With regard to the PFC, lesions of frontal brain regions are known to result in impulsive, externalizing behavior (Blumer & Benson, 1975; Damasio, Tranel, & Damasio, 1990), and individuals exhibiting or at risk for impulse control problems of various types show deficits on neuropsychological tests of frontal lobe function (Barkley, 1997; Morgan & Lilienfeld, 2000; Peterson & Pihl, 1990; Tarter, Alterman, & Edwards, 1985). The PFC is thought to be important for "top-down" processing—that is, the guidance of behavior by internal representations of goals or states. The PFC appears to be especially important for coping with novel or dynamic situations in which selection of appropriate behavioral responses needs to be made on the basis of internal representations of goals and strategies, rather than immediate stimulus cues alone (e.g., Cohen & Servan-Schreiber, 1992; Miller, 1999; Wise, Murray, & Gerfen, 1996). Miller and Cohen (2001) have proposed that the control functions of the PFC arise from its specialized capacity for online maintenance of goal representations: By maintaining patterns of activation corresponding to goals and strategies required to achieve them, the PFC provides biasing signals to other regions of the brain with which it connects. These signals serve to prime sensory–attentional, associative, and motor processes that support the performance of a designated task, by directing activity along relevant brain pathways.

In this regard, the PFC includes subdivisions that play differing roles in the guidance of behavior. The dorsolateral PFC, which has close connections with sensory association cortices and projects to varying premotor and motor areas in the medial and lateral frontal lobes, operates to encode relations between stimulus events and thereby represent rules (mappings) required to perform complex tasks. It is particularly important for active processes that involve top-down ("cognitive") control of behavioral responses (cf. Petrides, 2000). Ventromedial and orbitofrontal regions of the PFC (collectively termed the orbitomedial PFC; Blumer & Benson, 1975) connect more directly and extensively with medial temporal limbic structures (including the amygdala, hippocampus and associated neocortex, and hypothalamus) and appear to play a greater role in the anticipation of affective consequences of behavior (Bechara, Damasio, Tranel, & Damasio, 1997; Wagar & Thagard, 2004), in the unlearning of stimulus–reward associations (i.e., reversal learning) (Dias, Robbins, & Roberts, 1996; Rolls, 2000), and in the regulation of emotional reactivity and expression (Damasio et al., 1990; Davidson et al., 2000).

One brain response measure that has demonstrated reliable associations with differing forms of disinhibitory psychopathology is the P300 (or P3; see below)—a positive brain potential response, maximal over parietal scalp regions, that follows the occurrence of infrequent, attended targets in a stimulus sequence. It has long been known that individuals with (or at risk for) alcohol problems show reduced P300 response amplitude (e.g., Begleiter, Porjesz, Bihari, & Kissin, 1984; Porjesz, Begleiter, & Garozzo, 1980; for a review, see Polich, Pollock, & Bloom, 1994). More recent studies have shown reduced parietal P300 in relation to various other externalizing disorders, including drug dependence (e.g., Attou, Figiel, & Timsit-Berthier, 2001; Biggins, MacKay, Clark, & Fein, 1997), nicotine dependence (e.g., Anokhin et al., 2000), child conduct disorder (e.g., Bauer & Hesselbrock, 1999a, 1999b), and adult antisocial personality disorder (e.g., Bauer, O'Connor, & Hesselbrock, 1994). In addition, reduced P300 is known to be associated with risk for these other disorders, as well as with active symptoms (Brigham, Herning, & Moss, 1995; Iacono, Carlson, Malone, & McGue, 2002).

Patrick et al. (2006) tested the hypothesis that reduced P300 amplitude might reflect generalized externalizing vulnerability in a large sample of male twins (*N*

= 969). Higher scores on the externalizing factor estimated from symptoms of conduct disorder, adult antisocial behavior, alcohol dependence, drug dependence, and nicotine dependence were robustly associated with reduced amplitude of P300 response, and mediation analyses revealed that externalizing factor scores accounted for associations between individual diagnostic variables and reduced P300 amplitude. Furthermore, a principal-components analysis in which the diagnostic measures of externalizing were included along with P300 amplitude yielded a single dominant factor on which all indicators showed significant loadings. The fact that P300 loaded significantly (albeit modestly; see Figure 22.2) with the symptom variables on a common factor, rather than defining a separate method component, indicated that it was tapping the same underlying construct as the symptom variables. In a follow-up study that capitalized on the twin composition of this sample, Hicks and colleagues (2007) subsequently demonstrated that the relationship between externalizing factor scores and reduced P300 amplitude in this sample was attributable to overlapping genetic influences. These results indicate that reduced P300 directly reflects some alteration in brain function associated with the broad, strongly heritable vulnerability to disorders within the externalizing spectrum. Although for many years the P300 has been viewed as a distributed brain response reflecting activity in multiple brain regions, recent research on the neutral generators underlying this response points to an important role for prefrontal brain regions (see, e.g., Dien, Spencer, & Donchin, 2003; Nieuwenhuis, Aston-Jones, & Cohen, 2005).

Relevant to this, a follow-up study by our laboratory group has produced evidence of

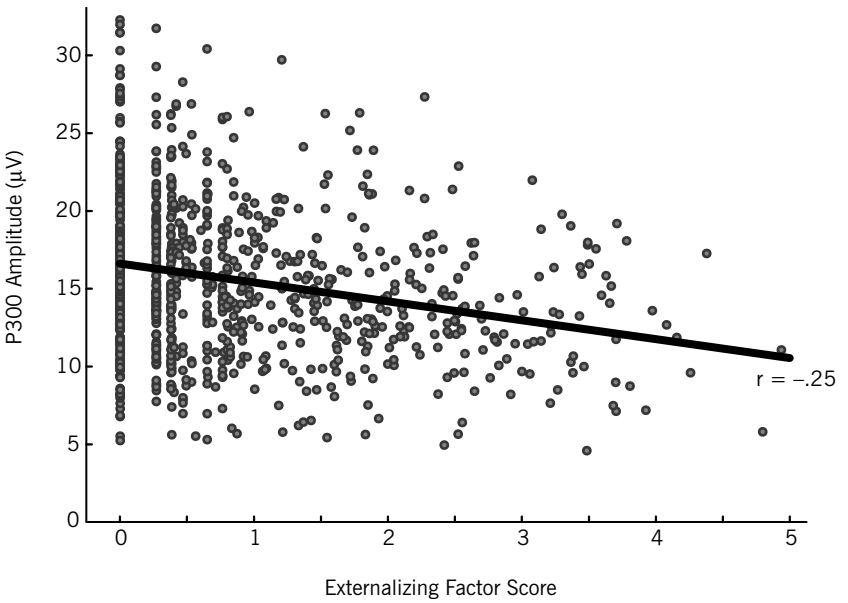


FIGURE 22.2. Scatterplot of the association, within a sample of male adolescents recruited from the community (N = 969), between mean amplitude of P300 response to task stimuli in a visual oddball procedure and continuous scores on the broad externalizing factor derived from a principal-components analysis of symptom scores for varying impulse control disorders. Solid line is best-fitting regression line. A standard geometric transformation was applied to the raw factor score data in order to vertically align low scores on externalizing; the data were normalized to unit-length axes prior to rotation and rescaled to the original units afterward. For purposes of plotting, the minimum score on the externalizing factor was subtracted from each resultant value, such that the 0 point on the x-axis represents the minimum score rather than the mean. From Patrick et al. (2006). Copyright 2006 by Blackwell Publishing. Reprinted by permission.

an augmented association between externalizing tendencies and diminished P300 at frontocentral versus parietal scalp sites, particularly for novel task stimuli that are known to preferentially activate anterior brain regions. Externalizing tendencies in this study were indexed by overall scores on an abbreviated (100-item) screening version of the ESI. The experimental task consisted of a three-stimulus visual oddball task (Bernat, Patrick, Cadwallader, van Mersbergen, & Seo, 2003). In addition to frequent nontarget (oval) and less frequent target (schematic "head") stimuli requiring a response, the task included infrequent novel stimuli, consisting of color picture stimuli. Target stimuli in a task of this sort elicit a P300 response that is maximal at parietal scalp sites. In contrast, novel stimuli evoke a P300 response—termed the "novelty P3" (Courchesne, Hillyard, & Galambos, 1975), to distinguish it from the target P300 (P3) response—that is maximal at frontocentral scalp sites. Available data indicate a prominent role of lateral PFC in the processing of novel stimuli (see Nieuwenhuis et al., 2005), and ERP source localization work points to a supporting role for the ACC in the generation of the novelty P3 response (Dien et al., 2003).

This study replicated our prior finding of reduced P3 amplitude to target stimuli as a function of higher externalizing tendencies—in this case, defined by scores on a carefully designed self-report inventory, the ESI, rather than by disorder symptoms as in the Patrick and colleagues (2006) study. In addition, we found a robust negative relationship between externalizing tendencies and P3 response to novel (picture) stimuli. The negative association between externalizing and P3 was stronger at anterior than at posterior scalp sites, particularly in the case of the novel picture stimuli. The enhanced magnitude of this effect at anterior sites is consistent with the hypothesis that the association between reduced P3 and externalizing reflects a deviation of some kind in frontal brain processing.

Another brain region important to regulating behavior is the ACC, which connects with premotor and supplementary motor regions as well as limbic structures (including the amygdala and hippocampus) and the PFC. The ACC has been conceptualized as a system that invokes the control functions of the PFC as needed to perform a task successfully, either by detecting errors in performance as they occur (Gehring, Coles, Meyers, & Donchin, 1995; Scheffers, Coles, Bernstein, Gehring, & Donchin, 1996), by monitoring conflict among competing response tendencies (Carter et al., 1998), or by estimating the likelihood of committing an error at the time a response is called for (Brown & Braver, 2005). The ACC has also been implicated in affective–evaluative processing, with rostral–ventral areas thought to be more involved in the processing of emotional information, and dorsal areas more involved in the processing of cognitive information (Bush, Luu, & Posner, 2000). Impairments in ACC function would be expected to interfere with the ability to inhibit prepotent behavioral responses, to mediate between conflicting action tendencies, and to avoid repetition of errors.

In addition to studying reduced P300, we have undertaken investigations of the error-related negativity (ERN) as a physiological indicator of disinhibitory/externalizing tendencies. The ERN, a brain potential response that occurs following errors in performance, is believed to arise from the ACC (Dehaene, Posner, & Tucker, 1994; Holroyd, Dien, & Coles, 1998; Luu, Flaisch, & Tucker, 2000; Miltner, Braun, & Coles, 1997). Two variants of the ERN have been documented in the literature: (1) the response ERN (rERN), which occurs following errors in a speeded performance task in the absence of feedback; and (2) the feedback ERN (fERN), which occurs in response to feedback signaling an undesired (loss) outcome. The rERN been interpreted as the product of an endogenous (internal) action-monitoring process that relies upon ongoing maintenance of a task directive in order to register the occurrence of errors (Holroyd & Coles, 2002; Holroyd, Larsen, & Cohen, 2004; Mars et al., 2005). The fERN, on the other hand, has been interpreted as a direct response to an exogenous (external) error cue. Recent published work by our group (Hall et al., 2007) has demonstrated significantly reduced rERN response following performance errors in a flanker task in individuals high in externalizing tendencies as indexed by the ESI-100. This finding coincides with other published work demonstrating reduced rERN in relation to

disinhibitory personality traits (Dikman & Allen, 2000; Pailing & Segalowitz, 2004) and states (Ridderinkhoff et al., 2002).

However, in a recent follow-up study (Bernat, Nelson, Steele, Patrick, & Gehring, 2010), we found no evidence of reduced fERN in high-externalizing individuals following *externally presented* loss feedback in a simulated gambling task. A technical challenge in this study was that the negative-going fERN response reflecting the motivational impact of the *loss* component of feedback overlapped in time with a positive-going P300 component reflecting elaborative postevent processing of feedback stimuli per se (i.e., beyond initial encoding of its gain–loss significance). As a consequence of this overlap, individuals high in externalizing appeared, if anything, to show *enhanced* negativity of response during the time window of the fERN—an effect clearly opposite to our prediction. To clarify this result, we employed a method called "time–frequency analysis" (Bernat, Williams, & Gehring, 2005)—a technique for separating distinct but overlapping ERP components by considering differences in underlying oscillatory (frequency) characteristics of one versus another—to isolate two distinct components underlying the ERP response to feedback stimuli: (1) a higher-frequency (theta band) component reflecting the fERN response to loss stimuli in particular, and (2) a lower-frequency (delta band) component reflecting the generic P300 response to gain as well as loss feedback stimuli. We found that high-externalizing individuals showed normal theta fERN activity following loss feedback, while showing significantly reduced P300 response to feedback stimuli in general (i.e., whether indicative of gain or loss). This pattern of results has two important implications. First, it indicates that reduced rERN response in high-externalizing individuals does not reflect a deficit in ACC function per se, but rather processing impairments in other brain regions crucial for endogenous action monitoring (e.g., regions of PFC) but not for registration of external performance feedback. Second, it indicates that deficits in inhibitory control associated with disorders of this type entail impairments in associative–elaborative processing of events (e.g., comparing and integrating transient cognitive–affective representations with representations stored in long-term memory; cf. Ericsson & Kinsch, 1995), rather than impairments in primary affective processing.

In summary, evidence to date points to dysfunctions in anterior brain circuitry, including the PFC and affiliated brain regions with which it interacts (such as the ACC), as a substrate for deficient inhibitory control. The consequence of an underlying weakness in this circuitry would be a propensity to act on the basis of salient cues in the immediate environment, rather than on the basis of internal representations of goals and methods for achieving them (cf. Miller & Cohen, 2001). As described by Patterson and Newman (1993), a weakness of this sort would impair an individual's ability to shift from an ongoing response set to a reflective orientation in the face of conflict, resulting in a failure to modify subsequent actions on the basis of undesirable outcomes. Furthermore, dysfunction in the PFC–ACC systems would compromise an individual's ability to (1) ascribe motivational significance to representations for more complex and distal, but ultimately more fulfilling, behavioral goals; (2) anticipate obstacles and formulate strategies for overcoming them before they become overwhelming (e.g., deal proactively with frustrating or threatening circumstances); (3) detect conflict between competing response tendencies (i.e., evaluate, online, the probability of making an error); and (4) monitor and regulate affective responses in the service of distal goals. As an example of this, Davidson and colleagues (2000) proposed that persistent impulsive–aggressive behavior arises from dysfunctions in PFC–ACC circuitry that lead to impairments in online conflict detection and down-regulation of negative affect.

Role of Defensive Reactivity and Inhibitory Control in Psychological Disorders

Internalizing Disorders

Fearfulness has both adaptive and maladaptive aspects. Defensive activation in the presence of threat cues is biologically adaptive, and thus normative. However, fear that is disproportionately intense or persistent in relation to evoking circumstances is consid-

ered pathological. As noted earlier, internalizing disorders appear to fall into two distinct subgroups: fear disorders and distress (anxious misery) disorders (Krueger, 1999b; Watson, 2005). Fear disorders are characterized by physiological hyperreactivity, either in relation to external eliciting stimuli (in the case of specific and social phobia) or internal physiological cues (in the case of panic disorder), whereas distress disorders are marked by prominent anxiety and dysphoria not tied to specific eliciting cues. Individuals who exclusively manifest fear disorders tend to be better adjusted psychologically and show less impairment in areas of life unrelated to their fears than individuals exhibiting distress disorders (Cook, Melamed, Cuthbert, McNeil, & Lang, 1988; Cuthbert et al., 2003). The implication is that fear disorders in themselves are less pathological conditions, reflecting extreme variants of normative fearfulness, in comparison with distress disorders.

What factors give rise to the more pathological, dysregulated NA characteristic of distress disorders? Rosen and Schulkin (1998) have theorized that the pathological anxiety observed in disorders like PTSD reflects hyperexcitability of brain systems that underlie fear expression, particularly the amygdala and extended amygdala. According to Rosen and Schulkin (1998), constitutional differences in the threshold for activation of these underlying brain systems, in conjunction with adverse experiences that activate them repeatedly or for protracted periods, lead to sensitization of these systems—manifested psychologically as intense and persistent negative mood, hypervigilance, and a sense of uncontrollability (i.e., the defining elements of distress disorders). Consistent with this model, we hypothesize that high dispositional defensive reactivity—defined in neurobiological terms as constitutionally high reactivity of the amygdaloid fear system—represents one crucial vulnerability factor for the development of internalizing disorders. As noted earlier, there is evidence that individual differences in reactivity of this system have a genetic contribution (e.g., Harari et al., 2002) and are evident behaviorally from a very early age (Goldsmith & Campos, 1982; Kagan, 1994). Reciprocally, there is evidence that individuals high in core personality features of psychopathy, who (as noted earlier) exhibit impairments in fear reactivity as indexed by startle potentiation, and who demonstrate reduced amygdala reactivity to fearful face stimuli (Blair, 2006; Marsh et al., 2008), show relative immunity to internalizing symptoms and syndromes (Blonigen et al., 2005; Hicks & Patrick, 2006).

Because the psychometric operationalization of defensive reactivity as trait fear focuses on scales that index the emotional experience of fear (or lack of it) in relation to events and situations, this construct is likely to have relevance to fear disorders—in particular, specific and social phobias, which by definition entail fear that is tied to specific stimuli and situations. However, if dispositional hyperreactivity of the core defensive motivational system contributes to pathological anxiety syndromes as postulated by Rosen and Schulkin (1998), high trait fear should also exhibit some association with internalizing disorders in the "distress" subcategory. Relevant to this, Table 22.1 presents data from a mixed-gender sample of adults from the community (N = 187) who were assessed for trait fear with an abbreviated 55-item screening inventory (TF-55) consisting of selected items from the various fear and fearlessness inventories examined by Kramer and colleagues (2010) that provided highly effective estimation (cross-validated multiple R = .94) of scores on the general fear–fearlessness factor emerging from their structural analysis.[2] Participants in this sample were also administered the Inventory of Depression and Anxiety Symptoms (IDAS; Watson et al., 2007), a self-report inventory designed to assess for symptoms of depression and affiliated anxiety disorders. The inventory includes (1) a broad scale assessing dysphoria (i.e., the overarching mood component of depression, indexed separately from its accompanying symptoms); (2) subscales assessing specific domains of depressive symptoms; (3) a broad scale indexing overall depressive symptoms (composed of selected items from the broad dysphoria scale and narrower depressive symptom scales); and (4) subscales assessing symptoms of anxiety disorders that commonly co-occur with depression (i.e., social phobia, panic disorder, PTSD).

Consistent with expectation, trait fear scores were correlated most robustly with

TABLE 22.1. Correlations of Trait Fear Scores with Broad Dysphoria/Depression Scales and Specific Symptom Subscales of the Inventory of Depression and Anxiety Symptoms (IDAS; Watson et al., 2007)

IDAS scale/subscale score	*r*	$r_{[/\text{Social Anxiety}]}$	$r_{[/\text{Dysphoria}]}$
Broad Dysphoria scale	.36**	.10	—
Broad Depression scale	.35**	.12	.05
Depression symptom subscales			
Lassitude	.26**	.03	.01
Insomnia	.14	–.01	–.10
Suicidality	.18*	.05	.10
Appetite Loss	.05	–.11	–.17
Appetite Gain	.21*	.06	.06
Ill Temper	.26**	.08	.05
Well-Being	–.31**	–.25*	–.24*
Anxiety symptom subscales			
Social Anxiety	.49**	—	.37**
Panic	.13	–.08	–.09
Traumatic Intrusions	.18*	.00	–.04

Note. Sample consisted of 187 adult men and women recruited from the community. Trait fear scores are total scores on a 55-item inventory composed of items from various established self-report measures of fear and fearlessness (for details, see Kramer et al., 2010). $r_{[/\text{Dysphoria}]}$, partial correlation between trait fear and IDAS scale/subscale after controlling for scores on the IDAS Dysphoria scale. $r_{[/\text{Social Anxiety}]}$, partial correlation between trait fear and IDAS scale/subscale after controlling for scores on the IDAS Social Anxiety scale.
*$p < .05$. **$p < .001$.

symptoms of social anxiety (i.e., social phobia; Table 22.1, leftmost *r* column). However, trait fear scores also evidenced reliable associations with dysphoric mood and overall depressive symptoms, as well as with some specific symptoms of depression—most notably lassitude (fatigability), ill temper (irritability), and low well-being. Trait fear also showed a reliable association with proneness to intrusive thoughts/images characteristic of PTSD but only a marginal relationship ($p = .07$) with discrete bodily symptoms characteristic of panic disorder. Furthermore, partial correlations depicted in the middle *r* column of Table 22.1 indicate that bivariate correlations of trait fear with broad dysphoria/depression and most affiliated symptoms of depression were accounted for by anxiety experienced in relation to social events/situations (i.e., social anxiety symptoms); in contrast, the association between trait fear and social anxiety symptoms remained highly significant after controlling for depressed mood (dysphoria; see Table 22.1, rightmost *r* column). These results are consistent with the idea that high dispositional fear is central to phobic disorders in particular, but that it also plays some role in distress-related syndromes.

A crucial question that remains to be answered is what factors lead some highly fearful individuals to develop disorders marked by generalized distress (i.e., chronic anxiety and dysphoria), whereas others develop only focal phobias. The answer, we believe, lies in other dispositional variables and pathogenic environmental influences that interact with high trait fear (hyperreactivity of the amygdala to aversive events) to promote sensitization of not only the core fear system but also its affiliated "anxiety system" (i.e., the extended amygdala/BNST). For example, dispositional factors that enable an individual to counteract or constrain defensive reactions to discrete environmental stressors, and thereby to limit the intensity and duration of fear episodes, would operate against general sensitization. On the other

hand, exposure to highly intense, repeated, unpredictable stress (e.g., such as that experienced by combat veterans or victims of chronic abuse) would operate to enhance sensitization (cf. Rosen & Schulkin, 1998). The availability of direct neurophysiological assessment of dispositional fear would provide a basis for prospective longitudinal studies of how vulnerability in the form of enhanced responsiveness of the core (amygdala) defensive system gives rise to distress or fear disorders of differing kinds as a function of other intersecting constitutional and environmental factors.

Externalizing Disorders

The conceptual model proposed here conceives of weak inhibitory control—defined in neurobiological terms as deviations in the functioning of anterior brain circuits that operate to modulate affect and behavior on the basis of distal (nonimmediate) goals/consequences (Davidson et al., 2000; Patrick & Bernat, 2009a; Patterson & Newman, 1993)—as an underlying vulnerability or diathesis for impulse control (externalizing) problems of various kinds. The fact that this general vulnerability is predominantly heritable (Kendler et al., 2003; Krueger et al., 2002; Young et al., 2000) and accounts for substantial variance in diverse disorders makes it a crucial target for neurobiological research on problems of this kind. Recent research operationalizing a hierarchical model of impulse control problems and affiliated traits in the form of a quantitatively sophisticated psychometric instrument, the ESI (Krueger, Markon, et al., 2007), provides the foundation for systematic investigations of neurobiological processes related to this general vulnerability factor.

As described earlier, we have conducted recent investigations of processing deviations associated with high levels of general externalizing tendencies indexed by overall scores on an abbreviated (100-item) screening version of the ESI. Overall scores on the ESI-100 correlate very highly ($r > .95$) with scores on the full 415-item ESI.[3] In an initial published study of this type involving undergraduate participants ($N = 92$; Hall et al., 2007), we reported correlations between scores on the ESI-100 and criterion variables consisting of well-established self-report measures of antisocial deviance (Behavior Report on Rule-Breaking; Nye & Short, 1957), alcohol dependence (Alcohol Dependence Scale; Skinner & Allen, 1982), drug abuse (Short Drug Abuse Screening Test; Skinner, 1982), and adherence to societal norms (Socialization Scale; Gough, 1960). These correlations are shown in Table 22.2. Table 22.2 also presents, for a different sample consisting of 144 incarcerated male offenders, correlations between scores on the ESI-100 and symptoms of varying DSM-IV-TR impulse control disorders assessed via clinical interview. Uniformly robust correlations are evident between generalized externalizing tendencies as indexed by the ESI-100 on the one hand, and relevant self-report and interview-based criterion measures on the other.

TABLE 22.2. Correlations of Externalizing Scores with (1) Criterion Variables Assessed via Self-Report and (2) Symptoms of Differing DSM-IV-TR Impulse Control Disorders Assessed via Clinical Interview

Measure	*r*
Self-report criterion variables[a]	
Behavior Report on Rule-Breaking	
Overall behaviors	.83**
Adult behaviors	.75**
Adolescent behaviors	.76**
Alcohol Dependence Scale	.64**
Short Drug Abuse Screening Test	.61**
Socialization Scale	–.61**
DSM-IV-TR disorder symptoms[b]	
Antisocial personality	
Overall symptoms	.54**
Child symptoms	.42**
Adult symptoms	.60**
Alcohol dependence	.30**
Nicotine dependence	.60**
Other drug dependence	.34**

Note. Externalizing scores are overall scores on a 100-item version of the Externalizing Spectrum Inventory (ESI; Krueger, Markon, et al., 2007).

[a]Sample for self-report criterion variables consisted of 92 male and female university students recruited from undergraduate classes (cf. Hall et al., 2007).

[b]Sample for DSM-IV-TR symptom variables consisted of 144 adult male offenders recruited from a state correctional facility.

**$p < .001$.

Personality Disorders

Prominent researchers in the personality disorders area (e.g., Clark, Livesley, Schroeder, & Irish, 1996; Krueger, Skodol, Livesley, Shrout, & Huang, 2007; Morey, Gunderson, Quigley, & Lyons, 2000; Widiger & Sanderson, 1995) have presented evidence that symptoms of these disorders can be organized along broad thematic lines paralleling major dimensions of personality. Researchers in this area have also postulated that common neurobiological mechanisms, with ties to broad personality and temperament constructs, contribute to the emergence of personality disorders of differing types (e.g., Siever, 2000; Siever & Davis, 1991).

Furthermore, it is well established that personality syndromes coded on Axis II of DSM co-occur systematically with major clinical disorders coded on Axis I. In this regard, clinical and epidemiological studies (cf. Zuckerman, 1999) have revealed high comorbidity of Cluster C personality disorders with Axis I internalizing (fear and distress) disorders in particular, and of Cluster B personality disorders with Axis I impulse control disorders (e.g., adult alcohol and drug dependence; childhood ADHD, oppositional defiant disorder, and conduct disorder; and adult antisocial personality disorder) as well as some Axis I internalizing disorders (those within the distress subgroup especially). Related to this, various writers (e.g., Krueger, 1999b; Tellegen, 1985; Tellegen & Waller, 2008; Trull, 1992; Trull & Sher, 1994) have presented evidence that broad trait-dispositional constructs show empirical relations with personality syndromes coded on Axis II of DSM, as well as with major clinical disorders coded on Axis I. These and other findings have led to calls for an integration of Axis I and Axis II disorders in terms of broad dimensional constructs (e.g., Krueger, 2005; Livesley, Schroeder, Jackson, & Jang, 1994).

The neurobehavioral constructs of defensive reactivity and inhibitory control emphasized in this chapter have clear conceptual relevance to varying personality disorders coded on Axis II of DSM. The construct of defensive reactivity appears relevant in particular to personality disorders represented in Cluster C, which are characterized by fearfulness and anxiety (American Psychiatric Association, 2000). The construct of weak inhibitory control in turn has particular relevance to disorders in Cluster B, which are marked by behavioral impulsiveness and dysregulated emotion. Findings shown in Table 22.3, based on data from a mixed-gender sample of adults recruited from the community (N = 190), provide empirical confirmation of these linkages. Participants in this sample were assessed for trait fear and externalizing tendencies with the abbreviated screening measures of these constructs described earlier (TF-55, ESI-100); in addition, they were assessed for symptoms of DSM-IV Cluster B and C personality disorders with the Screening Questionnaire of the Structured Clinical Interview for DSM-IV Personality Disorders (SCID-II; First, Spitzer, Gibbon, & Williams, 1997). Consistent with expectation, trait fear scores show robust positive correlations with all personality disorders in Cluster C (with the magnitude particularly strong for avoidant personality disorder), whereas externalizing scores show robust positive associations with all personality disorders in Cluster B (in this case, all similar in magnitude). In addition, trait fear shows a robust *positive* association specifically with borderline personality disorder in Cluster B,[4] and significant *negative* correlations with both histrionic and antisocial personality disorders. As a function of these relations, and as a function of the statistical independence between trait fear scores and externalizing scores (e.g., in this sample, $r = -.069$, $p > .34$), trait fear scores contributed to improved prediction of each of these Cluster B disorders when entered concurrently with externalizing scores in a regression model. In contrast, externalizing scores showed no association with any of the personality disorders in Cluster C. These results provide evidence of systematic relations between psychometric measures of these neurobehavioral constructs and DSM personality disorder syndromes, and they encourage the idea that trait variations in defensive reactivity and inhibitory control represent key neurobiological substrates for personality disorders of differing types.

Criminal Psychopathy

In his classic volume *The Mask of Sanity* (1976), Cleckley characterized psychopathy

TABLE 22.3. Predictive Associations for Trait Fear Scores and Externalizing Scores with Symptoms of DSM-IV-TR Cluster B and C Personality Disorders Assessed via the SCID-II Screening Questionnaire

	Trait fear		Externalizing		
Symptom score	*r*	*β*	*r*	*β*	*R*
Cluster B personality disorders					
Histrionic	–.18*	–.16*	.28**	.27**	.32**
Narcissistic	.07	.09	.26**	.26**	.27**
Borderline	.30**	.32**	.25**	.27**	.40**
Antisocial (child symptoms)	–.24*	–.22*	.32**	.31**	.39**
Cluster C personality disorders					
Avoidant	.70**	.70**	.07	.12	.71**
Dependent	.31**	.32**	–.02	.00	.32**
Obsessive–compulsive	.30**	.31**	.09	.11	.32**

Note. Sample consisted of 190 adult men and women recruited from the community. Trait fear scores are total scores on a 55-item inventory composed of items from various established self-report measures of fear and fearlessness (for details, see Kramer et al., 2010). Externalizing scores are overall scores on a 100-item version of the ESI (Krueger, Markon, et al., 2007). *r*, zero-order correlation of personality disorder variable with trait fear or externalizing scores. *β*, beta coefficient for prediction of personality variable by trait fear or externalizing when scores on both were included together in a regression model. *R*, multiple-regression coefficient for prediction of personality variable by trait fear and externalizing when scores on both were included together in a regression model. For antisocial personality under Cluster B personality disorders, data were available for child symptoms only because questions pertaining to the adult symptoms are not included in the SCID-II Screening Questionnaire.
*$p < .05$. **$p < .001$.

as a dualistic syndrome. On one hand, psychopathic individuals present as personable, carefree, and emotionally resilient. On the other, they exhibit severe behavioral problems that bring them into repeated conflict with society. The dominant assessment instrument in contemporary psychopathy research, Hare's (1991, 2003) Psychopathy Checklist—Revised (PCL-R), was developed to identify individuals fitting Cleckley's clinical description within correctional or forensic settings. Although the PCL-R was developed to measure psychopathy as a unitary construct, structural analyses have shown that it contains distinctive subgroups of items (factors) that, while correlated, nonetheless show diverging relations with external criterion variables. Most published research has focused on the original two-factor model (Hare et al., 1990; Harpur, Hakstian, & Hare, 1988), in which PCL-R factor 1 comprises the interpersonal and affective features of psychopathy and factor 2 encompasses the antisocial deviance features. Higher factor 1 scores are associated with higher narcissism and Machiavellianism (Hare, 1991; Harpur, Hare, & Hakstian, 1989) and lower empathy (Hare, 2003). Factor 1—in particular, its variance that is separate from factor 2—shows positive relations with measures of social dominance (Harpur et al., 1989; Verona, Patrick, & Joiner, 2001), and in some studies with achievement (Verona et al., 2001) and trait positive affect (Patrick, 1994). Thus scores on PCL-R factor 1 evidence positive relations with some adaptive personality traits (interpersonal dominance and, in some work, tendencies toward achievement and trait positive affect; cf. Patrick, 2007). In contrast, PCL-R factor 2 shows associations mainly with indicators of deviancy, including aggression, impulsivity, and general sensation seeking; child and adult symptoms of DSM antisocial personality disorder; criminal history variables, such as onset and frequency of offending; and alcohol and drug dependence.

A two-process theory of psychopathy has been formulated to account for the distinctive components of psychopathy evident in the PCL-R (Fowles & Dindo, 2006; Patrick & Bernat, 2009b; Patrick & Lang, 1999).

This model focuses on the neurobehavioral constructs of defensive reactivity and inhibitory control emphasized here. The affective–interpersonal features of psychopathy associated with PCL-R factor 1 are theorized to reflect in part a lack of normal defensive reactivity, whereas the behavioral deviance features associated with factor 2 are theorized to reflect impairments in inhibitory control systems. Consistent with this, as noted earlier, individuals high in affective–interpersonal features of psychopathy show reduced potentiation of the startle reflex during aversive cuing (e.g., Patrick, 1994; Patrick, Bradley, & Lang, 1993) and reduced amygdala responsiveness to fearful face stimuli (Blair, 2006; Marsh et al., 2008). With regard to factor 2, scores on this component of the PCL-R show a close association with the broad externalizing factor of psychopathology (Patrick, Hicks, Krueger, & Lang, 2005) and selectively predict enhanced errors of commission in a well-established conflict task (Molto, Poy, Segarra, Pastor, & Montanes, 2007) as well as reductions in oddball P300 response (Venables, Reich, Bernat, Hall, & Patrick, 2008). From the perspective of this model, a clearer understanding of etiological mechanisms underlying psychopathy can be gained by directly assessing individuals on psychometric dimensions of trait fear and externalizing, and by using physiological measures to investigate deviations in cognitive and affective processing associated with varying positions along these dimensions (Patrick & Bernat, 2009b). Research of this kind can both draw on and inform parallel work focusing on the roles of trait fear and externalizing and their neurobiological counterparts (defensive reactivity, inhibitory control) in disorders of anxiety/mood, impulse control, and pathological personality as defined within DSM.

Toward a Psychoneurometrics of Psychopathology: An Illustration

Although evidence discussed to this point indicates that psychometric measures of dispositional defensive reactivity (i.e., trait fear) and inhibitory control (i.e., externalizing tendencies) can help to bridge psychopathological phenotypes with neurobiological measures, our aim in this chapter is not to suggest that these psychometric variables should *replace* traditional diagnostic entities as referents for neurobiological studies of psychopathology. Rather, our aim is to encourage—alongside continuing neuroscientific studies of established diagnostic syndromes—systematic investigation of the neurophysiological correlates of these psychometric phenotypes as a step toward the development of direct *brain-based* measures of neurobehavioral trait constructs. This can be accomplished by routinely including precise psychometric measures of these target constructs in brain measurement studies involving moderate to large *N*'s, in order to identify reliable neurophysiological correlates of these constructs. Once multiple physiological indicators of these constructs have been identified, studies incorporating multiple known indicators (in the context of common as well as varying task procedures) can be conducted in order to map convergences and divergences among indicators.

To provide a concrete illustration of this approach, we have undertaken analyses of relations among differing brain potential response indicators of externalizing across differing tasks. Because participant samples for the three-stimulus oddball, rERN, and fERN studies described earlier (see the section, "Neurobiological Bases and Physiological Correlates") overlapped, we could directly compare brain response measures across these tasks for the 92 participants who completed all three. As mentioned, fERN theta response to explicit feedback stimuli was unrelated to ESI-100 externalizing scores, but the delta P3 response to these same feedback stimuli was reduced as a function of higher externalizing tendencies ($r = -.25$). Notably, delta P3 response in the feedback task showed a significant positive association with P3 reactivity to novel picture stimuli in the oddball task ($r = .30$), which (as mentioned earlier) was also reduced as a function of externalizing ($r = -.29$). Furthermore, both of these P3 response measures showed positive correlations with magnitude of rERN response in the flanker task (i.e., greater P3 predicted greater rERN; *r*'s = .31 and .37 for feedback P3 and novelty P3, respectively).

Thus these three brain response indicators, each of which showed a significant negative association with ESI-100 externalizing scores, correlated significantly with one another. The implication is that these differing brain response measures tap *some process in common* that is related to externalizing tendencies. Notably, externalizing-related reductions in all three of these response measures were maximal at anterior (frontocentral) scalp sites. Taken together with the finding of *intact* fERN responding, these data encourage the idea that reduced rERN responding in high-externalizing individuals reflects impairment in anterior brain regions that participate with the ACC in the process of endogenous action monitoring. As noted earlier, we hypothesize that the PFC (more specifically, the dorsolateral PFC) is one such region.

As a further analysis, we entered these three brain response measures into a principal-components analysis along with ESI-100 externalizing scores. The analysis yielded evidence of a single dominant component, accounting for close to 50% of the overall variance in these four measures. To quantify their varying levels of effectiveness as indicators of a common factor, we performed a principal-axis factor analysis of scores on these four measures, solving for a single factor (per the results of the initial analysis). The loading of ESI-100 scores on this common factor ($r = -.49$), reflecting the shared variance among indicators, was comparable to the loadings for the three brain response measures (for feedback P3, novelty P3, and rERN, r's = .50, .54, and .66, respectively). Notably, the common factor emerging from this analysis represents a predominantly *neurophysiological* (ERP-based) externalizing factor on which the self-report ESI-100 measure also loaded. This result has important implications. It indicates that variations in inhibitory control can be assessed in terms of a composite *physiological* dimension. Given evidence for the high heritability of general externalizing tendencies (e.g., Krueger et al., 2002), together with data indicating that associations of externalizing with brain response measures such as P3 are mediated by common genetic influences (e.g., Hicks et al., 2007), this finding points to the possibility that scores on a *physiologically defined* dimension of inhibitory control could be used in future research as a basis for selecting at-risk individuals for neuroimaging and genetic studies of impulse control disorders.

The process of identifying reliable physiological indicators of neurobehavioral constructs such as defensive reactivity and inhibitory control, for which reliable psychometric referents already exist, is a process in which multiple investigators can participate. As described in the foregoing illustration, differing brain response indicators of externalizing tendencies as indexed by the ESI have already been identified, including rERN amplitude and varying manifestations of P3 response across differing tasks. Our efforts to operationalize a coherent psychometric dimension of trait fear are more recent, and startle reflex potentiation is the one variable to date that we have directly evaluated as an indicator of this dimension. However, as noted above (see the section, "Neurobiological Bases and Physiological Correlates"), the available literature points to various other candidate physiological indicators of trait fear. Furthermore, as discussed in the final section below, the psychoneurometric approach can potentially be applied to other dimensional psychometric phenotypes of relevance to psychopathology—including the broad distress and narrower positive affect and anhedonia factors associated with disorders in the internalizing spectrum (cf. Watson, 2005), and the callous aggression and addiction proneness subfactors that link particular subsets of problems/traits within the externalizing spectrum (Krueger, Markon, et al., 2007).

Figure 22.3 provides a schematic illustration of the psychoneurometric approach. As depicted in the figure, the approach entails (1) systematic efforts to identify *reliable physiological correlates* of a relevant behavioral phenotype within one or more psychologically meaningful task contexts, followed by (2) efforts to evaluate the *structure* of these physiological indicators (particularly the variance in each that intersects with the behavioral phenotype of interest; cf. Iacono, 1991)—both with the aims of refining physiological measurement of the neurobehavioral construct of interest, and clarifying the psychological meaning of physiological indicators derived from differing tasks. These

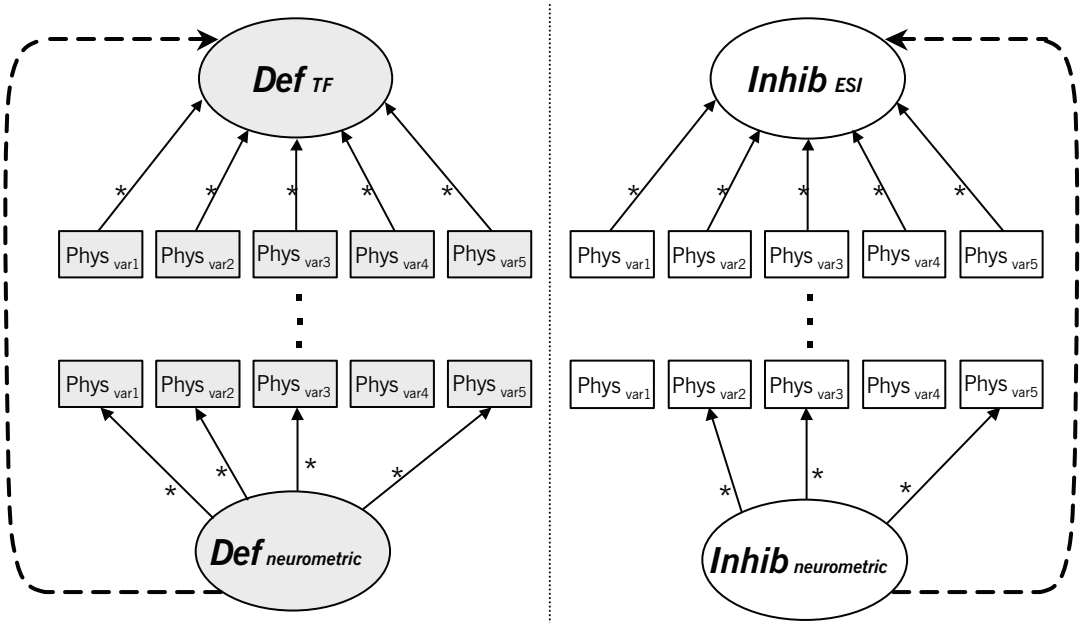


FIGURE 22.3. Schematic depiction of the psychoneurometric approach as applied to target constructs of defensive reactivity (*Def*) and inhibitory control (*Inhib*). The first stage of the approach involves identifying reliable physiological indicators ($Phys_{var1}$, $Phys_{var2}$, etc.) of these constructs operationalized *psychometrically*, as trait fear (Def_{TF}) and externalizing ($Inhib_{ESI}$). This is followed by mapping of interrelations among physiological indicators of each construct, in order to (1) establish statistically reliable *neurometric* measures of defensive reactivity ($Def_{neurometric}$) and inhibitory control ($Inhib_{neurometric}$), and (2) gain understanding of brain circuits/processes that underlie individual differences in defensive reactivity and inhibitory control. Information gained regarding the convergence of differing physiological indicators derived from designated behavioral tasks, and the brain mechanisms underlying this convergence, feeds back into psychometric conceptualization/measurement of these target constructs (large, curved, dashed arrows on left and right sides of figure). This process continues iteratively until a coherent set of neurometric tasks/measures exists for assessing each target construct precisely and reliably.

steps are followed by efforts to (3) update conceptualization of the target neurobehavioral construct to accommodate insights gained from the structural analysis of physiological indicators (while retaining linkages to psychopathology); (4) revise behavioral operationalization of the target construct to incorporate the revised conceptualization; and (5) implement new or modified task protocols designed to increase convergence between revised behavioral phenotypes and physiological response measures within those tasks. This process continues iteratively to the point where a coherent array of physiological tasks/measures exists for operationalizing the targeted neurobehavioral construct in a precise and reliable manner.

Conclusions and Future Directions

Research aimed at elucidating the neurobiological underpinnings of psychopathology has been identified as a high priority by authorities in the mental health field. However, there is growing recognition that new investigative approaches are needed to establish bridges between traditional conceptualizations of psychopathology and variations in brain circuitry and function that relate to individual differences in behavior (Hyman, 2007). As a method for elucidating neurobiological mechanisms in psychopathology, the psychoneurometric approach described here has a number of notable features. First, it confronts the issue of diagnostic comor-

bidity among mental disorders by focusing on broad dispositional factors that differing disorders share, while acknowledging the role of unique etiological contributors to specific disorders. Second, it addresses the gap between diagnostic phenotypes (clinical disorders) and neurobiological systems by focusing on neurobehavioral trait constructs with demonstrable relevance to psychopathology. Third, it provides a means by which high-level quantitative/statistical methods developed to quantify constructs in the domains of personality and performance can be applied to the development of reliable neurophysiological measures of trait constructs relevant to psychopathology. Fourth, it provides an interface through which behavioral conceptualizations can directly guide efforts to identify psychopathology-relevant neurobiological processes/circuits—and, reciprocally, through which knowledge gained about relevant neurobiological processes/circuits can feed back into behavioral conceptualizations of psychopathology.

There are some important practical challenges to implementing an approach of this kind at levels required to ensure significant sustained progress. Psychometric development efforts require large participant samples and repeated rounds of data collection in order to establish the measurement properties of items/subtests. Relative to self-report and performance-based assessments, neurophysiological assessment procedures are generally more costly, time-consuming, and resource-intensive. Neuroimaging methods in particular pose challenges in terms of availability and expense. We believe that these challenges can be surmounted through coordinated efforts of multiple investigators employing less costly electrocortical (EEG/ERP) and peripheral physiological measures in larger-scale mapping and refinement efforts. In turn, work of this kind can inform and draw upon smaller-scale investigative efforts using costlier methods such as hemodynamic neuroimaging to extend understanding of brain circuits of emerging interest. The first step in pursuing a psychoneurometric approach to the study of psychopathology consists of studies with moderate to large samples aimed at identifying reliable neurobiological correlates of constructs such as trait fear and externalizing. As noted, this is an effort in which multiple investigators can participate either independently or in collaboration with one another—and, in the case of defensive reactivity and inhibitory control constructs, a variety of candidate indicators can be identified on the basis of existing published literature.

Regarding physiological measurement, we encourage the use of EEG/ERP as a methodology in moderate- to large-*N* studies exploring candidate indicators and evaluating their convergence. Among other advantages, EEG/ERP measures (1) directly reflect neural activity and thus can be interpreted in relation to models of brain structure and function; (2) are informative about cognitive/attentional as well as affective/motivational processes (e.g., Lang et al., 1997); and (3) yield precise information regarding temporal (time) and spectral (frequency) characteristics of brain activity, along with spatial (scalp site) information that can be used to estimate underlying neural sources of activity (cf. Patrick & Bernat, 2009a). With regard to localization of neural activity origins, the precision with which underlying neural sources can be estimated from surface EEG activity can be enhanced by recording from multiple scalp sites and referencing the activity to brain images acquired via MRI (e.g., Ding et al., 2007).

A further point is that the current chapter is necessarily limited in scope. Given constraints of space, we have focused largely on disorders in the mood/anxiety and impulse control domains (along with affiliated personality syndromes) because these represent some of the most commonly occurring disorders in the population. Furthermore, we have focused primarily on constructs of defensive reactivity and inhibitory control because these represent examples of trait constructs with clear neurobiological referents, and because available data point to a role for these constructs in multiple internalizing and externalizing disorders. However, the basic investigative strategy we have outlined is applicable to disorders of other sorts—including developmental disorders, appetitive (e.g., eating, sexual) disorders, and psychotic syndromes. Regarding target constructs for study, it seems likely that deviations in defensive reactivity and inhibitory control contribute to the symptomatic expression of at least some of these other disorders (see, e.g., Meehl, 1990). In addi-

tion, other neurobehavioral constructs in the domains of motivation, attention/cognition, and perception will need to be considered in relation to disorders of these other types.

As a final note, it bears emphasis that the methodological approach described here is intended as a supplement to, rather than as a substitute for—existing research strategies. In particular, we view the psychoneurometric approach as a paradigm for linking psychopathological conditions to neurobiological systems, not as a prescription for a particular program of research. Besides contributing to our understanding of brain substrates of psychopathology, we believe that this approach offers a path toward the development of reliable neurophysiological composite measures of trait constructs relevant to psychopathology. Neurophysiological trait measures of this type are likely to prove especially effective as selection criteria for neuroimaging and genetic studies of individuals at biological risk for psychopathology.

Note

1. When fearfulness is operationalized in terms of preference for safe but unstimulating activities over risky activities (e.g., as in the SSS-TAS subscale or the MPQ Harm Avoidance scale), fear scores tend to be uncorrelated with scores on trait anxiety measures (Tellegen & Waller, 2008). In contrast, when fearfulness is defined in terms of degree of negative emotion experienced in relation to unfamiliar or threatening objects or situations (as in the EAS-Fear, the FSS, and the TPQ-HA scale), fear scores tend to be moderately correlated with levels of trait anxiousness (Buss & Plomin, 1984).
2. A list of the TF-55 items can be obtained from us upon request.
3. Copies of both the full and abbreviated versions of the ESI can be obtained from us upon request.
4. Consistent with findings for borderline personality in Table 22.3, James and Taylor (2008) reported positive associations of borderline personality symptoms (assessed via self-report) with both internalizing and externalizing factors of psychopathology (assessed via computer-assisted interview). Notably, in this analysis, borderline personality operated more as an indicator of the anxious misery (distress) subfactor of internalizing than the fear disorder subfactor. However, the anxious misery and fear subfactors were highly correlated in this study (cf. Krueger, 1999b), and borderline personality symptoms evidenced significant bivariate relations with fear disorder symptoms. The implication is that borderline personality is related primarily to distress disorders and secondarily to fear disorders.

Acknowledgments

Preparation of this chapter was supported by Grant Nos. MH65137, MH072850, MH080239, and MH089727 from the National Institute of Mental Health. We are grateful to Greg Miller for his comments on an earlier version of this chapter.

References

Achenbach, T. M., & Edelbrock, C. S. (1978). The classification of child psychopathology: A review and analysis of empirical efforts. *Psychological Bulletin*, *85*, 1275–1301.

Adolphs, R., Tranel, D., Hamann, S., Young, A. W., Calder, A. J., Phelps, E. A., et al. (1999). Recognition of facial emotion in nine individuals with bilateral amygdala damage. *Neuropsychologia*, *37*, 1111–1117.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.

Anokhin, A. P., Vedeniapin, A. B., Sirevaag, E. J., Bauer, L. O., O'Connor, S. J., Kuperman, S., et al. (2000). The P300 brain potential is reduced in smokers. *Psychopharmacology*, *149*, 409–413.

Arrindell, W. A., Emmelkamp, P. M. G., & van der Ende, J. (1984). Phobic dimensions: I. Reliability and generalizability across samples, gender, and nations. *Advances in Behavior Research and Therapy*, *6*, 207–254.

Attou, A., Figiel, C., & Timsit-Berthier, M. (2001). ERP assessment of heroin detoxification and methadone treatment in chronic heroin users. *Clinical Neurophysiology*, *31*, 171–180.

Barch, D. M., Carter, C. S., Braver, T. S., Sabb, F. W., MacDonald, A. W., Noll, D. C., et al. (2001). Selective deficits in prefrontal cortex function in medication naïve patients with schizophrenia. *Archives of General Psychiatry*, *58*, 280–288.

Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive functions: Constructing a unified theory of ADHD. *Psychological Bulletin*, *121*, 65–94.

Bauer, L. O., & Hesselbrock, V. M. (1999a). P300 decrements in teenagers with conduct problems: Implications for substance abuse risk and brain development. *Biological Psychiatry*, *46*, 263–272.

Bauer, L. O., & Hesselbrock, V. M. (1999b). Subtypes of family history and conduct disorder: Effects on P300 during the Stroop test. *Neuropsychopharmacology*, *21*, 51–62.

Bauer, L. O., O'Connor, S., & Hesselbrock, V. M. (1994). Frontal P300 decrements in antisocial personality disorder. *Alcoholism: Clinical and Experimental Research*, *18*, 1300–1305.

Bechara, A., Damasio, H., Tranel, D., & Damasio, A. R. (1997). Deciding advantageously before knowing the advantageous strategy. *Science*, *275*, 1293–1295.

Begleiter, H., Porjesz, B., Bihari, B., & Kissin, B. (1984). Event-related brain potentials in boys at risk for alcoholism. *Science*, *225*, 1493–1496.

Benning, S. D., Patrick, C. J., Blonigen, D. M., Hicks, B. M., & Iacono, W. G. (2005). Estimating facets of psychopathy from normal personality traits: A step toward community-epidemiological investigations. *Assessment*, *12*, 3–18.

Benning, S. D., Patrick, C. J., Hicks, B. M., Blonigen, D. M., & Krueger, R. F. (2003). Factor structure of the Psychopathic Personality Inventory: Validity and implications for clinical assessment. *Psychological Assessment*, *15*, 340–350.

Benning, S. D., Patrick, C. J., & Iacono, W. G. (2005). Psychopathy, startle blink modulation, and electrodermal reactivity in twin men. *Psychophysiology*, *42*, 753–762.

Bernat, E., Patrick, C. J., Benning, S. D., & Tellegen, A. (2006). Thematic content and affective intensity: Modulation of startle reflex, electromyogram, heart rate, and skin conductance. *Psychophysiology*, *43*, 93–103.

Bernat, E. M., Nelson, L. D., Steele, V. R., Patrick, C. J., & Gehring, W. J. (2010). *Externalizing psychopathology and gain/loss feedback in a simulated gambling task: Dissociable components of brain response revealed by time-frequency analysis.* Manuscript submitted for publication.

Bernat, E. M., Patrick, C. J., Cadwallader, M., van Mersbergen, M., & Seo, D. (2003). Affective orienting responses: A novelty P3 study with emotional pictures. *Psychophysiology*, *40*, S25.

Bernat, E. M., Williams, W. J., & Gehring, W. J. (2005). Decomposing ERP time-frequency energy using PCA. *Clinical Neurophysiology*, *116*, 1314–1334.

Biggins, C. A., MacKay, S., Clark, W., & Fein, G. (1997). Event-related potential evidence for frontal cortex effects of chronic cocaine dependence. *Biological Psychiatry*, *42*, 472–485.

Bishop, S. J., Duncan, J., & Lawrence, A. D. (2004). State anxiety modulation of the amygdala response to unattended threat-related stimuli. *Journal of Neuroscience*, *24*, 10364–10368.

Blair, R. J. R. (2006). Subcortical brain systems in psychopathy: The amygdala and associated structures. In C. J. Patrick (Ed.), *Handbook of psychopathy* (pp. 296–312). New York: Guilford Press.

Blair, R. J. R., Colledge, E., Murray, L., & Mitchell, D. G. (2001). A selective impairment in the processing of sad and fearful expressions in children with psychopathic tendencies. *Journal of Abnormal Child Psychology*, *29*, 491–498.

Block, J., & Block, J. H. (1980). The role of ego-resiliency and ego-control in the organization of behavior. In W. A. Collins (Ed.), *Minnesota Symposium on Child Psychology* (Vol. 13, pp. 39–101). Hillsdale, NJ: Erlbaum.

Blonigen, D., Hicks, B., Patrick, C., Krueger, R., Iacono, W., & McGue, M. (2005). Psychopathic personality traits: Heritability and genetic overlap with internalizing and externalizing pathology. *Psychological Medicine*, *35*, 637–648.

Blumer, D., & Benson, D. F. (1975). Personality changes with frontal and temporal lobe lesions. In D. F. Benson & D. Blumer (Eds.), *Psychiatric aspects of neurological disease* (pp. 151–169). New York: Grune & Stratton.

Bradley, M. M., Codispoti, M., Cuthbert, B. N., & Lang, P. J. (2001). Emotion and motivation: I. Defensive and appetitive reactions in picture processing. *Emotion*, *1*, 276–298.

Brigham, J., Herning, R. I., & Moss, H. B. (1995). Event-related potentials and alpha synchronization in preadolescent boys at risk for psychoactive substance use. *Biological Psychiatry*, *37*, 834–846.

Brown, J. W., & Braver, T. S. (2005). Learned predictions of error likelihood in the anterior cingulate cortex. *Science*, *307*, 1118–1121.

Bush, G., Luu, P., & Posner, M. I. (2000). Cognitive and emotional influences in anterior cingulate cortex. *Trends in Cognitive Sciences*, *4*, 215–222.

Buss, A. H., & Plomin, R. (1975). *A temperament theory of personality development*. New York: Wiley.

Buss, A. H., & Plomin, R. (1984). *Temperament: Early developing personality traits*. Hillsdale, NJ: Erlbaum.

Campbell, D. T., & Fiske, D. W. (1959). Convergent and discriminant validation by the multitrait–multimethod matrix. *Psychological Bulletin*, *56*, 81–105.

Campbell, D. T., & Stanley, J. C. (1966). *Experimental and quasi-experimental designs for research*. Chicago: Rand McNally.

Carter, C. S., Braver, T. S., Barch, D. M., Botvinick, M. M., Noll, D. N., & Cohen, J. D. (1998). Anterior cingulate cortex, error detection, and the online monitoring of performance. *Science*, *280*, 747–749.

Clark, L. A., Livesley, W. J., Schroeder, M. L., & Irish, S. (1996). The structure of maladaptive personality traits: Convergent validity between two systems. *Psychological Assessment*, *8*, 294–303.

Clark, L. A., & Watson, D. (1999). Temperament: A new paradigm for trait psychology. In L. A. Pervin & O. John (Eds.), *Handbook of personality: Theory and research* (2nd ed., pp. 399–423). New York: Guilford Press.

Clark, L. A., Watson, D., & Reynolds, S. (1995). Diagnosis and classification of psychopathology: Challenges to the current system and future directions. *Annual Review of Psychology*, *46*, 121–153.

Cleckley, H. (1976). *The mask of sanity* (5th ed.). St. Louis, MO: Mosby.

Cloninger, C. R. (1987). A systematic method for clinical description and classification of personality variants. *Archives of General Psychiatry*, *44*, 573–588.

Cohen, J. D., & Servan-Schreiber, D. (1992). Context, cortex, and dopamine: A connectionist approach to behavior and biology in schizophrenia. *Psychological Review*, *99*, 45–77.

Cook, E. W., III. (1999). Affective individual differences, psychopathology, and startle reflex modulation. In M. E. Dawson, A. M. Schell, & A. H. Boehmelt (Eds.), *Startle modification: Implications for clinical science, cognitive science, and neuroscience* (pp. 187–208). New York: Cambridge University Press.

Cook, E. W., III, Melamed, B. G., Cuthbert, B. N., McNeil, D. W., & Lang, P. J. (1988). Emotional imagery and the differential diagnosis of anxiety. *Journal of Consulting and Clinical Psychology*, *56*, 734–740.

Corr, P. J., Kumari, V., Wilson, G. D., Checkley, S., & Gray, J. A. (1997). Harm avoidance and affective modulation of the startle reflex: A replication. *Personality and Individual Differences*, *22*, 591–593.

Corr, P. J., Wilson, G. D., Fotiadou, M., Kumari, V., Gray, N. S., Checkley, S., et al. (1995). Personality and affective modulation of the startle reflex. *Personality and Individual Differences*, *19*, 543–553.

Courchesne, E., Hillyard, S. A., & Galambos, R. (1975). Stimulus novelty, task relevance, and the visual evoked potential in man. *Electroencephalography and Clinical Neurophysiology*, *39*, 131–143.

Curtin, J. J., Patrick, C. J., Lang, A. R., Cacioppo, J. T., & Birbaumer, N. (2001). Alcohol affects emotion through cognition. *Psychological Science*, *12*, 527–531.

Cuthbert, B. N., Lang, P. J., Strauss, C., Drobes, D., Patrick, C. J., & Bradley, M. M. (2003). The psychophysiology of anxiety disorder: Fear memory imagery. *Psychophysiology*, *40*, 407–422.

Damasio, A. R., Tranel, D., & Damasio, H. (1990). Individuals with sociopathic behaviour caused by frontal damage fail to respond autonomically to social stimuli. *Behavioural Brain Research*, *41*, 81–94.

Davidson, R. J., Pizzigalli, D., Nitschke, J. B., & Putnam, K. M. (2002). Depression: Perspectives from affective neuroscience. *Annual Review of Psychology*, *53*, 545–574.

Davidson, R. J., Putnam, K. M., & Larson, C. L. (2000). Dysfunction in the neural circuitry of emotion regulation: A possible prelude to violence. *Science*, *289*, 591–594.

Davis, M. (1979). Diazepam and flurazepam: Effects on conditioned fear as measured with the potentiated startle paradigm. *Psychopharmacology*, *62*, 1–7.

Davis, M. (1989). Neural systems involved in fear-potentiated startle. *Annals of the New York Academy of Sciences*, *563*, 165–183.

Davis, M. (1992). The role of the amygdala in conditioned fear. In J. Aggleton (Ed.), *The amygdala: Neurobiological aspects of emotion, memory and mental dysfunction* (pp. 255–305). New York: Wiley.

Davis, M., Falls, W. A., Campeau, S., & Kim, M. (1993). Fear-potentiated startle: A neural and

pharmacological analysis. *Behavioural Brain Research*, *58*, 175–198.

Davis, M., Walker, D. L., & Lee, Y. (1997). Roles of the amygdala and bed nucleus of the stria terminalis in fear and anxiety measured with the acoustic startle reflex. *Annals of the New York Academy of Sciences*, *831*, 305–331.

Dehaene, S., Posner, M. I., & Tucker, D. M. (1994). Localization of a neural system for error detection and compensation. *Psychological Science*, *5*(5), 303–305.

Depue, R. A., & Iacono, W. G. (1989). Neurobehavioral aspects of affective disorders. *Annual Review of Psychology*, *40*, 457–492.

Depue, R. A., Luciana, M., Arbisi, P., Collins, P., & Leon, A. (1994). Dopamine and the structure of personality: Relation of agonist-induced dopamine activity to positive emotionality. *Journal of Personality and Social Psychology*, *67*, 485–498.

Dias, R., Robbins, T. W., & Roberts, A. C. (1996). Dissociation in prefrontal cortex of affective and attentional shifts. *Nature*, *380*, 69–72.

Dien, J., Spencer, K. M., & Donchin, E. (2003). Localization of the event-related potential novelty response as defined by principal components analysis. *Cognitive Brain Research*, *17(3)*, 637–650.

Dikman, Z. V., & Allen, J. J. (2000). Error monitoring during reward and avoidance learning in high- and low-socialized individuals. *Psychophysiology*, *37*(1), 43–54.

Ding, L., Wilke, C., Xu, B., Xu, X., van Drongelen, W., Kohrman, M., et al. (2007). EEG source imaging: Correlating source locations and extents with electrocorticography and surgical resections in epilepsy patients. *Journal of Clinical Neurophysiology*, *24*, 130–136.

Dvorak-Bertsch, J. D., Curtin, J. J., Rubinstein, T. J., & Newman, J. P. (2007). Anxiety moderates the interplay between cognitive and affective processing. *Psychological Science*, *18*, 699–705.

Ericsson, K., & Kinsch, W. (1995). Long-term working memory. *Psychological Review*, 102(2), 211–245.

Eysenck, H. J. (1967). *The biological basis of personality*. Springfield, IL: Thomas.

Falconer, D. S. (1989). *Introduction to quantitative genetics* (3rd ed.). New York: Wiley.

Fanselow, M. S. (1994). Neural organization of the defensive behavior system responsible for fear. *Psychonomic Bulletin and Review*, *1*, 429–438.

First, M., Spitzer, R., Gibbon, M., & Williams, J. (1997). *Structured Clinical Interview for DSM-IV Personality Disorders (SCID-II)*. Washington, DC: American Psychiatric Press.

Flor, H., Birbaumer, N., Hermann, C., Ziegler, S., & Patrick, C. J. (2002). Aversive Pavlovian conditioning in psychopaths: Peripheral and central correlates. *Psychophysiology*, *39*, 505–518.

Fowles, D. C., & Dindo, L. A (2006). Dual-deficit model of psychopathy. In C. J. Patrick (Ed.), *Handbook of psychopathy* (pp. 14–34). New York: Guilford Press.

Gehring, W. J., Coles, M. G. H., Meyer, D. E., & Donchin, E. (1995). A brain potential manifestation of error-related processing. In G. Karmos, M. Molnar, V. Csepe, I. Czigler, & J. E. Desmedt (Eds.), *Perspectives of event-related potentials research* (pp. 267–272). Amsterdam: Elsevier.

Goldsmith, H. H., & Campos, J. J. (1982). Toward a theory of infant temperament. In R. N. Emde & R. J. Harmon (Eds.), *The development of attachment and affiliative systems* (pp. 161–193). New York: Plenum Press.

Goldsmith, H. H., Lemery, K. S., Buss, K. A., & Campos, J. J. (1999). Genetic analyses of focal aspects of infant temperament. *Developmental Psychology*, *35*, 972–985.

Gorenstein, E. E., & Newman, J. P. (1980). Disinhibitory psychopathology: A new perspective and a model for research. *Psychological Review*, *87*, 301–315.

Gough, H. G. (1960). Theory and measurement of socialization. *Journal of Consulting Psychology*, *24*, 23–30.

Gotlib, I. H., Sivers, H., Gabrieli, J. D. E., Whitfield-Gabrieli, S., Goldin, P., Minor, K. L., et al. (2005). Subgenual anterior cingulate activation to valenced emotional stimuli in major depression. *NeuroReport*, *16*, 1731–1734.

Gray, J. A. (1991). Neural systems, emotion and personality. In J. Madden (Ed.), *Neurobiology of learning, emotion and affect* (pp. 273–306). New York: Raven Press.

Grillon, C., & Baas, J. (2003). A review of the modulation of the startle reflex by affective states and its application in psychiatry. *Clinical Neurophysiology*, *114*, 1557–1579.

Hall, J. R., Bernat, E. M., & Patrick, C. J. (2007). Externalizing psychopathology and the error-related negativity. *Psychological Science*, *18*, 326–333.

Hamm, A. O., Cuthbert, B. N., Globisch, J., &

Vaitl, D. (1997). Fear and the startle reflex: Blink modulation and autonomic response patterns in animal and mutilation fearful subjects. *Psychophysiology*, *34*, 97–107.

Harari, A. R., Mattay, V. S., Tessitore, A., Kolachana, B., Fera, F., Goldman, D., et al. (2002). Serotonin transporter genetic variation and the response of the human amygdala. *Science*, *297*, 400–403.

Hare, R. D. (1978). Electrodermal and cardiovascular correlates of psychopathy. In R. D. Hare & D. Schalling (Eds.), *Psychopathic behavior: Approaches to research* (pp. 107–143). Chichester, UK: Wiley.

Hare, R. D. (1991). *The Hare Psychopathy Checklist—Revised*. Toronto: Multi-Health Systems.

Hare, R. D. (2003). *The Hare Psychopathy Checklist—Revised* (2nd ed.). Toronto: Multi-Health Systems.

Hare, R. D., Harpur, T. J., Hakstian, A. R., Forth, A. E., Hart, S. D., & Newman, J. P. (1990). The Revised Psychopathy Checklist: Reliability and factor structure. *Psychological Assessment*, *2*, 338–341.

Harpur, T. J., Hakstian, A. R., & Hare, R. D. (1988). Factor structure of the Psychopathy Checklist. *Journal of Consulting and Clinical Psychology*, *56*, 741–747.

Harpur, T. J., Hare, R. D., & Hakstian, A. R. (1989). Two-factor conceptualization of psychopathy: Construct validity and assessment implications. *Psychological Assessment*, *1*, 6–17.

Heller, W., & Nitschke, J. (1998). The puzzle of regional brain activity in anxiety and depression: The importance of subtypes and comorbidity. *Cognition and Emotion*, *12*, 421–447.

Heller, W., Nitschke, J., Etienne, M. A., & Miller, G. A. (1997). Patterns of regional brain activity differentiate types of anxiety. *Journal of Abnormal Psychology*, *106*, 376–385.

Hettema, J. M., Anna, P., Neale, M., Kendler, K. A., & Fredrikson, M. (2003). A twin study of the genetics of fear conditioning. *Archives of General Psychiatry*, *60*, 702–708.

Hicks, B. M., Bernat, E. M., Malone, S. M., Iacono, W. G., Patrick, C. J., Krueger R. F., et al. (2007). Genes mediate the association between P3 amplitude and externalizing disorders. *Psychophysiology*, *44*, 98–105.

Hicks, B. M., & Patrick, C. J. (2006). Psychopathy and negative emotionality: Analyses of suppressor effects reveal distinct relations with emotional distress, fearfulness, and anger–hostility. *Journal of Abnormal Psychology*, *115*, 276–287.

Holroyd, C. B., & Coles, M. G. H. (2002). The neural basis of human error processing: Reinforcement learning, dopamine, and the error-related negativity. *Psychological Review*, *109*, 679–709.

Holroyd, C. B., Dien, J., & Coles, M. G. (1998). Error-related scalp potentials elicited by hand and foot movements: Evidence for an output-independent error-processing system in humans. *Neuroscience Letters*, *242*(2), 65–68.

Holroyd, C. B., Larsen, J. T., & Cohen, J. D. (2004). Context dependence of the event-related brain potential associated with reward and punishment. *Psychophysiology*, *41*(2), 245–253.

Hyman, S. M. (2007). Can neuroscience be integrated into the DSM-V? *Nature Reviews Neuroscience*, *8*, 725–732.

Iacono, W. G. (1991). Psychophysiological assessment of psychopathology. *Psychological Assessment*, *3*, 309–320.

Iacono, W. G., Carlson, S. R., Malone, S. M., & McGue, M. (2002). P3 event-related potential amplitude and risk for disinhibitory disorders in adolescent boys. *Archives of General Psychiatry*, *59*, 750–757.

Insel, T. R., & Scolnick, E. M. (2006). Cure therapeutics and strategic prevention: Raising the bar for mental health research. *Molecular Psychiatry*, *11*, 11–17.

Jackson, D. C., Mueller, C. J., Dolski, I., Dalton, K. M., Nitschke, J. B., Urry, H. L., et al. (2003). Now you feel it, now you don't: Frontal EEG asymmetry and individual differences in emotion regulation. *Psychological Science*, *14*, 612–617.

James, L. M., & Taylor, J. (2008). Revisiting the structure of mental disorders: Borderline personality disorder and the internalizing/externalizing spectra. *British Journal of Clinical Psychology*, *47*, 361–380.

James, W. (1983). *The principles of psychology*. Cambridge, MA: Harvard University Press. (Original work published 1890)

Jiang, Y., & He, S. (2006) Cortical responses to invisible faces: Dissociating subsystems for facial-information processing. *Current Biology*, *16*, 2023–2029.

Jiang, Y., Shannon, R. W., Vizueta, N., Bernat, E. M., Patrick, C. J., & He, S. (2009). Dynamics of processing invisible faces in the brain: Automatic neural encoding of facial expression information. *NeuroImage*, *44*, 1171–1177.

Junghoefer, M., Bradley, M. M., Elbert, T. R., & Lang, P. J. (2001). Fleeting images: A new look at early emotion discrimination. *Psychophysiology*, *38*, 175–178.

Kagan, J. (1994). *Galen's prophecy: Temperament in human nature*. New York: Basic Books.

Kagan, J., & Snidman, N. (1999). Early childhood predictors of adult anxiety disorders. *Biological Psychiatry*, *46*, 1536–1541.

Kendler, K. S., Prescott, C. A., Myers, J., & Neale, M. C. (2003). The structure of genetic and environmental risk factors for common psychiatric and substance use disorders in men and women. *Archives of General Psychiatry*, *60*, 929–937.

Kochanska, G. K. (1997). Multiple pathways to conscience for children with different temperaments: From toddlerhood to age 5. *Developmental Psychology*, *33*, 228–240.

Kramer, M. D., Patrick, C. J., Krueger, R. F., & Bayevsky, M. (2010). *Phenotypic and etiologic parallels in the structure of fear and fearlessness*. Manuscript submitted for publication.

Krueger, R. F. (1999a). Personality traits in late adolescence predict mental disorders in early adulthood: A prospective–epidemiological study. *Journal of Personality*, *67*, 39–65.

Krueger, R. F. (1999b). The structure of common mental disorders. *Archives of General Psychiatry*, *56*, 921–926.

Krueger, R. F. (2005). Continuity of Axes I and II: Toward a unified model of personality, personality disorders, and clinical disorders. *Journal of Personality Disorders*, *19*, 233–261.

Krueger, R. F., Hicks, B. M., Patrick, C. J., Carlson, S. R., Iacono, W. G., & McGue, M. (2002). Etiologic connections among substance dependence, antisocial behavior, and personality: Modeling the externalizing spectrum. *Journal of Abnormal Psychology*, *111*, 411–424.

Krueger, R. F., Markon, K. E., Patrick, C. J., Benning, S. D., & Kramer, M. D. (2007). Linking antisocial behavior, substance use, and personality: An integrative quantitative model of the adult externalizing spectrum. *Journal of Abnormal Psychology*, *116*, 645–666.

Krueger, R. F., Skodol, A. E., Livesley, W. J., Shrout, P., & Huang, Y. (2007). Synthesizing dimensional and categorical approaches to personality disorders: Refining the research agenda for DSM-V Axis II. *International Journal of Methods in Psychiatric Research*, *16*, S65–S73.

Lang, P. J. (1995). The emotion probe: Studies of motivation and attention. *American Psychologist*, *50*, 372–385.

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (1990). Emotion, attention, and the startle reflex. *Psychological Review*, *97*, 377–395.

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (1997). Motivated attention: Affect, activation, and action. In P. J. Lang, R. F. Simons, & M. T. Balaban (Eds.), *Attention and orienting: Sensory and motivational processes* (pp. 97–135). Hillsdale, NJ: Erlbaum.

LeDoux, J. E. (1995). Emotion: Clues from the brain. *Annual Review of Psychology*, *46*, 209–235.

Levenston, G. K., Patrick, C. J., Bradley, M. M., & Lang, P. J. (2000). The psychopath as observer: Emotion and attention in picture processing. *Journal of Abnormal Psychology*, *109*, 373–385.

Lilienfeld, S. O., & Andrews, B. P. (1996). Development and preliminary validation of a self report measure of psychopathic personality traits in noncriminal populations. *Journal of Personality Assessment*, *66*, 488–524.

Lissek, S., & Powers, A. S. (2003). Sensation seeking and startle modulation by physically threatening images. *Biological Psychology*, *63*, 179–197.

Livesley, W. J., Schroeder, M. L., Jackson, D. N., & Jang, K. L. (1994). Categorical distinctions in the study of personality disorder: implications for classification. *Journal of Abnormal Psychology*, *103*, 6–17.

Luu, P., Flaisch, T., & Tucker, D. M. (2000). Medial frontal cortex in action monitoring. *Journal of Neuroscience*, *20*, 464–469.

Lykken, D. T. (1957). A study of anxiety in the sociopathic personality. *Journal of Abnormal and Clinical Psychology*, *55*, 6–10.

Lykken, D. T. (1995). *The antisocial personalities*. Mahwah, NJ: Erlbaum.

Maher, B. (1968). *Principles of psychopathology: An experimental approach*. New York: McGraw-Hill.

Mars, R. B., Coles, M. G., Grol, M. J., Holroyd, C. B., Nieuwenhuis, S., Hulstijn, W., et al. (2005). Neural dynamics of error processing in medial frontal cortex. *NeuroImage*, *28*(4), 1007–1013.

Marsh, A. A., Finger, E. C., Mitchell, G. V., Reid, M. E., Sims, C., Kosson, D. S., et al. (2008). Reduced amygdala response to fearful expressions in children and adolescents with callous–unemotional traits and disruptive behavior

disorders. *American Journal of Psychiatry*, *165*, 712–720.

McGhie, A., & Chapman, J. (1961). Disorders of attention and perception in early schizophrenia. *British Journal of Medical Psychology*, *34*, 103–116.

Meehl, P. E. (1990). Toward an integrated theory of schizotaxia, schizotypy, and schizophrenia. *Journal of Personality Disorders*, *4*, 1–99.

Melzig, C. A., Weike, A. I., Zimmermann, J., & Hamm, A. O. (2007). Startle reflex modulation and autonomic responses during anxious apprehension in panic disorder patients. *Psychophysiology*, *45*, 846–854.

Miller, E. K. (1999). The prefrontal cortex: Complex neural properties for complex behavior. *Neuron*, *22*, 15–17.

Miller, E. K., & Cohen, J. D. (2001). Integrative theory of prefrontal cortex function. *Annual Review of Neuroscience*, *24*(24), 167–202.

Miltner, W. H. R., Braun, C. H., & Coles, M. G. H. (1997). Event-related brain potentials following incorrect feedback in a time-estimation task: Evidence for a "generic" neural system for error detection. *Journal of Cognitive Neuroscience*, *9*, 788–798.

Mineka, S., Watson, D., & Clark, L. E. A. (1998). Comorbidity of anxiety and unipolar mood disorders. *Annual Review of Psychology*, *49*, 377–412.

Molto, J., Poy, R., Segarra, P., Pastor, M. C., & Montanes, S. (2007). Response perseveration in psychopaths: Interpersonal/affective or social deviance traits? *Journal of Abnormal Psychology*, *116*, 632–637.

Morey, L. C., Gunderson, J., Quigley, B. D., & Lyons M. (2000). Dimensions and categories: The "Big Five" factors and the DSM personality disorders. *Assessment*, *7*, 203–216.

Morgan, A. B., & Lilienfeld, S. O. (2000). A meta-analytic review of the relation between antisocial behavior and neuropsychological measures of executive function. *Clinical Psychology Review*, *20*, 113–136.

Morris, J. S., Frith, C. D., Perrett, D. I., Rowland, D., Young, A. W., Calder, A. J., et al. (1996). A differential neural response in the human amygdala to fearful and happy facial expressions. *Nature*, *383*, 812–815.

Nieuwenhuis, S., Aston-Jones, G., & Cohen, J. D. (2005). Decision making, the P3, and the locus coeruleus–norepinephrine system. *Psychological Bulletin*, *131*(4), 510–532.

Nye, F. I., & Short, J. F., Jr. (1957). Scaling delinquent behavior. *American Sociological Review*, *22*, 326–331.

Öhman, A., & Soares, J. J. F. (1994). "Unconscious anxiety": Phobic responses to masked stimuli. *Journal of Abnormal Psychology*, *103*, 231–240.

Pailing, P. E., & Segalowitz, S. J. (2004). The error-related negativity as a state and trait measure: Motivation, personality, and ERPs in response to errors. *Psychophysiology*, *41*(1), 84–95.

Patrick, C. J. (1994). Emotion and psychopathy: Startling new insights. *Psychophysiology*, *31*, 319–330.

Patrick, C. J. (2007). Antisocial personality disorder and psychopathy. In W. O'Donohue, K. A. Fowler, & S. O. Lilienfeld (Eds.), *Handbook of personality disorders* (pp. 109–166). New York: Sage.

Patrick, C. J., & Bernat, E. (2006). The construct of emotion as a bridge between personality and psychopathology. In R. F. Krueger & J. Tackett (Eds.), *Personality and psychopathology* (pp. 174–209). New York: Guilford Press.

Patrick, C. J., Bernat, E., Malone, S. M., Iacono, W. G., Krueger, R. F., & McGue, M. K. (2006). P300 amplitude as an indicator of externalizing in adolescent males. *Psychophysiology*, *43*, 84–92.

Patrick, C. J., & Bernat, E. M. (2009a). From markers to mechanisms: Using psychophysiological measures to elucidate basic processes underlying aggressive externalizing behavior. In S. Hodgins, E. Viding, & A. Plodowski (Eds.), *Persistent violent offenders: Neuroscience and rehabilitation* (pp. 223–250). London: Oxford University Press.

Patrick, C. J., & Bernat, E. M. (2009b). Neurobiology of psychopathy: A two-process theory. In G. G. Berntson & J. T. Cacioppo (Eds.), *Handbook of neuroscience for the behavioral sciences* (pp. 1110–1131). Hoboken, NJ: Wiley.

Patrick, C. J., Berthot, B., & Moore, J. D. (1996). Diazepam blocks fear-potentiated startle in humans. *Journal of Abnormal Psychology*, *105*, 89–96.

Patrick, C. J., Bradley, M. M., & Lang, P. J. (1993). Emotion in the criminal psychopath: Startle reflex modulation. *Journal of Abnormal Psychology*, *102*, 82–92.

Patrick, C. J., Fowles, D. C., & Krueger, R. F. (2009). Triarchic conceptualization of psychopathy: Developmental origins of disinhibi-

tion, boldness, and meanness. *Development and Psychopathology*, *21*(3), 913–938.

Patrick, C. J., Hicks, B. M., Krueger, R. F., & Lang, A. R. (2005). Relations between psychopathy facets and externalizing in a criminal offender sample. *Journal of Personality Disorders*, *19*, 339–356.

Patrick, C. J., & Lang, A. R. (1999). Psychopathic traits and intoxicated states: Affective concomitants and conceptual links. In M. E. Dawson, A. M. Schell, & A. H. Bohmelt (Eds.), *Startle modification: Implications for neuroscience, cognitive science, and clinical science* (pp. 209–230). New York: Cambridge University Press.

Patterson, C. M., & Newman, J. P. (1993). Reflectivity and learning from aversive events: Toward a psychological mechanism for the syndromes of disinhibition. *Psychological Review*, *100*, 716–736.

Peterson, J. B., & Pihl, R. O. (1990). Information processing, neuropsychological function, and the inherited predisposition to alcoholism. *Neuropsychology Review*, *1*, 343–369.

Petrides, M. (2000). Dissociable roles of mid-dorsolateral prefrontal and anterior inferotemporal cortex in visual working memory. *Journal of Neuroscience*, *20*, 7496–7503.

Polich, J., Pollock, V. E., & Bloom, F. E. (1994). Meta-analysis of P300 amplitude from males at risk for alcoholism. *Psychological Bulletin*, *115*, 55–73.

Porjesz, B., Begleiter, H., & Garozzo, R. (1980). Visual evoked potential correlates of information processing deficits in chronic alcoholics. In H. Begleiter (Ed.), *Biological effects of alcohol* (pp. 603–623). New York: Plenum Press.

Ridderinkhoff, K. R., de Vlugt, Y., Bramlage, A., Spaan, M., Elton, M., Snel, J., et al. (2002). Alcohol consumption impairs detection of performance errors in mediofrontal cortex. *Science*, *298*, 2209–2211.

Rolls, E. T. (2000). The orbitofrontal cortex and reward. *Cerebral Cortex*, *10*, 284–294.

Rosen, J. B., & Schulkin, J. (1998). From normal fear to pathological anxiety. *Psychological Review*, *105*, 325–350.

Rothbart, M. K. (1981). Measurement of temperament in infancy. *Child Development*, *52*, 569–578.

Rothbart, M. K., & Ahadi, S. A. (1994). Temperament and the development of personality. *Journal of Abnormal Psychology*, *103*, 55–66.

Scheffers, M., Coles, M. G. H., Bernstein, P., Gehring, W. J., & Donchin, E. (1996). Event-related potentials and error-related processing: An analysis of incorrect responses to go and no-go stimuli. *Psychophysiology*, *33*, 42–53.

Schwartz, C. E., Wright, C. I., Shin, L. M., Kagan, J., & Rauch, S. L. (2003). Inhibited and uninhibited infants grown up: Adult amygdalar response to novelty. *Science*, *300*, 1952–1953.

Sher, K. J., & Trull, T. J. (1994). Personality and disinhibitory psychopathology: Alcoholism and antisocial personality disorder. *Journal of Abnormal Psychology*, *103*, 92–102.

Sellbom, M., Ben-Porath, Y. S., & Bagby, R. M. (2008). On the hierarchical structure of mood and anxiety disorders: Confirmatory evidence and an elaborated model of temperament markers. *Journal of Abnormal Psychology*, *117*, 576–590.

Siever, L. J. (2000). Genetics and neurobiology of personality disorders. *European Psychiatry*, *15*, 54–57.

Siever, L. J., & Davis, K. L. (1991). A psychobiological perspective on the personality disorders. *American Journal of Psychiatry*, *148*, 1647–1658.

Skinner, A. (1982). The Drug Abuse Screening Test. *Addictive Behaviors*, *7*, 363–371.

Skinner, H. A., & Allen, B. A. (1982). Alcohol dependence syndrome: Measurement and validation. *Journal of Abnormal Psychology*, *91*, 199–209.

Spencer, K. M., Dien, J., & Donchin, E. (1999). A componential analysis of the ERP elicited by novel events using a dense electrode array. *Psychophysiology*, *36*(3), 409–414.

Tarter, R. E., Alterman, A. I., & Edwards, K. L. (1985). Vulnerability to alcoholism in men: A behavior-genetic perspective. *Journal of Studies on Alcohol*, *46*, 329–356.

Tellegen, A. (1982). *Brief manual for the Multidimensional Personality Questionnaire*. Unpublished manuscript, University of Minnesota.

Tellegen, A. (1985). Structures of mood and personality and their relevance to assessing anxiety, with an emphasis on self-report. In A. H. Tuma & J. D. Maser (Eds.), *Anxiety and the anxiety disorders* (pp. 681–706). Hillsdale, NJ: Erlbaum.

Tellegen, A., & Waller, N. G. (2008). Exploring personality through test construction: Development of the Multidimensional Personality

Questionnaire. In G. J. Boyle, G. Matthews, & D. H. Saklofske (Eds.), *The Sage handbook of personality theory and assessment: Vol. II. Personality measurement and testing* (pp. 261–292). London: Sage.

Trull, T. J. (1992). DSM-IIIR personality disorders and the five-factor model of personality: An empirical comparison. *Journal of Abnormal Psychology*, *101*, 553–560.

Trull, T. J., & Sher, K. J. (1994). Relationship between the five-factor model of personality and Axis I disorders in a nonclinical sample. *Journal of Abnormal Psychology*, *103*, 350–360.

Vaidyanathan, U., Patrick, C. J., & Bernat, E. M. (2009). Startle reflex potentiation during aversive picture viewing as an index of trait fear. *Psychophysiology*, *46*, 75–85.

Venables, N., Reich, E., Bernat, E. M., Hall, J. R., & Patrick, C. J. (2008). Reduced P300 amplitude in criminal psychopathy is related to externalizing tendencies. *Psychophysiology*, *45*, S46.

Verona, E., Patrick, C. J., & Joiner, T. E. (2001). Psychopathy, antisocial personality, and suicide risk. *Journal of Abnormal Psychology*, *110*, 462–470.

Vollebergh, W. A. M., Iedema, J., Bijl, R. V., de Graaf, R., Smit, F., & Ormel, J. (2001). The structure and stability of common mental disorders: The NEMESIS Study. *Archives of General Psychiatry*, *58*, 597–603.

Vrana, S. R., Constantine, J. A., & Westman, J. S. (1992). Startle reflex modulation as an outcome measure in the treatment of phobia: Two case studies. *Behavioral Assessment*, *14*, 279–291.

Vul, E., Harris, C., Winkielman, P., & Pashler, H. (2009). Puzzlingly high correlations in fMRI studies of emotion, personality, and social cognition. *Perspectives on Psychological Science*, *4*, 274–290.

Wagar, B. M., & Thagard, P. (2004). Spiking Phineas Gage: A neurocomputational theory of cognitive–affective integration in decision-making. *Psychological Review*, *111*, 67–79.

Watson, D. (2005). Rethinking the mood and anxiety disorders. *Journal of Abnormal Psychology*, *114*, 522–536.

Watson, D., O'Hara, M. W., Simms, L. J., Kotov, R., Chmielewski, M., Montez, E., et al. (2007). Development and validation of the Inventory for Depression and Anxiety Symptoms. *Psychological Assessment*, *19*, 253–268.

Whalen, P. J. (1998). Fear, vigilance and ambiguity: Initial neuroimaging studies of the human amygdala. *Current Directions in Psychological Science*, *7*, 177–188.

Widiger, T. A., & Sanderson, C. J. (1995). Towards a dimensional model of personality disorders in DSM-IV and DSM-V. In W. J. Livesley (Ed.), *The DSM-IV personality disorders* (pp. 433–458). New York: Guilford Press.

Wise, S. P., Murray, E. A., & Gerfen, C. R. (1996). The frontal–basal ganglia system in primates. *Critical Reviews in Neurobiology*, *10*, 317–356.

Young, S. E., Stallings, M. C., Corley, R. P., Krauter, K. S., & Hewitt, J. K. (2000). Genetic and environmental influences on behavioral disinhibition. *American Journal of Medical Genetics (Neuropsychiatric Genetics)*, *96*, 684–695.

Zuckerman, M. (1979). *Sensation seeking: Beyond the optimal level of arousal*. Hillsdale, NJ: Erlbaum.

Zuckerman, M. (1999). *Vulnerability to psychopathology: A biosocial model*. Washington, DC: American Psychological Association.

CHAPTER 23

Using Evolutionary Principles for Deducing Normal and Abnormal Personality Patterns

THEODORE MILLON

Owing to the multiple perspectives that can legitimately be brought to bear in defining "normality" and "abnormality" (Offer & Sabshin, 1966; Strack, 2006), as well as the many values, functions, and goals these constructs may serve, there is little likelihood that any set of criteria would be satisfactory to all. That multiple criteria are needed should be self-evident, although the specifics of which they are composed will remain controversial. Given my belief in the applicability of evolutionary principles to all spheres of nature's expression, I have sought to anchor my criteria as closely as possible to these "universal" principles in the realm of human functioning—that is, in those transactions that relate to existential survival (pain–pleasure), ecological adaptation (passive–active), and species replication (others–self).

As the title of this chapter suggests, I am attempting to formulate a schema that is neither doctrinaire nor loosely eclectic in its approach. Rather, the theory presented is intended to be both broad in scope and sufficiently systematic in its application of principles to enable the major varieties of normal and abnormal personality to be derived logically and coherently.

Biosocial Development

For pedagogical purposes, it is often necessary to separate biogenic from psychogenic factors as influences in personality development; this bifurcation does not exist in reality. Biological and experiential determinants combine and interact in a reciprocal interplay throughout life. Each step in this biogenic–psychogenic interplay builds upon prior interactions, and in turn creates new potentialities for future reactivity and experience. Development may be viewed, then, as a process in which intraorganismic and environmental forces display not only a reciprocity and circularity of influence, but an orderly and sequential continuity throughout the life of an individual.

The circular feedback and serially unfolding character of the developmental process defy simplification, and must constantly be kept in mind when the backgrounds of both normal and abnormal personalities are analyzed. There are few unidirectional effects in development; it is a multideterminant transaction in which unique patterns of biogenic potentials and of psychogenic influences mold each other in a reciprocal and successively more intricate fashion.

Each individual is endowed at conception with a unique set of chromosomes that shapes the course of his or her physical maturation and psychological development. The physical and psychological characteristics of children are in large measure similar to those of their parents because they possess many of the same genetic units. Children are genetically disposed to be similar to their parents not only physically, but also in stamina, energy, emotional sensitivity, and intelligence.

Each infant displays a distinctive pattern of behaviors from the first moments after birth. These characteristics are attributed usually to the infant's "nature" (i.e., constitutional makeup), since it is displayed prior to the effects of postnatal influences. It is erroneous to assume that children of the same chronological age are comparable with respect to the level and character of their biological capacities. Each infant not only starts life with a distinctive pattern of neurological, physiochemical, and sensory equipment, but progresses at his or her own maturational rate toward some ultimate but unknown level of potential. Thus, above and beyond initial differences and their not insignificant consequences, differences exist in the rate with which the typical sequence of maturation unfolds. Furthermore, different regions in the complex nervous system within a single child may mature at different rates. To top it all, the potential or ultimate level of development of each of these neurological capacities will vary widely, not only among children but within each child.

The maturation of the biological substrate for psychological capacities is anchored initially to genetic processes, but its epigenetic development is substantially dependent on environmental stimulation. The concept of "stimulus nutriment" may be introduced to represent the belief that the quantity of environmental experience activates chemical processes requisite to the maturation of neural collaterals. "Stimulus impoverishment" may lead to irrevocable deficiencies in neural development and their associated psychological functions; "stimulus enrichment" may prove equally deleterious by producing pathological overdevelopments or imbalances among these functions.

The notion of "sensitive developmental periods" may be proposed to convey the belief that stimuli produce different effects at different ages; that is, there are limited time periods during maturation when particular stimuli have pronounced effects that they do not have either before or after these periods. It may be suggested further that these peak periods occur at points in maturation when the potential is greatest for growth and expansion of neural collaterals and other psychologically relevant structures.

Four neuropsychological stages of development, representing peak periods in neurological maturation, may be proposed. Each developmental stage reflects transactions between constitutional and experiential influences, which combine to set a foundation for subsequent stages; if the interactions at one stage are deficient or distorted, all subsequent stages will be affected, since they rest on a defective base.

The first stage, termed "sensory attachment" in the theory, predominates from birth to approximately 18 months of age. This period is characterized by a rapid maturation of neurological substrates for sensory processes, and by the infant's attachment and dependency on others.

The second stage, referred to as "sensorimotor autonomy," begins roughly at 12 months and extends in its peak development through the 6th year. It is characterized by a rapid differentiation of motor capacities which coordinate with established sensory functions; this coalescence enables the young child to locomote, manipulate, and verbalize in increasingly skillful ways.

The third and fourth stages are called the periods of "pubertal–gender identity" and "intracortical initiative." The former is connected to hormonal changes activated between the 11th and 15th years; the latter is primary from about the 4th year through adolescence. There are rapid growth potentials among the higher cortical centers during this latter stage, enabling the child to reflect, plan, and act independently of parental supervision. Integrations developed during the third stage undergo substantial reorganization as a product of the biological and social effects of puberty.

Maladaptive consequences can arise as a result of either stimulus impoverishment or stimulus enrichment at each of the four stages.

From experimental animal research and naturalistic studies with human infants, it

appears that marked stimulus impoverishment during the period of sensory attachment will produce deficiencies in sensory capacities and a marked diminution of interpersonal sensitivity and behavior. There is little evidence available with regard to the effects of stimulus enrichment during this stage; it may be proposed, however, that excessive stimulation results in hypersensitivities, stimulus-seeking behaviors, and abnormal interpersonal dependencies.

If deprived of adequate stimulation during the stage of sensorimotor autonomy, a child will be deficient in skills for behavioral autonomy, will display a lack of exploratory and competitive activity, and will be characterized by timidity and submissiveness. In contrast, excessive enrichment and indulgence of sensorimotor capacities may result in uncontrolled self-expression, narcissism, and social irresponsibility.

Among the consequences of understimulation during the pubertal–gender identity and intracortical initiative stages are an identity diffusion; an inability to fashion an integrated and consistent purpose for one's existence; and an inefficiency in channeling and directing one's energies, capacities and impulses. Excessive stimulation, in the form of overtraining and overguidance, may result in the loss of several functions—notably spontaneity, flexibility, and creativity.

There has been little systematic attention to children's own contributions to the course of their development. Environmental theorists of personality have viewed disorders as the results of detrimental experiences that individuals have had no part of producing themselves. This is a gross simplification. Each infant possesses a biologically based pattern of reaction sensitivities and behavioral dispositions, which shapes the nature of his or her experiences and may contribute directly to the creation of environmental difficulties.

The biological dispositions of the maturing child are important because they strengthen the probability that certain kinds of behavior will be learned. Highly active and responsive children relate to and learn about their environment quickly. Their liveliness, zest, and power may lead them to a high measure of personal gratification. Conversely, their energy and exploratory behavior may result in excess frustration if they overaspire or run into insuperable barriers; unable to gratify their activity needs effectively, they may grope and strike out in erratic and maladaptive ways.

Adaptive learning in constitutionally passive children is also shaped by their biological equipment. Ill disposed to deal with their environment assertively, and little inclined to discharge their tensions physically, they may learn to avoid conflicts and step aside when difficulties arise. They are less likely to develop guilt feelings about misbehavior than active youngsters, who more frequently get into trouble, receive more punishment, and are therefore inclined to develop aggressive feelings toward others. But in their passivity, these youngsters may deprive themselves of rewarding experiences and relationships; they may feel "left out of things" and become dependent on others to fight their battles and to protect them from experiences they are ill equipped to handle on their own.

It appears clear from studies of early reactivity patterns that constitutional tendencies evoke counterreactions from others that accentuate these initial dispositions. Children's biological endowment shapes not only their behavior, but that of their parents as well. If a child's primary disposition is cheerful and adaptable and has made his or her care easy, the mother will tend quickly to display a positive reciprocal attitude. Conversely, if the child is tense and wound up, or if his or her care is difficult and time-consuming, the mother will react with dismay, fatigue, or hostility. Through their own behavioral dispositions, then, children elicit parental behaviors that reinforce their initial patterns.

Unfortunately, the reciprocal interplay of primary patterns and parental reactions has not been sufficiently explored. It may prove to be one of the most fruitful spheres of research concerning the etiology of psychopathology and merits the serious attention of investigators. The biosocial–learning approach presented in this chapter stems largely from the thesis that children's constitutional patterns shape and interact with their social reinforcement experiences.

The fact that early experiences are likely to contribute a disproportionate share to learned behavior is attributable in part to the fact that their effects are difficult to extinguish. This resistance to extinction stems largely from the fact that learning in early

life is presymbolic, random, and highly generalized. Additional factors contributing to the persistence and continuity of early learnings are social factors, such as the repetitive nature of experience, the tendency for interpersonal relations to be reciprocally reinforcing, and the perseverance of early character stereotypes. Beyond these are a number of self-perpetuating processes derived from an individual's own actions. Among them are protective efforts that constrict the person's awareness and experience, the tendency to distort events both perceptually and cognitively in line with expectancies, the inappropriate generalization to new events of old behavior patterns, and the repetitive compulsion to create conditions that parallel the past.

Children learn complicated sequences of attitudes, reactions, and expectancies in response to the experiences to which they were exposed. Initially, these responses are specific to the particular events that prompted them; they are piecemeal, scattered, and changeable. Over the course of time, however, through learning what responses are successful in obtaining rewards and avoiding punishments, a child begins to crystallize a stable pattern of instrumental behaviors for handling the events of everyday life. These coping and adaptive strategies come to characterize the child's way of relating to others, and constitute one of the most important facets of what we may term his or her "personality pattern."

A balance or imbalance is usually struck between the two extremes of each developmental polarity. A measure of balance among the four basic polarities is an index of normality. Normality does not require precise equidistance between polar extremes. Positions of balance will vary as a function of the overall configurations both within and among polarities, which in turn will depend on the wider ecosystems within which individuals operate. In other words, and as is well recognized, there is no one form or expression of normality. Various polarity positions, and the traits and behaviors they underlie, will permit diverse "styles of normality," just as marked deficits and imbalances among the polarities may manifest themselves in diverse "styles of abnormality" (Millon, 1990).

Given the numerous and diverse ecological milieus that humans face in our complex modern environment, there is reason to expect that most humans will display multiple adaptive styles—sometimes more active, sometimes less so; occasionally focused on self, occasionally on others; at times oriented to pleasure, at times oriented to the avoidance of pain. Despite the presence of relatively enduring and characteristic styles, adaptive flexibility typifies most normal individuals. That is, they are able to shift from one position on a polar continuum to another as the circumstances of life change.

Let us turn next to polarity-based criteria for normality and abnormality. They are grouped, two each, under three polarity headings—namely, "existential survival," "ecological adaptation," and "species replication." Elaborated within each polarity are data or theory supportive of normality, as well as clinical illustrations that demonstrate some of the pathological consequences following from failures.

Existential Survival

An interweaving and shifting balance between the two extremes of the pain–pleasure polarity typifies normality. Both of the following criteria should be met in varying degrees as life circumstances require. In essence, a synchronous and coordinated personal style should have developed to answer the question of whether the person should focus on experiencing only the pleasures of life or should concentrate on avoiding its pains.

Life Preservation: Avoiding Danger and Threat

One might assume that a criterion based on the avoidance of psychic or physical pain would be sufficiently self-evident not to require specification. As is well known, debates have arisen in the literature as to whether mental health or normality reflects the absence of mental disorder (i.e., is merely the reverse side of the mental illness or abnormality coin). That there is a relationship between health and disease cannot be questioned; the two are intimately connected, both conceptually and physically. On the

other hand, to define health solely as the absence of disorder will not suffice. As a single criterion among several, however, features of behavior and experience that signify both the lack of (e.g., anxiety, depression) and an aversion to (e.g., threats to safety and security) pain in its many and diverse forms provide a necessary foundation upon which other, more positively constructed criteria may rest. Substantively, positive normality must include elements beyond mere non-normality or abnormality. And despite the complexities and inconsistencies of personality, normality does preclude non-normality from a definitional point of view.

Turning to the evolutionary aspect of pain avoidance—that pertaining to a distancing from life-threatening circumstances, psychic and otherwise—we find an early historical reference in the writings of Herbert Spencer, a supportive contemporary of Darwin. Spencer (1870) averred:

> Pains are the correlative of actions injurious to the organism, while pleasures are the correlatives of actions conducive to its welfare.
>
> Those races of beings only can have survived in which, on the average, agreeable or desired feelings went along with activities conducive to the maintenance of life, while disagreeable and habitually avoided feelings went along with activities directly or indirectly destructive of life.
>
> Every animal habitually persists in each act which gives pleasure, so long as it does so, and desists from each act which gives pain. . . . It is manifest that in proportion as this guidance approaches completeness, the life will be long; and that the life will be short in proportion as it falls short of completeness.
>
> We accept the inevitable corollary from the general doctrine of Evolution, that pleasures are the incentives to life-supporting acts and pains the deterrents from life-destroying acts. (pp. 279–284)

More recently, Freedman and Roe (1958) wrote:

> We . . . hypothesize that psychological warning and warding-off mechanisms, if properly studied, might provide a kind of psychological evolutionary systematics. Exposure to pain, anxiety, or danger is likely to be followed by efforts to avoid a repetition of the noxious stimulus situation with which the experience is associated. Obviously an animal with a more highly developed system for anticipating and avoiding the threatening circumstance is more efficiently equipped for adaptation and survival. Such unpleasant situations may arise either from within, in its simplest form as tissue deprivation, or from without, by the infliction of pain or injury. Man's psychological superstructure may be viewed, in part, as a system of highly developed warning mechanisms. (p. 458)

As for the biological substrate of "pain" signals, Gray (1975) suggests two systems, both of which alert the organism to possible dangers in the environment. The one mediating the behavioral effects of unconditioned (instinctive?) aversive events is termed the "fight–flight system." This system elicits defensive aggression and escape and is subserved, according to Gray's pharmacological inferences, by the amgydala, the ventromedial hypothalamus, and the central gray of the midbrain; neurochemically, evidence suggests a difficult-to-unravel interaction among aminobutyric acids (e.g., gamma-aminobutyric acid), serotonin, and endogenous opiates (e.g., endorphins). The second major source of sensitivity and action in response to "pain" signals is referred to by Gray as the "behavioral inhibition system" (BIS); it consists of the interplay of the septal–hippocampal system, its cholinergic projections and monoamine transmissions to the hypothalamus, and then transmissions from the hypothalamus to the cingulate and prefrontal cortex. Activated by signals of punishment or nonreward, the BIS suppresses associated behaviors, refocuses the organism's attention, and redirects activity toward alternate stimuli.

"Harm avoidance" is a concept proposed by Cloninger (1986, 1987). He defines it as a heritable tendency to respond intensely to signals of aversive stimuli (pain) and to learn to inhibit behaviors that might lead to punishment and frustrative nonreward. Those high on this dimension are characterized as cautious, apprehensive, and inhibited; those low on this valence are likely to be confident, optimistic, and carefree. Cloninger subscribes essentially to Gray's BIS concept in explicating this polarity, as well as to the neuroanatomical and neurochemical hypotheses Gray has proposed as the substrates for its pain-avoidant mechanisms.

Let us now shift from biological/evolutionary concepts to proposals of a similar cast offered by thinkers of a distinctly psychological turn of mind. Notable here are the contributions of Maslow (1968, 1970), particularly his hierarchical listing of "needs." Best known are the five fundamental needs that lead to self-actualization, the first two of which relate to the evolutionary criterion of life preservation. Included in the first group are the "physiological" needs, such as air, water, food, and sleep—qualities of the ecosystem essential for survival. Next, and equally necessary to avoid danger and threat, are what Maslow terms the "safety" needs, including freedom from jeopardy, the security of physical protection, and psychic stability, as well as the presence of social order and interpersonal predictability.

That pathological consequences can ensue from the failure to attend to the realities that portend danger is obvious; the lack of air, water, and food are not issues of great concern in civilized societies today, although these are matters of considerable importance to environmentalists of the future and in contemporary poverty-stricken nations.

It may be of interest next to record some of the psychic pathologies of personality that can be traced to aberrations in meeting this first criterion of normality. For example, among those described as having avoidant personalities (Millon, 1981), we see an excessive preoccupation with threats to one's psychic security; an expectation of and hyperalertness to the signs of potential rejection lead these persons to disengage from everyday relationships and pleasures. At the other extreme of the criterion, we see a risk-taking attitude, a proclivity to chance hazards and to endanger one's life and liberty—a behavioral pattern characteristic of those we describe as having antisocial personalities. Here there is little of the caution and prudence expected in the normality criterion of avoiding danger and threat. Rather, we observe its opposite: a rash willingness to put one's safety in jeopardy, to play with fire, and to throw caution to the wind. Another pathological style illustrative of a failure to fulfill this evolutionary criterion is seen among those variously designated as having "masochistic" or "self-defeating" personalities. Rather than avoid circumstances that may prove painful and self-endangering, such individuals set in motion situations in which they will come to suffer physically and/or psychically. By virtue of either habit or guilt absolution, they induce rather than avoid pain for themselves.

Life Enhancement: Seeking Rewarding Experiences

At the other end of the pain–pleasure polarity are attitudes and behaviors designed to foster and enrich life—to generate joy, pleasure, contentment, and fulfillment, and thereby to strengthen an individual's capacity to remain vital and competent physically and psychically. This criterion asserts that existence/survival calls for more than life preservation alone; beyond pain avoidance is pleasure enhancement.

This criterion asks us to go at least one step further than Freud's (1940) parallel notion that life's motivation is chiefly that of "reducing tensions" (i.e., avoiding/minimizing pain), and thereby maintaining a homeostatic balance and inner stability. In accord with my view of evolution's polarities, I would assert that normal humans are driven also by the desire to enrich their lives, to seek invigorating sensations and challenges, to venture and explore—all to the end of magnifying (if not escalating) the probabilities of both individual viability and species replicability.

Regarding the key instrumental role of "the pleasures," Spencer (1870) put it well more than a century ago: "Pleasures are the correlatives of actions conducive to [organismic] welfare . . . the incentives to life-supporting acts" (pp. 279, 284). The view that there exists an organismic striving to expand one's inherent potentialities (as well as those of one's kin and species) has been implicit in the literature for ages. That "the pleasures" may be both sign and vehicle for this realization was recorded even in the Talmud: "Everyone will have to justify himself in the life hereafter for every failure to enjoy a legitimately offered pleasure in this world" (quoted in Jahoda, 1958, p. 45).

As far as contemporary psychobiological theorists are concerned, brief mention is made again of the contributions of Gray (1975, 1981) and Cloninger (1986, 1987).

Gray's neurobiological model centers heavily on activation and inhibition (active–passive polarity) as well as on signals of reward and punishment (pleasure–pain polarity). Basing his deductions primarily on pharmacological investigations of animal behavior, Gray has proposed the existence of several interrelated and neuroanatomically grounded response systems that activate various positive and negative affects. He refers to what he terms the "behavioral activation system" (BAS) as an "approach system" that is subserved by the reward center uncovered originally by Olds and Milner (1954). Ostensibly mediated at brainstem and cerebellar levels, it is likely to include dopaminergic projections across various strata and is defined as responding to conditioned rewarding and safety stimuli by facilitating behaviors that maximize their future recurrence (Gray, 1975). There are intricacies in the manner with which the BAS is linked to external stimuli and its anatomical substrates, but Gray views it as a system that subserves signals of reward, punishment relief, and pleasure.

Cloninger (1986, 1987) has generated a theoretical model composed of three dimensions, which he terms "reward dependence," "harm avoidance" (to which I have referred previously), and "novelty seeking." Proposing that each is a heritable personality disposition, he relates them explicitly to specific monoaminergic pathways: High reward dependence is connected to low noradrenergic activity, harm avoidance to high serotonergic activity, and high novelty seeking to low dopaminergic activity. Cloninger's reward dependence dimension reflects highs and lows on the positive/gratifying/pleasure valence, whereas the harm avoidance dimension represents highs and lows on the negative/pain/displeasure valence. Reward dependence is hypothesized to be a heritable neurobiological tendency to respond to signals of reward (pleasure), particularly verbal signals of social approval, sentiment, and succor, as well as to resist events that might extinguish behaviors previously associated with these rewards. Cloninger portrays those high on reward dependence to be sociable, sympathetic, and pleasant; in contrast, those low on this polarity are characterized as detached, cool, and practical. Describing the undergirding substrate for the reward/pleasure valence as the "behavior maintenance system," Cloninger speculates that its prime neuromodulator is likely to be norepinephrine, with its major ascending pathways arising in the pons, projecting onward to hypothalamic and limbic structures, and then branching upward to the neocortex.

Turning again to pure psychological formulations, both Rogers (1963) and Maslow (1968) have proposed concepts akin to my criterion of enhancing pleasure. In his notion of "openness to experience," Rogers asserts that a fully functioning person has no aspect of his or her nature closed off. Such individuals are not only receptive to the experiences that life offers, but are able also to use them in expanding all of life's emotions, as well as being open to all forms of personal expression. In a similar vein, Maslow speaks of the ability to maintain a freshness to experience—to keep up one's capacity to appreciate relationships and events. No matter how often events or persons are encountered, one is neither sated nor bored, but is disposed to view them with an ongoing sense of "awe and wonder."

My view is perhaps less dramatic than the conceptions of either Rogers and Maslow: I believe that this openness and freshness to life's transactions is an instrumental means for extending life, for strengthening one's competencies and options, and for maximizing the viability and replicability of one's species. More mundane and pragmatic in orientation than their views, this conception seems both more substantive theoretically and more consonant a rationale for explicating the role the pleasures play in undergirding "reward experience" and "openness to experience."

As before, a note or two should be recorded on the pathological consequences of a failure to meet a criterion. These are seen most clearly in the schizoid and avoidant personality disorders. In the former there is a marked hedonic deficiency, stemming either from an inherent deficit in affective substrates or the failure of stimulative experience to develop either or both attachment behaviors or affective capacity (Millon, 1981). Among those designated as having avoidant personalities, constitutional sensitivities or abusive life experiences have led to an intense attentional sensitivity to psychic pain and a con-

sequent distrust in either the genuineness or durability of "the pleasures," such that these individuals can no longer permit themselves to experience them. Both of these personality types tend to be withdrawn and isolated, joyless and grim, neither seeking nor sharing in the rewards of life.

Ecological Adaptation

As with the pair of criteria representing the aims of existence, a balance should be achieved between the two criteria comprising modes of adaptation: those related to ecological accommodation and ecological modification, or what I have termed the passive–active polarity. Normality calls for a synchronous and coordinated personal style that weaves a balanced answer to the question of whether one should accept what the fates have brought forth or take the initiative in altering the circumstances of one's life.

Ecological Accommodation: Abiding Hospitable Realities

On first reflection, it would seem to be less than optimal to submit meekly to what life presents—to "adjust" obligingly to one's destiny. As described earlier, however, the evolution of plants is essentially grounded (no pun intended) in environmental accommodation, in an adaptive acquiescence to the ecosystem. Crucial to this adaptive course, however, is the capacity of these surroundings to provide the nourishment and protection requisite to the thriving of a species.

Could the same be true for the human species? Are there not circumstances of life that provide significant and assured levels of sustenance and safekeeping (both psychic and physical?) And if that were the case, would not the acquisition of an accommodating attitude and passive lifestyle be a logical consequence? The answer, it would seem, is yes. If one's upbringing has been substantially secure and nurturant, would it not be "abnormal" to flee or overturn it?

We know that circumstances, other than in infancy and early childhood, rarely persist throughout life. Autonomy and independence is almost inevitable as a stage of maturation, ultimately requiring the adoption of "adult" responsibilities that call for a measure of initiative, decision making, and action. Nevertheless, to the extent that the events of life have been and continue to be caring and giving, is it not perhaps wisest, from an evolutionary perspective, to accept this good fortune and "let matters be"? This accommodating or passive life philosophy has worked extremely well in sustaining and fostering those complex organisms that make up the plant kingdom. Hence passivity, the yielding to environmental forces, may be in itself not only unproblematic but, where events and circumstances provide the pleasures of life and protect against their pains, positively adaptive and constructive. Accepting rather than overturning a hospitable reality seems a sound course; or, as it is said, "If it ain't broke, don't fix it."

Is passivity part of the repertoire of the human species? Does it serve useful functions? and Where and how is it exhibited? A few words in response to these questions may demonstrate that passivity is not mere inactivity, but a stance or process that achieves useful gains.

For example, universal among mammalian species are two basic modes of learning: the respondent or conditioned type, and the operant or instrumental type. The former is essentially a *passive* process; it is the simple pairing of an innate or reflexive response to a stimulus that previously did not elicit that response. In similar passive fashion, environmental elements that occur either simultaneously or in close temporal order become connected to each other in the organism's repertoire of learning, such that if one of these elements recurs in the future, the expectation is that the others will follow or be elicited. The organisms do not have to do anything active to achieve these learnings; inborn reflexive responses and/or environmental events are merely associated by contiguity.

Operant or instrumental learnings, in contrast, represent the outcome of an active process on the organism's part—one requiring effort and execution that have the effect of altering the environment. Whereas respondent conditioning occurs as a result of the passive observation of a conjoining of events, operant conditioning occurs only as a result of an active modification by the organism of

its surroundings; this performance is usually followed either by a positive reinforcer (pleasure) or by the successful avoidance of a negative one (pain). Unconditioned reflexes, such as a leg jerk in reaction to a knee tap, will become a passively acquired conditioned response if a bell is regularly sounded prior to the tap. The shrinking reflex of an eye pupil will become passively conditioned to that bell if it regularly precedes exposure to a shining light.

The passive–active polarity is central to formulations of psychoanalytic theory. Prior to the impressive literature on "self" and "object relations" theory of the 1970s and 1980s, the passive–active antithesis had a major role in both classical "instinct" and post–World War II "ego" schools of analytic thought. The later focus on "self" and "object" is considered in discussions of the third polarity, that of self–other. However, we should not overlook the once key and now less popular constructs of both instinct theory and ego theory. It may be worth noting, as well as of special interest to the evolutionary model presented in this chapter, that the beginnings of psychoanalytic metapsychology were oriented initially to instinctual derivatives (where pleasure and pain were given prominence), and then progressed subsequently to the apparatuses of the ego (Hartmann, 1939; Rapaport, 1953/1967), where passivity and activity were centrally involved.

The model of activity, as described by Rapaport (1953/1967), is a dual one. First, the ego is strong enough to defend against or control the intensity of the id's drive tensions; or, second, through the competence and energy of its apparatuses, the ego is successful in uncovering or creating in reality the object of the id's instinctual drives. Rapaport also conceives the model of passivity as a dual one. First, either the ego gradually modulates or indirectly discharges the instinctual energies of the id; or, second, lacking an adequately controlling apparatus, the ego is rendered powerless and subject thereby to instinctual forces. To translate these formulations into evolutionary terms, effective actions by the ego will successfully manage the internal forces of the id, whereas passivity will result in either accommodations or exposure to the internal demands of the id.

To turn to theorists more directly concerned with normality and health, the humanistic psychologist Maslow (1970) states that "self-actualized" individuals accept their nature as it is, despite personal weaknesses and imperfections. Comfortable with themselves and the world around them, they do not seek to change "the water because it is wet, or the rocks because they are hard" (p. 153). They have learned to accept the natural order of things. Passively accepting nature, they need not hide behind false masks or transform others to fit "distorted needs." Accepting themselves without shame or apology, they are equally at peace with the shortcomings of those with whom they live and relate.

Where do we find clinical non-normality that reflects failures to meet the accommodating/abiding criterion? One example of an inability to leave things as they are is seen in what DSM terms histrionic personality disorder. These individuals achieve their goals of maximizing protection, nurturance, and reproductive success by engaging busily in a series of manipulative, seductive, gregarious, and attention-getting maneuvers. Their persistent and unrelenting manipulation of events is designed to maximize the receipt of attention and favors, as well as to avoid social uninterest and disapproval. They show an insatiable if not indiscriminate search for stimulation and approval. Their clever and often artful social behaviors may give the appearance of an inner confidence and self-assurance; however, beneath this guise lies a fear that a failure on their part to ensure the receipt of attention will shortly result in indifference or rejection, and hence their desperate need for reassurance and repeated signs of approval. Tribute and affection must constantly be replenished and are sought from every interpersonal source. Because they are quickly bored and sated, they keep stirring up things, becoming enthusiastic about one activity and then another. There is a restless stimulus-seeking quality in which they cannot leave well enough alone.

At the other end of the polarity are personality disorders that exhibit an excess of passivity, and thereby a lack of direction in life. Several Axis II disorders demonstrate this passive style, although their passivity derives from and is expressed in apprecia-

bly different ways. Individuals with schizoid personalities, for example, are passive owing to their relative incapacity to experience pleasure and pain; without the rewards these emotional valences normally activate, they are devoid of the drive to acquire rewards, leading them to become apathetically passive observers of the ongoing scene. Those with dependent personalities are typically average on the pleasure–pain polarity, yet they are usually as passive as those with schizoid personalities. Strongly oriented to others, they are notably weak with regard to the self. Their passivity stems from deficits in self-confidence and competence, leading to deficits in initiative and autonomous skills, as well as a tendency to wait for others to assume leadership and guide them. Passivity among individuals with obsessive–compulsive personalities stems from their fear of acting independently, owing to intrapsychic resolutions they have made to quell hidden thoughts and emotions generated by their intense self–other ambivalence. Dreading the possibility of making mistakes or engaging in disapproved behaviors, they became indecisive, immobilized, restrained, and passive. High on pain and low on both pleasure and self, those with self-defeating personalities operate on the assumption that they dare not expect or deserve to have life go their way; giving up any efforts to achieve a life that accords with their "true" desires, they passively submit to others' wishes, acquiescently accepting their fate. Finally, individuals with narcissistic personality disorder, especially high on self and low on others, benignly assume that good things will come their way with little or no effort on their part; this passive exploitation of others is a consequence of the unexplored confidence that underlies their self-centered presumptions.

Ecological Modification: Mastering One's Environment

The active end of the passive–active bipolarity signifies the taking of initiative in altering and shaping life's events. As stated previously, such persons are best characterized by their alertness, vigilance, liveliness, vigor, forcefulness, stimulus-seeking energy, and drive. Some plan strategies and scan alternatives to circumvent obstacles or avoid the distress of punishment, rejection, and anxiety. Others are impulsive, precipitate, excitable, rash, and hasty, seeking to elicit pleasures and rewards. Although specific goals vary and change from time to time, actively aroused individuals intrude on passing events and modify the circumstances of their environment energetically and busily.

Neurobiological research has proven to be highly supportive of the activity or arousal construct ever since Papez (1937), Moruzzi and Magoun (1949), and MacLean (1949, 1952) assigned what were to be termed the "reticular" and "limbic" systems both energizing and expressive roles in the central nervous system.

The first among the historic figures to pursue this theme was Ivan Pavlov. In speaking of the basic properties of the nervous system, Pavlov referred to the strength of the processes of "excitation" and "inhibition," the equilibrium between their respective strengths, and the mobility of these processes. Although Pavlov's (1927) theoretical formulations dealt with what Donald Hebb (1955) termed a "conceptual nervous system," his experiments and those of his students led to innumerable direct investigations of brain activity. Central to Pavlov's thesis was the distinction between strong and weak types of nervous systems.

Closely aligned to Pavlovian theory is Gray's (1964) assertion that those with weak nervous systems are easily aroused, non-sensation-seeking introverts who prefer to experience low stimulation rather than high levels. Conversely, those with strong nervous systems are slow to arouse and thus are likely to be sensation-seeking extroverts who find low stimulation levels boring and high levels both exciting and pleasant.

Akin also to the active modality are the views of Cloninger (1986, 1987). To him, novelty seeking is a heritable tendency toward excitement in response to novel stimuli or cues for reward (pleasure) or punishment relief (pain)—a tendency that leads to exploratory activity. Consonant with its correspondence to the activity polarity, individuals who are assumed to be high in novelty seeking are characterized as impulsive, excitable, and quickly distracted or bored. Conversely, those at the passive polarity or the low end of the novelty-seeking dimension are portrayed as reflective, stoic, slow-

tempered, orderly, and only slowly engaged in new interests.

Turning from ostensive biological substrates to speculative psychological constructs, de Charms (1968) has proposed that "Man's primary motivational propensity is to be effective in producing changes in his environment" (p. 269). A similar view has been conveyed by White (1959) in his concept of "effectance"—an intrinsic motive, as he views it, that activates persons to impose their desires upon environments. de Charms elaborates his theme with reference to man as "Origin" and as "Pawn" (constructs akin to the active polarity on the one hand, and to the passive polarity on the other). He states this distinction as follows:

> That man is the origin of his behavior means that he is constantly struggling against being confined and constrained by external forces, against being moved like a pawn into situations not of his own choosing. . . . An Origin is a person who perceives his behavior as determined by his own choosing; a Pawn is a person who perceives his behavior as determined by external forces beyond his control. . . . An Origin has strong feelings of personal causation, a feeling that the locus for causation of effects in his environment lies within himself. The feedback that reinforces this feeling comes from changes in his environment that are attributable to personal behavior. This is the crux of personal causation, and it is a powerful motivational force directing future behavior. (pp. 273–274)

Allport (1955) argued earlier that history records many individuals who were not content with an existence that offered them little variety, a lack of psychic tension, and minimal challenge. Allport considers it normal to be "pulled forward" by a vision of the future that awakens within persons their drive to alter the course of their lives. He suggests that people possess a need to "invent" motives and purposes that will consume their inner energies. In a similar vein, Fromm (1955) has proposed a human need to rise above the roles of passive creatures in an accidental if not random world. To him, humans are driven to transcend the state of merely having been created; instead, they seek to become the creators, the active shapers of their own destiny. Rising above the passive and accidental nature of existence, humans generate their own purposes and thereby provide themselves with a true basis of freedom.

Species Replication

As before, I consider both of the following criteria necessary to the definition and determination of normality. I see no necessary antithesis between the two. Humans can be both self-actualizing and other-encouraging, although most persons are likely to lean toward one or the other side. A balance that coordinates the two provides a satisfactory answer to the question of whether one should be devoted to the support and welfare of others or should fashion one's life in accord with one's own needs and desires.

Reproductive Nurturance: Constructively Loving Others

As described earlier, recombinant replication achieved by sexual mating entails a balanced though asymmetrical parental investment in both the genesis and nurturance of offspring. By virtue of her small number of eggs and extended pregnancy, the female's strategy for replicative success among most mammals is characterized by the intensive care and protection of a limited number of offspring. Oriented to reproductive nurturance rather than reproductive propagation, most adult human females, at least until recent decades in Western society, bred close to the limit of their capacity, attaining a reproductive ceiling of approximately 20 viable births. By contrast, not only are males free of the unproductive pregnancy interlude for mating, but they may substantially increase their reproductive output by engaging in repetitive matings with as many available females as possible.

The other–self antithesis follows from additional aspects of this asymmetrical replication strategy. Not only must the female be oriented to and vigilant in identifying the needs of, and dangers that may face, each of her few offspring; it is reproductively advantageous for her to be sensitive to and discriminating in her assessment of potential mates. A "bad" mating—one that results in a defective or weak offspring—has graver consequences for the female than for the

male. Not only will such an event appreciably reduce her limited reproductive possibilities and cause her to forgo a better mate for a period of time, but she may exhaust many of her nurturing and protective energies in attempting to revitalize an inviable or infertile offspring. By contrast, if a male indulges in a "bad" mating, all he has lost are some quickly replaceable sperm—a loss that does little to diminish his future reproductive potentials and activities.

Before we turn to other indices and views of the self–other polarity, let us be mindful that these conceptually derived extremes do not evince themselves in sharp and distinct gender differences. Such proclivities are matters of degree, not absolutes—owing not only to the consequences of recombinant "shuffling" and "crossing over" of genes, but to the influential effects of cultural values and social learning. Consequently, most "normal" individuals exhibit intermediate characteristics on this as well as on the other two polarities.

The reasoning behind different replication strategies derives from the concept of "inclusive fitness," the logic of which we owe to the theoretical biologist W. D. Hamilton (1964). The concept's rationale is well articulated by Daly and Wilson (1978):

> Suppose a particular gene somehow disposes its bearers to help their siblings. Any child of a parent that has this gene has a one-half of probability of carrying that same gene by virtue of common descent from the same parent bearer. . . . From the gene's point of view, it is as useful to help a brother or sister as it is to help the child.
>
> When we assess the fitness of a . . . bit of behavior, we must consider more than the reproductive consequences for the individual animal. We must also consider whether the reproductive prospects of any kin are in any way altered. *Inclusive fitness is a sum of the consequences for one's own reproduction, plus the consequences for the reproduction of kin multiplied by the degree of relatedness of those kin.*
>
> An animal's behavior can therefore be said to serve a strategy whose goal is the maximization of inclusive fitness. (pp. 30–31; emphasis added)

Mutual support and encouragement represent efforts leading to reciprocal fitness—a behavioral pattern consonant with Darwin's fundamental notions. Altruism, however, is a form of behavior in which there is denial of self for the benefit of others—a behavioral pattern acknowledged by Darwin himself (1871, p. 130) as seemingly inconsistent with his theory. A simple extrapolation from natural selection suggests that those disposed to engage in self-sacrifice would ultimately leave fewer and fewer descendants; as a consequence, organisms motivated by "self-benefiting" genes would prevail over those motivated by "other-benefiting" genes, a result leading to the eventual extinction of genes oriented to the welfare of others. The distinguished sociobiologist E. O. Wilson (1978, p. 153) states the problem directly: "How then does altruism persist?" An entomologist of note, Wilson has no hesitation in claiming that altruism not only persists but is of paramount significance in the lives of social insects. In accord with his sociobiological thesis, he illustrates the presence of altruism in animals as diverse as birds, deer, porpoises, and chimpanzees, which share food and provide mutual defense. For example, to protect the colony's hives, bees enact behaviors that lead invariably to their death.

Two underlying mechanisms have been proposed to account for cooperative behaviors such as altruism. One derives from the concept of inclusive fitness, briefly described in the preceding paragraphs; Wilson (1978) terms this form of cooperative behavior "hard-core" altruism, by which he means that the act is "unilaterally directed" for the benefit of others and that the bestower neither expects nor expresses a desire for a comparable return. Following the line of reasoning originally formulated by Hamilton (1964), J. P. Rushton (1984), a controversial Canadian researcher who has carried out illuminating r/K studies of human behavior, explicates this mechanism as follows:

> Individuals behave so as to maximize their inclusive fitness rather than only their individual fitness; they maximize the production of successful offspring by both themselves and their relatives. . . . Social ants, for example, are one of the most altruistic species so far discovered. The self-sacrificing, sterile worker and soldier ants . . . share 75% of their genes with their sisters and so by devoting their entire existence to the needs of others . . . they help to propagate their own genes. (p. 6)

Wilson terms the second proposed mechanism underlying other-oriented and cooperative behaviors "soft-core" altruism, to represent his belief that the bestower's actions are ultimately self-serving. The original line of reasoning here stems from Trivers's (1971) notion of "reciprocity," a thesis suggesting that genetically based dispositions to cooperative behavior can be explained without requiring the assumption of kinship relatedness. All that is necessary is that the performance of cooperative acts be mutual (i.e., result in concurrent or subsequent behaviors that are comparably beneficial in terms of enhancing the original bestower's survivability and/or reproductive fertility).

Wilson's conclusion that the self–other dimension is a bedrock of evolutionary theory is worth quoting:

> In order to understand this idea more clearly, return with me for a moment to the basic theory of evolution. Imagine a spectrum of self-serving behavior. At one extreme only the individual is meant to benefit, then the nuclear family, next the extended family (including cousins, grandparents, and others who might play a role in kin selection), then the band, the tribe, chiefdoms, and finally, at the other extreme, the highest sociopolitical units. (1978, p. 158)

Intriguing data and ideas have been proposed by several researchers seeking to identify specific substrates that may relate to the other-oriented polarities. In what has been termed the affiliation/attachment drive, Everly (1988), for example, provides evidence favoring an anatomical role for the cingulate gyrus. Referring to the work of Henry and Stephens (1977), MacLean (1985), and Steklis and Kling (1985), Everly concludes that the ablation of the cingulate eliminates both affiliative and grooming behaviors. The proximal physiology of this drive has been hypothesized as including serotonergic, noradrenergic, and opioid neurotransmission systems (Everly, 1988; Redmond, Maas, & Kling, 1971). MacLean (1985) has argued that the affiliative drive may be phylogenically coded in the limbic system and may undergird the "concept of family" in primates. The drive toward other-oriented behaviors, such as attachment, nurturing, affection, reliability, and collaborative play, has been referred to as the "cement of society" by Henry and Stephens (1977).

Let us move now to the realm of psychological and social proposals. Dorothy Conrad (1952) has specified a straightforward list of constructive behaviors that manifest "reproductive nurturance" in the interpersonal sphere:

> *Has positive affective relationship:* The person who is able to relate affectively to even one person demonstrates that he is potentially able to relate to other persons and to society.
>
> *Promotes another's welfare:* Affective relationships make it possible for the person to enlarge his world and to act for the benefit of another, even though that person may profit only remotely.
>
> *Works with another for mutual benefit.*, The person is largely formed through social interaction. Perhaps he is most completely a person when he participates in a mutually beneficial relationship. (pp. 456–457)

More eloquent proposals of a similar character have been formulated by the noted psychologists Maslow, Allport, and Fromm. According to Maslow (1970), once our basic human safety and security needs are met, we next turn to satisfy our belonging and love needs. Here we establish intimate and caring relationships with significant others, in which it is just as important to give love as it is to receive it. Noting the difficulty in satisfying these needs in our unstable and changing modern world, Maslow sees the basis here for the immense popularity of communes and family therapy. These settings are ways to escape the isolation and loneliness that result from our failures to achieve love and belonging.

One of Allport's (1961) criteria for the "mature" personality, which he terms a warm relating of self to others, refers to the capability of displaying intimacy and love for a parent, child, spouse, or close friend. Here the person manifests an authentic oneness with the other and a deep concern for his or her welfare. Beyond one's intimate family and friends, there is an extension of warmth in the mature person to humankind at large—an understanding of the human condition and a kinship with all peoples.

To Fromm (1968), humans are aware of the growing loss of their ties with nature as well as with each other, feeling increasingly separate and alone. Fromm believes that humans must pursue new ties with others to

replace those that have been lost or can no longer be depended on. To counter the loss of communion with nature, he feels that health requires us to fulfill our need through a closer linkage with humankind—a sense of involvement, concern, and relatedness with the world. And with those with whom ties have been maintained or reestablished, humans must fulfill their other-oriented needs by being vitally concerned with their well-being, as well as fostering their growth and productivity.

In a lovely coda to a paper on the role of evolutionary and human behavior, Freedman and Roe (1958) wrote:

> Since his Neolithic days, in spite of his murders and wars, his robberies and rapes, man has become a man-binding and a time-binding creature. He has maintained the biological continuity of his family and the social continuity of aggregates of families. He has related his own life experiences with the social traditions of those who have preceded him, and has anticipated those of his progeny. He has accumulated and transmitted his acquired goods and values through his family and through his organizations. He has become bound to other men by feelings of identity and by shared emotions, by what clinicians call empathy. His sexual nature may yet lead him to widening ambits of human affection, his acquisitive propensities to an optimum balance of work and leisure, and his aggressive drives to heightened social efficiency through attacks on perils common to all men. (p. 457)

The pathological consequences of a failure to embrace the polarity criterion of "others" are seen most clearly in the antisocial and narcissistic personality disorders. Persons with these disorders exhibit an imbalance in their replication strategy; in this case, however, there is a primary reliance on self rather than others. They have learned that reproductive success as well as maximum pleasure and minimum pain is achieved by turning exclusively to themselves. The tendency to focus on self follows two major lines of development.

In the narcissistic personality, development reflects the acquisition of a self-image of superior worth, learned largely in response to admiring and doting parents. Providing self-rewards is highly gratifying if one values oneself or possesses either a "real" or inflated sense of self-worth. Displaying manifest confidence, arrogance, and an exploitative egocentricity in social contexts, this self-orientation has been termed the "passive–independent" style in the theory, as the individual already has all that is important (him- or herself).

Individuals with narcissistic personalities are noted for their egotistic self-involvement, experiencing primary pleasure simply by passively being or attending to themselves. Early experience has taught them to overvalue their self-worth. This confidence and superiority may be founded on false premises, however; that is, it may be unsustainable by real or mature achievements. Nevertheless, they blithely assume that others will recognize their specialness. Hence they maintain an air of arrogant self-assurance and, without much thought or even conscious intent, benignly exploit others to their own advantage. Although the tributes of others are both welcome and encouraged, their air of snobbish and pretentious superiority requires little confirmation through either genuine accomplishment or social approval. Their sublime confidence that things will work out well provides them with little incentive to engage in the reciprocal give and take of social life.

Those whom the theory characterizes as exhibiting the "active–independent" orientation exhibit the outlook, temperament, and socially unacceptable behaviors of DSM-defined antisocial personality disorder. They act to counter the expectation of pain at the hand of others; this is done by actively engaging in duplicitous or illegal behaviors in which they seek to exploit others for self-gain. Skeptical about the motives of others, they desire autonomy and wish revenge for what are felt as past injustices. Many are irresponsible and impulsive—actions they see as justified because they judge others to be unreliable and disloyal. Insensitivity and ruthlessness with others are the primary means they have learned to head off abuse and victimization.

In contrast to the narcissistic personality, this second pattern of self-orientation develops as a form of protection and counteraction. These individuals turn to themselves, first, to avoid the depredation they anticipate, and second, to compensate by furnishing self-generated rewards in the stead of

this depredation. Learning that they cannot depend on others, they counterbalance this loss not only by trusting themselves alone, but by actively seeking retribution for what they see as past humiliations. Turning to self and seeking actively to gain strength, power, and revenge, they act irresponsibly, exploiting and usurping what others possess as sweet reprisal. Their security is never fully "assured," however, even when they have aggrandized themselves beyond their lesser origins.

In both the narcissistic and antisocial personality disorders, we see non-normality arising from an inability to experience a constructive love for others. In the one, there is an excessive self-centeredness; in the other, there is the acquisition of a compensatory destructiveness driven by social retribution and self-aggrandizement.

Reproductive Propagation: Actualizing Self-Potentials

The converse of reproductive nurturance is *not* reproductive propagation, but rather the lack of reproductive nurturance. Thus failing to love others constructively does not assure the actualization of one's potentials. Both may and should exist in normal/healthy individuals.

Although the dimension of self–other is arranged to highlight its polar extremes, it should be evident that many, if not most, behaviors are employed to facilitate the reproduction of both self and kin. Both aims are often simultaneously achieved; at other times, one or the other may predominate. The behaviors constituting these strategies are "driven," so to speak, by a blend of activation and affect. That is, combinations arise from intermediary positions reflecting both the life enhancement and life preservation polarity of pleasure–pain, interwoven with similar intermediary positions on the ecological accommodation and ecological modification polarity of activity–passivity. Phrasing "replication" in terms of these abstruse and metaphorical constructs does not obscure it, I hope, but rather sets this third polarity on the deeper foundations of existence and adaptation—foundations composed of the first two polarities previously described. Here I provide a few words on certain ostensible biological substrates associated with a self-orientation, outline the views of several contemporary psychologists and psychiatrists who have assigned the criterion of self-actualization a central role in their formulations, and note an example or two of how the failure to meet this criterion can often result in specific pathologies of personality.

At the self-oriented pole, Everly (1988) proposes an autonomy/aggression substrate that manifests itself in a strong need for control and domination, as well as in hierarchical status striving. According to MacLean (1986), it appears that the amygdaloid complex may play a key role in driving organisms into self-oriented behaviors. Early studies of animals with ablated amygdalas showed a notable increase in their docility (Kluver & Bucy, 1939), and nonhuman primates thus treated have exhibited significant decreases in social hierarchy status (Pribram, 1962). Although the evidence remains somewhat equivocal, norepinephrine and dopamine seem to be the prime neurotransmitters of this drive; the hormone testosterone appears similarly implicated (Feldman & Quenzar, 1984).

To turn to psychological constructs that parallel the notion of self-actualization, their earliest equivalent appeared in the writings of Spinoza (1677/1986), who viewed development as that of becoming what one was intended to be—nothing other than that, no matter how exalted the alternative might appear to be. Carl Jung's (1961) concept of individuation also shares important features with that of self-actualization, in that any deterrent to becoming the individual one may have become would be detrimental to life. Any imposed "collective standard is a serious check to individuality," injurious to the vitality of the person—a form of "artificial stunting."

Perhaps it was my own early mentor, Kurt Goldstein (1939, 1940), who first applied the "self-actualization" designation to the concept under review. As he phrased it, "There is only one motive by which human activity is set going: the tendency to actualize oneself" (1939, p. 196). The early views of Jung and Goldstein have been enriched by later theorists, notably Fromm, Perls, Rogers, and Maslow. Focusing on what he terms the "sense of identity," Fromm (1955) has spoken of the need to establish oneself as

a unique individual, a state that places the person apart from others. Furthermore—and it is here where Fromm makes a distinct self-oriented commitment—the extent to which this sense of identity emerges depends on how successful the person is in breaking "incestuous ties" to the family or clan. Persons with well-developed feelings of identity experience being in control of their lives, rather than being controlled by the lives of others.

Perls (1969) has enlarged on this theme by contrasting self-regulation and external regulation. Normal/healthy persons do their own regulating, with no external interference (whether this stems from the needs and demands of others or from the strictures of a social code). What we must actualize is the "true inner self," not an image we have of what our ideal selves should be. That is the "curse of the ideal." To Perls, each person must be what he or she "really is."

Following the views of his forerunners, Maslow (1970) has stated that self-actualization is the "supreme development" and use of all our abilities, ultimately becoming what we have the potential to become. Noting that self-actualists often require detachment and solitude, Maslow has asserted that such persons are strongly self-centered and self-directed, make up their own minds, and reach their own decisions, without the need to gain social approval.

In like manner, Rogers (1963) as posited a single, overreaching motive for normal/healthy persons: maintaining, actualizing, and enhancing their own potential. The goal is not to maintain a homeostatic balance or a high degree of ease and comfort, but rather to move forward in becoming what is intrinsic to self and to enhance further that which one has already become. Believing that humans have an innate urge to create, Rogers has stated that the most creative product of all is one's own self.

Where do we see failures in the achievement of self-actualization, a giving up of self to gain the approbation of others? Two personality disorders can be drawn upon to illustrate forms of self-denial. First, those with dependent personalities have learned that feeling good, secure, confident, and so on—that is, any feeling associated with pleasure or the avoidance of pain—is provided almost exclusively in their relationships with others. Behaviorally, these persons display a strong need for external support and attention; should they be deprived of affection and nurturance, they will experience marked discomfort, if not sadness and anxiety. Any number of early experiences may set the stage for this other-oriented imbalance. Dependent individuals often include those who have been exposed to an overprotective training regimen and who thereby fail to acquire competencies for autonomy and initiative. Experiencing peer failures and low self-esteem leads them to forgo attempts at self-assertion and self-gratification. They learn early that they themselves do not readily achieve rewarding experiences; these experiences are secured better by leaning on others. They learn not only to turn to others as their source of nurturance and security, but to wait passively for others to take the initiative in providing safety and sustenance. Clinically, most are characterized as searching for relationships in which others will reliably furnish affection, protection, and leadership. Lacking both initiative and autonomy, they assume a dependent role in interpersonal relations—accepting what kindness and support they may find, and willingly submitting to the wishes of others in order to maintain nurturance and security.

A less benign but equally problematic centering on the wishes of others and the denial of self is seen in those with obsessive–compulsive personalities. These persons display a picture of distinct other-directedness, a consistency in social compliance and interpersonal respect. Their histories usually indicate having been subjected to constraint and discipline when they transgressed parental strictures and expectations. Beneath the conforming other-oriented veneer they exhibit are intense desires to rebel and assert their own self-oriented feelings and impulses. They are trapped in ambivalence: To avoid intimidation and punishment, they have learned to deny the validity of their own wishes and emotions, and in their stead have adopted as "true" the values and precepts set forth by others. The disparity they sense between their own urges and the behaviors they must display to avoid condemnation often leads to omnipresent physical tensions and rigid psychological controls.

A Theoretical Formulation of Personality Patterns

As noted above, in the first years of life, children engage in a wide variety of spontaneous behaviors. Although they display certain characteristics consonant with their innate or constitutional dispositions, their ways of reacting to others and coping with their environment tend at first to be capricious and unpredictable; flexibility and changeability characterize their moods, attitudes, and behaviors. These seemingly random behaviors serve an exploratory function; each child is "trying out" and testing during this period alternative modes for coping with the environment. As time progresses, the child learns which techniques "work"—that is, which of these varied behaviors enable him or her to achieve desires and avoid discomforts. Endowed with a distinctive pattern of capacities, energies, and temperaments, which serve as a base, the child learns specific preferences among activities and goals. Perhaps of greater importance, the child learns that certain types of behaviors and strategies are especially successful for him or her in obtaining these goals. In interactions with parents, siblings, and peers, he or she learns to discriminate which goals are permissible, which are rewarded, and which are not.

Throughout these years, then, a shaping process takes place in which the range of initially diverse behaviors is narrowed down and finally, crystallizes into particular preferred modes of seeking and achieving. In time, these behaviors persist and become accentuated; not only are they highly resistant to extinction, but they are reinforced by the restrictions and repetitions of a limited social environment, and are perpetuated and intensified by the child's own perceptions, needs, and actions. Thus, given a continuity in basic biological equipment, and a narrow band of experiences for learning behavioral alternatives, the child develops a distinctive pattern of characteristics that are deeply etched, cannot be eradicated easily, and pervade every facet of his or her functioning. In short, these characteristics *are* the essence and sum of the child's personality—automatic ways of perceiving, feeling, thinking, and behaving.

When I speak of a "personality pattern," then, I am referring to those intrinsic and pervasive modes of functioning that emerge from the entire matrix of an individual's developmental history, and that now characterize the person's perceptions and ways of dealing with his environment. I have chosen the term "pattern" for two reasons: first, to focus on the fact that these behaviors and attitudes derive from the constant and pervasive interaction of both biological dispositions and learned experience; and second, to denote the fact that these personality characteristics are not just a potpourri of unrelated behavior tendencies, but a tightly knit organization of needs, attitudes, and behaviors. People may start out in life with random and diverse reactions, but the repetitive sequence of reinforcing experiences to which they are exposed gradually narrows their repertoires to certain habitual strategies, perceptions, and behaviors, which become prepotent and come to characterize their distinctive ways of relating to the world.

I now turn to a formulation that employs a set of theoretical concepts for deducing and coordinating personality syndromes. The full scope of this schema has been published in earlier texts (Millon, 1969, 1981, 1990, 1996). Identified as a biosocial–learning and evolutionary theory, it attempts to generate established and recognized personality categories through formal deduction and to show their covariation with other mental disorders.

Personality patterns may be viewed as complex forms of instrumental coping behaviors—that is, ways of achieving positive reinforcements and avoiding negative reinforcements. These strategies reflect what kinds of reinforcements individuals have learned to seek or avoid (pleasure–pain), where individuals look to obtain them (self–others), and how individuals have learned to behave in order to elicit or escape them (active–passive).

A major distinction derived from the theoretical model is that people may be differentiated in terms of whether their primary source of reinforcement is within themselves or within others. This distinction corresponds to an interpersonally imbalanced spectrum—namely, the dependent and independent personality patterns. Individuals with dependent patterns are those who have learned that feeling good, secure, confident, and so on—that is, those feelings associated

with pleasure or the avoidance of pain—are best provided by others. Behaviorally, these personalities display a strong need for external support and attention; should they be deprived of affection and nurturance they will experience marked discomfort, if not sadness and anxiety. Independent patterns, in contrast, are characterized by a reliance on the self. These individuals have learned that they obtain maximum pleasure and minimum pain if they depend on themselves rather than others. In both the dependent and independent patterns, individuals demonstrate a distinct preference as to whether to turn to others or to themselves to gain security and comfort.

Such clear-cut commitments are not made by all personalities. Some, whom I describe as "ambivalent" or "dissonant," remain unsure as to which way to turn; that is, they are in conflict regarding whether to depend on themselves for reinforcement or on others. Some of these patients vacillate between turning to others in agreeable conformity at one point, and turning to themselves in efforts at independence the next. Other ambivalent personalities display overt dependence and compliance; beneath these outwardly conforming behaviors, however, are strong desires to assert independent and often hostile feelings and impulses.

Finally, certain patients are characterized by their diminished ability to experience both pain and pleasure; they have neither a normal need for pleasure nor a normal need to avoid punishment. Other patients are also distinguished by a diminished ability to feel pleasurable reinforcers, but they are notably sensitive to pain; life is experienced as possessing few gratifications and much anguish. Both groups share a deficient capacity to sense pleasurable reinforcers, although one is hyperreactive to pain. I describe both of these as "detached" patterns; unable to experience rewards from themselves or from others, such persons drift increasingly into socially isolated and self-alienated behaviors.

Another theory-derived distinction reflects the fact that people instrumentally elicit the reinforcements they seek in essentially one of two ways: actively or passively. Descriptively, those who are typically active tend to be characterized by their alertness, vigilance, persistence, decisiveness, and ambitiousness in a goal-directed behavior. They plan strategies, scan alternatives, manipulate events, and circumvent obstacles—all to the end of eliciting pleasures and rewards, or avoiding the distress of punishment, rejection, and anxiety. Although their goals may differ from time to time, they initiate events and are enterprising and energetically intent on controlling the circumstances of their environment. By contrast, persons who are typically passive engage in few overtly manipulative strategies to gain their ends. They often display a seeming inertness, a lack of ambition and persistence, an acquiescence, and a resigned attitude in which they initiate little to shape events and wait for the circumstances of their environment to take their course.

Using these polarities as a basis, the evolution-based theory derives a classification that combines in a 5×2 matrix the dependent, independent, ambivalent, dissonant, and detached styles with the activity–passivity dimension. This produces 10 basic types, with 5 severe variants, for a total of 15 theory-derived personality patterns. Despite their close correspondence to the official DSM personality disorders, these patterns of personality spectra are conceived as heuristic and not as reified diagnostic entities. Readers interested in the several variants deduced from the theoretical model may wish to look into other books (Millon, 1996; Millon & Grossman, 2007a, 2007b, 2007c; Millon, in press).

Acknowledgment

Portions of this chapter have been adapted and updated from Millon and Davis (1996). Copyright 1996 by John Wiley & Sons. Adapted by permission.

References

Allport, G. (1955). *Becoming: Basic considerations for a psychology of personality.* New Haven, CT: Yale University Press.

Allport, G. (1961). *Pattern and growth in personality.* New York: Holt, Rinehart & Winston.

Cloninger, C. R. (1986). A unified biosocial theory of personality and its role in the develop-

ment of anxiety states. *Psychiatric Developments*, *3*, 167–226.
Cloninger, C. R. (1987). A systematic method for clinical description and classification of personality variants. *Archives of General Psychiatry*, *44*, 573–588.
Conrad, D. C. (1952). Toward a more productive concept of mental health. *Mental Hygiene*, *36*, 456–466.
Daly, M., & Wilson, M. (1978). *Sex, evolution and behavior*. Boston: Grant Press.
Darwin, C. R. (1871). *The descent of man and selection in relation to sex*. London: Murray.
de Charms, R. (1968). *Personal causation: The internal affective determinants of behavior*. New York: Academic Press.
Everly, G. (1988, August). *The biological basis of personality: The contribution of paleo-cortical anatomy and physiology*. Paper presented at the First International Congress on Disorders of Personality, Copenhagen, Denmark.
Feldman, R., & Quenzar, L. (1984). *Fundamentals of neuropharmacology*. Sunderland, MA: Sinauer.
Freedman, L. Z., & Roe, A. (1958). Evolution and human behavior. In A. Roe & G. Simpson (Eds.), *Behavior and evolution* (pp. 455–479). New Haven, CT: Yale University Press.
Freud, S. (1940). *An outline of psychoanalysis*. New York: Live Right.
Fromm, E. (1955). *The sane society*. New York: Holt, Rinehart & Winston.
Fromm, E. (1968). *The revolution of hope: Toward a humanized technology*. New York: Harper & Row.
Goldstein, K. (1939). *The organism*. New York: American Book.
Goldstein, K. (1940). *Human nature in the light of psychopathology*. Cambridge, MA: Harvard University Press.
Gray, J. A. (Ed.). (1964). *Pavlov's typology*. New York: Pergamon Press.
Gray, J. A. (1975). *Elements of a two-process theory of learning*. New York: Academic Press.
Gray, J. A. (1981). A critique of Eysenck's theory of personality. In H. J. Eysenck (Ed.), *A model for personality*. New York: Springer-Verlag.
Hamilton, W. D. (1964). The genetical evolution of social behavior: I and II. *Journal of Theoretical Biology*, *7*, 1–52.
Hartmann, H. (1939). *Ego psychology and the problem of adaptation*. New York: International Universities Press.
Hebb, D. O. (1955). Drives and the C.N.S. (conceptual nervous system). *Psychological Review*, *62*, 243–254.
Henry, J. P., & Stephens, P. (1977). *Stress, health, and the social environment*. New York: Springer-Verlag.
Jahoda, M. (1958). *Current concepts of positive mental health*. New York: Basic Books.
Jung, C. G. (1961). *Memories, dreams, reflections*. New York: Vintage Books.
Kluver, H., & Bucy, P. (1939). Preliminary analysis of functions of the temporal lobes in monkeys. *Archives of Neurology and Psychiatry*, *42*, 979–1000.
MacLean, P. (1949). Psychosomatic disease and the "visceral brain." *Psychosomatic Medicine*, *11*, 338–353.
MacLean, P. (1952). Some psychiatric implications of physiologic studies on fronto temporal portions of the limbic system. *Electroencephalography and Clinical Neurophysiology*, *4*, 407–418.
MacLean, P. (1985). Brain evolution relating to family, play, and the separation call. *Archives of General Psychiatry*, *42*, 405–417.
MacLean, P. (1986). Culminating developments in the evolution of the limbic system. In B. Doane & K. Livingston (Eds.), *The limbic system*. New York: Raven Press.
Maslow, A. H. (1968). *Toward a psychology of being* (2nd ed.). New York: Van Nostrand.
Maslow, A. H. (1970). *Motivation and personality* (2nd ed.). New York: Harper & Row.
Millon, T. (1969). *Modern psychopathology*. Philadelphia: Saunders.
Millon, T. (1981). *Disorders of personality: DSM-III, Axis II*. New York: Wiley-Interscience.
Millon, T. (1990). *Toward a new personology: An evolutionary model*. New York: Wiley-Interscience.
Millon, T. (in press). *Disorders of personality* (3rd ed.). Hoboken, NJ: Wiley.
Millon, T. (with Davis, R. D.). (1996). *Disorders of personality: DSM-IV and beyond* (2nd ed.). New York: Wiley.
Millon, T., & Grossman, S. (2007a). *Moderating severe personality disorders: A personalized psychotherapy approach*. Hoboken, NJ: Wiley.
Millon, T., & Grossman, S. (2007b). *Overcoming resistant personality disorders: A personalized psychotherapy approach*. Hoboken, NJ: Wiley.
Millon, T., & Grossman, S. (2007c). *Resolving difficult clinical syndromes: A personal-*

ized psychotherapy approach. Hoboken, NJ: Wiley.

Moruzzi, G., & Magoun, H. (1949). Brain stem reticular formation and activation of the EEG. *Electroencephalography and Clinical Neurophysiology*, *1*, 45–73.

Offer, D., & Sabshin, M. (1966). *Normality*. New York: Basic Books.

Olds, J., & Milner, P. (1954). Positive reinforcement produced by the electrical stimulation of septal region and other regions of rat brain. *Journal of Comparative and Physiological Psychology*, *47*, 419–427.

Papez, J. (1937). A proposed mechanism of emotion. *Archives of Neurology and Psychiatry*, *38*, 725–743.

Pavlov, I. (1927). *Conditioned reflexes*. Oxford, UK: Oxford University Press.

Perls, F. (1969). *Gestalt therapy verbatim*. Lafayette, CA: Real People Press.

Pribram, K. (1962). Interrelations of psychology and the neurological disciplines. In S. Koch (Ed.), *Psychology: A study of a science* (pp. 119–157). New York: McGraw-Hill.

Rapaport, D. (1967). Some metapsychological considerations concerning activity and passivity. In M. M. Gill (Ed.), *The collected papers of David Rapaport* (pp. 530–568). New York: Basic Books. (Original work published 1953)

Redmond, D., Maas, J., & Kling, A. (1971). Social behavior of monkeys selectively depleted of monoamines. *Science*, *174*, 428–431.

Rogers, C. R. (1963). Toward a science of the person. *Journal of Humanistic Psychology*, *3*, 79–92.

Rushton, J. P. (1984). Sociobiology: Toward a theory of individual and group differences in personality and social behavior. In J. R. Royce & L. P. Moos (Eds.), *Annals of theoretical psychology* (Vol. 2, pp. 1–48). New York: Plenum Press.

Spencer, H. (1870). *The principles of psychology*. London: Williams & Norgate.

Spinoza, B., de. (1986). *Ethics: On the correction of understanding*, London: Dent. (Original work published 1677)

Steklis, H., & Kling, A. (1985). Neurobiology of affiliation in primates. In M. Reite & T. Field (Eds.), *The psychobiology of attachment and separation*. Orlando, FL: Academic Press.

Strack, S. (Ed.). (2006). *Differentiating normal and abnormal personality* (2nd ed.). New York: Springer.

Trivers, R. L. (1971). The evolution of reciprocal altruism. *Quarterly Review of Biology*, *46*, 35–57.

White, R. W. (1959). Motivation reconsidered: The concept of competence. *Psychological Review*, *66*, 297–323.

Wilson, E.O. (1978). *On human nature*. Cambridge, MA: Harvard University Press.

CHAPTER 24

Biopsychosocial Models and Psychiatric Diagnosis

JOEL PARIS

The DSM System: Etiology and Phenomenology

Classification of diseases in medicine should, in principle, be based on the identification of specific etiological pathways and specific methods of treatment. When the origins of pathology remain a mystery, and when no established method of treatment exists, categories tend to describe syndromes rather than diseases. With few exceptions, this has been the case for all classifications of mental disorders.

The first two editions of DSM attempted to categorize mental disorders on the basis of theoretical concepts. But when theories are wrong, categories derived from them will not be valid. Thus neuroses cannot be defined by "unconscious conflict" if no one knows how to measure such a construct. Nor can depressions cannot be divided into "reactive" and "endogenous" types if mood disorders depend on gene–environment interactions.

DSM-III (American Psychiatric Association, 1980) moved sharply away from unproven theories. It cut the Gordian knot by avoiding discussion of etiology wherever it was unknown (i.e., almost everywhere). By restricting diagnostic criteria to observable phenomena, the DSM system attempted to adopt an "atheoretical" orientation. The change was in most ways salutary; however, DSM-III never stimulated studies to determine whether its categories were valid. In the past 30 years, there has been very little systematic research examining the discriminant validity of criteria for the mental disorders listed in the manual.

In the meantime, theory crept back in by stealth. A biological model currently dominates contemporary psychiatry. The DSM categories have been around for so long that many people assume they are real diseases. Whereas in medicine the pathophysiology of many diseases has been mapped out, this is not the case for psychiatry. Attempts to find biological correlates for each diagnosis have thus far been largely unsuccessful. But the ideology of biological psychiatry continues to support hope. Some authors (e.g., Insel & Quirion, 2005) propose that genes, brain imaging, neurotransmitters, and neural networks will be the basis of future classifica-

tions, and that psychiatry will rejoin neurology as a new discipline based on "applied neuroscience."

Unfortunately, neuroscience is not sufficiently advanced to answer the most basic questions about the causes of mental disorders. We can take bets on the future, but no one knows whether its promise will be fulfilled. Although no one can doubt that great progress has been made in genetics, neurochemistry, and imaging, it has shed more light on how the brain works than on the causes of mental illness.

With these limitations in mind, a phenomenological approach to diagnosis will have to remain in use for some time to come. Moreover, it is doubtful whether genes and biological markers will *ever* be able, by themselves, to describe disease processes. Biology is usually associated with traits and endophenotypes, not with disorders (Gottesman & Gould, 2003). These processes underlie vulnerability to disorder, but do not determine whether overt pathology will emerge. By and large, symptoms develop in response to interactions between genes and environmental stressors (Caspi et al., 2002, 2003).

If etiology is complex and interactive, it may be impractical for DSM-V or its successors to apply it to classification. We are unlikely to be able to define mental disorders on the basis of a single gene, a single protein, or a single neurotransmitter. A broader model is needed to address the complexity of mental illness.

The Biopsychosocial Model

George Engel introduced the "biopsychosocial" (BPS) model to solve problems in the theory and practice of medicine. Engel was a psychiatrist at the University of Rochester who had training in psychoanalysis, but whose main interest was in psychosomatic medicine. Engel was concerned that medicine had become reductionistic in theory and inhumane in practice. Physicians no longer felt it necessary to listen to patients and their life histories, but were satisfied to be engineers repairing broken body parts. In Engel's view, taking psychosocial factors into account was necessary to provide better care.

Engel (1977) published a seminal article in the journal *Science* describing a new and broader model to support a humane and nonreductionistic approach to disease and to patients. Then, in the same year that DSM-III was published, Engel (1980) wrote an article in the *American Journal of Psychiatry* that applied the BPS model to his own specialty. Engel touched a raw nerve, and the BPS model gained many adherents. Many psychiatrists felt that their specialty, once the province of humanism, had become flat-footedly reductionistic. With the decline of influence of psychoanalysis (and all forms of psychotherapy), psychiatry aspired to be as valid as internal medicine. But in spite of the protests of Engel (1997) and other leaders of psychiatry, the trend has continued, and theory and practice have become even more biomedical (Paris, 2008).

Biological models in psychiatry propose that maladies of the mind can be explained at the level of genes, proteins, and neurons. They seek to replicate discoveries of molecular processes that have been shown to underlie diseases affecting other organ systems. Furthermore, this approach is expected to lead to the development of pharmacological interventions that could have specific effects on abnormal molecules.

The problem with these biological models is that they assume that everything can be explained by reducing behavioral complexity to the molecular level. They fail to consider that complex systems have emergent properties, which do not depend on components. Models such as general systems theory (Bertalanffy, 1975) have emphasized this point, but have never been very influential in medicine.

Biological models are actually not even adequate to explain the causes of diseases affecting organs other than the brain. One cannot understand coronary artery disease or cancer without considering gene–environment interactions between underlying vulnerability and exposure to environmental stressors deriving from diet or lifestyle. This point tends to be downplayed when physicians applaud therapeutic triumphs produced by drug development.

It must be admitted, however, that nonbiological models can be equally reductionistic. Until a few decades ago, psychiatry, dominated by psychosocial models, was

dramatically different from all other medical specialties. Whether theories focused on bad parents or on social stress, they ignored biological vulnerability, and modeled psychopathology through linear cause-and-effect relationships, rather than interactions.

Psychiatry has been deeply divided by such narrow perspectives, and integrative points of view are fairly recent developments. Over 50 years ago, Hollingshead and Redlich (1958) described practitioners as falling into one of two opposed and ideologically rigid camps: One camp prescribed drugs, while the other almost exclusively used talking therapy. Organic psychiatrists were contemptuous of psychotherapists, while psychotherapists felt they had little to learn from medical tradition. This dichotomy was also reflected in the way mental illness was classified. Disorders were seen as *either* organic or psychogenic (as in the case of depression). The idea that most diseases fall into *both* categories has only recently taken hold.

Thus, although no one doubted that delirium and dementia reflect biological changes in the brain, there was disagreement as to whether the same principle could be applied to common mental disorders, particularly anxiety and mood disorders. When DSM-III was published, it met passionate opposition from some clinicians (particularly psychoanalysts) who viewed its approach as a rejection of human psychology. DSM-III also met opposition from social psychiatrists who opposed its universalist view of mental illness.

But psychosocial ideas in psychiatry eventually suffered a great defeat. By dismissing biology, psychological and social theorists guaranteed their own failure. It has been clearly shown that every mental disorder has a biological and genetic component (Paris, 1999). It has also been shown that many disorders can be often treated more effectively with pharmacology than with psychotherapy (Paris, 2008). The result was that psychological and social ideas came close to being discredited.

While biology was triumphant, prominent psychiatrists (Eisenberg, 2004) pointed out that its approach can be simplistic. Decoding the genome might identify susceptibility to disease, but does not solve the problem of how mental disorders develop. The need for a broader model remained.

The BPS Model and Psychiatry

The idea that mental disorders arise from interactions among biological, psychological, and social factors is not new. Adolf Meyer, a psychiatrist who influenced a generation of American academic leaders (Shorter, 1997), taught a version of this model to his students called "psychobiology" (Meyer, 1952). Roy Grinker (1969), the psychoanalyst who founded the *Archives of General Psychiatry*, actively promoted general systems theory as an alternative to reductionism. Ironically, though Grinker always took the position that mental phenomena cannot be reduced to effects at the molecular level, after his death his journal largely reflected the biological *Zeitgeist* of contemporary psychiatry.

George Engel (1980) had hoped that the BPS paradigm could bridge the factionalism that has so long affected the discipline of psychiatry. He hoped that the model would be an antidote to the tendency to fragment into "schools"—a phenomenon that Kuhn (1970) described as reflecting immature science. Neither the popular view that life's adversities lead inevitably to mental disorders, nor the view that mental illnesses are due to aberrant molecules, is consistent with a BPS model.

Since the BPS model is integrative, it is in accord with changes in medicine, in psychology, and in science as a whole. Direct relationships between cause and effect are rare in nature. Instead, most phenomena are determined by interactions. Modern statistical science reflects this change in perspective, replacing simple *t*-tests, correlations, and chi-squares with complex regression, path-analytic, and model-testing methods that consider multiple predictors and multiple outcomes. But it requires effort to think interactively; the human mind seems to be constructed to prefer linearity.

If one takes the BPS model seriously, one implication is that one cannot understand psychopathology without knowledge of many disciplines. Unfortunately, it is rare for psychosocial theorists to know much about biology. It is equally rare for biological theorists to have a deep knowledge of psychosocial risks. The result is that studies using sophisticated measures of all aspects of the BPS model are unusual.

The nonlinearity of an integrative model leads inevitably to the conclusion that there are multiple pathways to disorder. Many risk factors lead to a single outcome; different mixtures of risk and protective factors can lead to the same outcome; and the same risk and protective factors can yield many different outcomes. The concepts that different risks can produce the same outcome ("equifinality"), and that similar risks can produce different outcomes ("multifinality"), are defining principles in developmental psychopathology (Cicchetti & Rogosch, 1996).

The BPS model can also lead to a broad approach to clinical practice. In many of the disorders seen by mental health professionals, multiple interventions are required. Patients often need drugs to contain biological vulnerabilities. They also need psychotherapy to help them deal with life stressors. They may also benefit from management of their social environment. Most patients need all of these interventions. Treatment should be as broad and interactive as our current concepts of etiology are. Unfortunately, integrative therapy is more the exception than the rule in practice.

Criticisms of the BPS Model

The BPS model has been popular among clinicians, in that everyone adheres to it in principle. How could anyone be against this theory? After all, we all like to think of ourselves as broad-minded.

Postgraduate educational programs for residents in psychiatry, as well as board exams, often expect a BPS "formulation" of cases (Ghaemi, 2006). This procedure trains young psychiatrists to think about multiple pathways to disease. However, it is not clear whether such procedures meet the standards they set for themselves (McClain, O'Sullivan, & Clardy, 2004). It is also not known whether this exercise affects the way psychiatrists behave once they are in practice.

The BPS paradigm could be used as an excuse for sloppy thinking. The model does not tell us much about the specific pathways that lead to disorder. It does not identify the sufficient conditions for developing pathology, or even which risk factors are necessary. The BPS model lacks precision, and sometimes threatens to turn into meaningless mush.

Several criticisms of the BPS model along these lines have been published (Ghaemi, 2006; McLaren, 2000; Richter, 1999). The gist of these arguments is that the eclecticism of BPS can be used to justifies an "anything goes" approach. Ghaemi (2006) has proposed that pluralism (Jaspers, 1913/1997) is a better model, in that each element of a complex theory is subject to systematic scientific scrutiny. But all he is saying is that we should not wave our hands and call ourselves "biopsychosocial" without solid evidence about risks, pathways, and development.

Some Factors Are More Equal Than Others

The biological, psychological, and social factors in mental disorders are not necessarily equal. Biological vulnerability is a necessary but not sufficient condition for most disorders, while traits define the specificity determining which disorders can develop in any individual (Paris, 1998).

This line of thought suggests an alternative to (or revision of) the BPS model: "stress–diathesis" theory (Monroe & Simons, 1991). The difference is that instead of allowing for *any* mixture of risk factors, a stress–diathesis model proposes that no matter how strong the stress, disorder will not develop without vulnerability. Stress functions to tip an individual over from underlying diatheses to overt symptoms. Thus, whereas adherence to a BPS model requires us to consider all risk factors, this model gives biology a certain degree of primacy. Biological vulnerability would be a necessary factor in most disorders, while psychosocial stressors would not be.

Unfortunately, the BPS and stress–diathesis models have only occasionally been applied in research. As noted above, investigators tend to be trained in narrow paradigms, and do not necessarily collaborate with colleagues with different skills and interests. Thus research continues to be based on small communities that reflect ideological divisions between the "two cultures" of psychiatry. Biological research focuses on diatheses, often failing to consider the role of stressors. Psychosocial research focuses on stressors, only rarely taking biological risk factors into account.

Epigenetics and Gene–Environment Interactions

The excitement created by advances in genomics has raised the possibility of defining all mental disorders on the basis of specific genetic variations. Unfortunately, this possibility is an illusion. Loose talk about "genes for schizophrenia" or "genes for bipolar disorder" are misleading because there is no such thing as a gene for any disease. (Genes make proteins, not illnesses.)

Moreover, genetic influences on disease generally involve "complex inheritance" (Morton, 2001). Inherited variations affect phenotypes through combinations of alleles at multiple loci. Only a few illnesses follow a Mendelian pattern, in which a single allele is responsible for a disease. The more typical pattern is for many genes, interacting in different proportions, to produce interactive genetic vulnerability. This explains why, even when genes have been found to be associated with mental disorder, they almost always explain only a small percentage of the variance (Kendler, 2006).

It has also become apparent that reading the genome, by itself, can provide little insight into how individual genes work. Each of the 20,000 or so human genes has one or several functions in building proteins or in regulating the action of other genes. The task of determining their precise action has led to the development of a new discipline called "proteomics" (Tyers & Mann, 2003). But even if we knew how every protein in the body is made (and folded), we might not be able to proceed from that knowledge to an understanding of psychopathology.

Finally, research shows that genes can be either active or inactive. This should not be surprising, since every cell carries the entire genome, yet only a few genes are expressed. New data show that any gene can be silenced or activated by chemical processes (attaching methyl or histone groups) that are in turn influenced by the environment, and these processes can even be transmitted to the next generation through a quasi-Lamarckian mechanism (Meaney & Szyf, 2005).

Thus in the new scientific domain of epigenetics (or epigenomics), environmental factors are responsible for turning genes "on" and "off." This model provides a more precise mechanism for gene–environment interactions. A large body of research confirms the central importance of these interactions. For example, a large-scale study of monozygotic twins showed that epigenetic differences, accumulating over time, can produce striking differences in health outcomes (Fraga et al., 2005).

Behavior genetics, usually based on studies of twin samples, shows that genes account for no more than half of the variance in most mental disorders, with the environment playing an equally important role (Plomin, DeFries, McClearn, & Rutter, 2001). Longitudinal research confirms interactions between these factors. Well-known studies in a prospectively followed community cohort by Caspi and colleagues (2002, 2003) suggested the possibility that only a combination of genetic vulnerability and adverse life events can account for the risk for such common problems as depression and antisocial behavior.

The BPS Model in Clinical Practice

The division between biological and psychosocial models greatly afflicts clinical practice. It has become rare for patients not to receive medication of some kind for their symptoms. Yet in most forms of depression, large-scale studies have shown that psychotherapy is as effective as drugs (Elkin, Shea, Watkins, & Imber, 1989), and that antidepressants are only somewhat better than placebo (Moncrieff, Wessely, & Hardy, 2004). Yet patients are receiving too many prescriptions and are not being prescribed effective evidence-based psychotherapies.

The fact is that psychopharmacology, as presently practiced, remains an empirical science producing hit-and-miss results. The identification of "endophenotypes" (Gottesman & Gould, 2003), the biological processes that underlie observable illness, could provide more precise targets for pharmacological treatment. But without an understanding of how diatheses interact with stressors, even that approach would only define the correlates of disorder, as opposed to basic mechanisms that could predict outcome.

Similarly, psychosocial research continues on its own track, correlating adversities with outcomes without considering biologi-

cal vulnerability. For example, developmental psychology is surprisingly uninformed about genetics and biology (Harris, 1998). Here too, adopting the BPS model could be a useful antidote to mindless reductionism of all kinds (Eisenberg, 1986).

Is the BPS model really used in practice, or does it only earn lip service? Psychiatrists and psychologists may claim to take a BPS approach, but would their patients agree? In an era dominated by neuroscience and psychopharmacology, reductionism is the ruling paradigm. One recent study found that psychiatrists still commonly embrace a mind–body dualism (Miresco & Kirmayer, 2006). For many of its practitioners, drug prescriptions follow directly from clinical diagnosis.

The BPS Model and Major Mental Disorders

The BPS model can be applied to any mental disorder. But let us focus on four areas where the paradigm is of particular clinical importance: schizophrenia, depression, substance use disorders, and personality disorders.

Schizophrenia

Kraepelin (1919) thought of schizophrenia as an organic illness of unknown cause. But the mystery of its origins has not been solved. Over the years, the problem stimulated much unwarranted speculation. Some psychoanalysts attributed schizophrenia to bad mothering and tried to cure it with psychotherapy (Dolnick, 1998). That idea has died out, and today, with the advent of modern genetic and biological research (and with the success of neuroleptic drugs), the disorder has once again been seen as almost entirely biological in origin.

Yet several lines of evidence suggest that a broader model is necessary to account fully for how schizophrenia develops. Only 50% of monozygotic twins are concordant for the disorder (Gottesman, 1991). This fact demonstrates that environmental factors determine whether thresholds are crossed. Some could be intrauterine, while others might only have an impact at later periods of development.

There is also evidence for social influences on the etiology of schizophrenia. Research shows that the prevalence of the disorder is high among West Indian immigrants to England, as well as among Moroccan immigrants to Holland, but not among comparable groups remaining in their countries of origin (Cantor-Graae, 2007). These data suggest that the social context of immigration can trigger schizophrenia in susceptible individuals. Finally, recent data from large-scale trials of antipsychotic medication not only emphasize the limitations of these agents, but provide evidence that psychotherapy plays a useful role (Turkington, Kingdon, & Weiden, 2006). Although effective treatment does not prove that psychosocial factors are etiological, it still supports a BPS approach to schizophrenia (Meyer, 2007).

Depression

Depression has come to be thought of as a primarily biological disorder. This is a dramatic change from the recent past, when it was seen as a pathological variant of grief. There is no doubt that depression runs in families and is influenced by genetic vulnerability. But there is also robust evidence that common episodes of major depression are triggered by adverse life events (Kendler et al., 1995). Moreover, not all forms of depression reflect the same illness process (Parker, 2005). Melancholia may be a separate disease, with a stronger and more specific genetic/biological contribution. This concept challenges the long-held belief that all depressions lie on a continuum of severity.

The "garden-variety" depressions that have high community prevalence and are common in practice may develop from a different mix of risk factors (Parker, 2005). They may also not respond to the same treatment, as shown by the sobering findings of a recent large-scale study of the effectiveness of antidepressants (Rubinow, 2006).

There is also strong evidence that social factors affect the prevalence of depression (Horwath, Cohen, & Weissman, 2000). These conditions vary in prevalence in different social settings, and have shown a dramatic increase in prevalence over the last several decades.

For all these reasons, the BPS model provides the best account of the etiology and pathogenesis of depression (Garcia-Toro & Aguirre, 2007). In fact, major depression is best understood as reflecting the emergence of symptoms in vulnerable individuals after

exposure to adverse life events (Kendler et al., 1995). These findings have important clinical implications. There is little evidence that depression is a chemical imbalance that can be routinely treated with antidepressants. Multiple treatment interventions are needed to address all its components.

Substance Use Disorders

Substance use disorders are affected by biological, psychological, and social factors. Although no specific biological factors have been identified, alcoholism and drug addictions have been shown to be associated with genetic vulnerability (Galanter & Kleber, 2004). These problems run in families, and the risk is particularly high for children whose substance misuse starts early in life (Schuckit & Smith, 1995).

But another large body of evidence shows that social factors play a crucial role in the development of substance use disorders (Westermeyer, 1991). For example, prevalence can vary from ubiquity to rarity, and is markedly different in different cultures (Helzer & Canino, 1992). Moreover, the prevalence of substance misuse has greatly increased in Western societies over the last few decades (Rutter & Smith, 1995).

Misuse of drugs and alcohol can be best understood within a BPS model. The symptomatic pattern develops in individuals with trait vulnerabilities who are also exposed both to adverse life events and to a social environment that reinforces and permits the use of substances. The model has many advantages for treating this important population of patients, who almost always need psychosocial interventions to recover (Marlatt, 1992).

Personality Disorders

As their name indicates, the personality disorders are characterized by dysfunction in personality—affecting work and relationships, beginning early in life, independent of context, and continuing over years. They are highly prevalent in clinical practice (Zimmerman, Rothschild, & Chelminski, 2005).

In the past, personality disorders were considered almost entirely psychogenic. However, personality has a large genetic component (Plomin et al., 2001), and personality disorders are equally heritable (Torgersen et al., 2000). Again, the best model is one in which trait vulnerabilities are activated by adverse psychosocial circumstances.

Most research has been conducted on the antisocial and borderline categories. A BPS model can be applied to antisocial personality disorder (Paris, 1996)—either the broader concept defined in DSM, or the more narrowly defined entity of psychopathy (Harpur, Hart, & Hare, 1994; Paris, 1998). The model conceptualizes a genetic vulnerability arising early in life that can be amplified by psychosocial stressors, particularly dysfunctional families and a pathological social environment. Pathology presents itself early in life as conduct disorder, and continues into adulthood as a personality disorder. The failure of most treatments for this condition suggests that an important biological component could be present, even if it is not yet understood. But patients with this disorder almost always come from highly dysfunctional families (Robins, 1966).

Similarly, borderline personality disorder can be conceptualized as emerging from interactions between underlying traits (affective instability and impulsivity) and the impact of adverse or traumatic life events (Paris, 1994). This is a disorder that typically begins in adolescence, and tends to continue over the young adult years (Paris, 2003). Its treatment requires a combination of biological, psychological, and social interventions (Paris, 2005).

The Decline and Fall of the BPS Model

The goal of the BPS model has always been to encourage clinicians to conduct a broadly based practice. If there are biological risk factors in illness, they may be contained by drugs. If there are psychological risk factors, they can be addressed by psychotherapy. If there are social risk factors, treatment could involve some form of environmental intervention.

But theoretical models do not always drive clinical practice. Economic and ideological factors tend to be more important. Psychiatry has become dominated by biological treatment, with fewer of its practitioners trained to conduct psychosocial interventions (Paris, 2008). Moreover, divisions in mental health care are reinforced by a

structure in which psychologists offer most psychotherapy while physicians do most of the prescribing. The BPS model of care that Engel proposed for psychiatry has been largely pushed aside. Nor is there is reason to believe that BPS models have had a great influence on the practice of internal medicine—the area on which Engel first focused his attention. But even if the BPS approach is an ideal, it describes more or less what most patients need (Gabbard & Kay, 2001).

The BPS Model and DSM-V

A BPS model, however comprehensive, would not easily be applied to problems of classification. In some ways, it could make problems even more difficult.

Medicine has been able to use a knowledge of etiology and pathogenesis to classify some of its syndromes. For example, we no longer consider jaundice a disease, but a symptom that can reflect liver failure, blood diseases, or other inflammatory and/or immunological defects.

The etiological pathways that lead to mental disorders are complex, as are the human brain and mind. Interactions between multiple variables would not be readily captured in categories. Medical specialists do not routinely classify diseases like hypertension or cancer on the basis of etiology; there are just too many risk factors involved to make doing so practicable. These complex diseases are still generally classified in terms of their signs and symptoms.

Given our continued lack of knowledge about the causes of mental disorders, DSM-V should focus on developing criteria for its categories that would at least have discriminant validity. Valid categories would provide a better basis for research into the biological, psychological, and social risk factors for every mental disorder. And those kinds of data might allow us to develop a BPS model that accounts better for psychopathology.

References

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

Bertalanffy, L., von. (1975). *Perspectives on general system theory* (E. Taschdjian, Ed.). New York: Braziller.

Cantor-Graae, E. (2007). The contribution of social factors to the development of schizophrenia: A review of recent findings. *Canadian Journal of Psychiatry*, *52*(5), 277–286.

Caspi, A., McClay, J., Moffitt, T. E., Mill, J., Martin, J., Craig, I. W., et al. (2002). Role of genotype in the cycle of violence in maltreated children. *Science*, *297*, 851–854.

Caspi, A., Sugden, K., Moffitt, T. E., Taylor, A., Craig, I. W., Harrington, H., et al. (2003). Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. *Science*, *301*, 386–389.

Cicchetti, D., & Rogosch, F. A. (1996). Equifinality and multifinality in developmental psychopathology. *Development and Psychopathology*, *8*, 597–600.

Dolnick, E. (1998). *Madness on the couch*. New York: Simon and Schuster.

Eisenberg, L. (1986). Mindlessness and brainlessness in psychiatry. *British Journal of Psychiatry*, *148*, 497–508.

Eisenberg, L. (2004). Social psychiatry and the human genome: Contextualising heritability. *British Journal of Psychiatry*, *184*, 101–103.

Elkin, I., Shea, T., Watkins, J. T., & Imber, S. D. (1989). National Institute of Mental Health Treatment of Depression Collaborative Research Program: General effectiveness of treatments. *Archives of General Psychiatry*, *46*, 971–982.

Engel, G. L. (1977). The need for a new medical model. *Science*, *196*, 129–136.

Engel, G. L. (1980). The clinical application of the biopsychosocial model. *American Journal of Psychiatry*, *137*, 535–544.

Engel, G. L. (1997). From biomedical to biopsychosocial: Being scientific in the human domain. *Psychosomatics*, *38*, 521–528.

Fraga, M. F., Ballestar, E., Paz, M. F., Ropero, S., Setien, F., Ballestar, M. L., et al. (2005). Epigenetic differences arise during the lifetime of monozygotic twins. *Proceedings of the National Academy of Sciences USA*, *102*, 10604–10609.

Gabbard, G. O., & Kay, J. (2001). The fate of integrated treatment: Whatever happened to the biopsychosocial psychiatrist? *American Journal of Psychiatry*, *158*, 1956–1963.

Galanter, M., & Kleber, H. (Eds.). (2004). *The American Psychiatric Publishing textbook of substance abuse treatment* (3rd ed.). Washington, DC: American Psychiatric Publishing.

Garcia-Toro, M., & Aguirre, I. (2007). The biopsychosocial model in depression revisited. *Medical Hypotheses*, *68*, 683–691.

Ghaemi, S. N. (2006). Paradigms of psychiatry: Eclecticism and its discontents. *Current Opinion in Psychiatry*, *19*, 619–624.

Gottesman, I. I. (1991). *Schizophrenia genesis*. New York: Freeman.

Gottesman, I. I., & Gould, T. D. (2003). The endophenotype concept in psychiatry: Etymology and strategic intentions. *American Journal of Psychiatry*, *160*, 636–645.

Grinker, R. R., Sr. (1969). Emerging concepts of mental illness and models of treatment: The medical point of view. *American Journal of Psychiatry*, *125*, 865–869.

Harpur, T. J., Hart, S. D., & Hare, R. D. (1994). Personality of the psychopath. In P. T. Costa & T. A. Widiger (Eds.), *Personality disorders and the five-factor model* (pp. 149–174). Washington, DC: American Psychological Association.

Harris, J. R. (1998). *The nurture assumption*. New York: Free Press.

Helzer, J. E., & Canino, G. J. (Eds.). (1992). *Alcoholism in North America, Europe, and Asia*. New York: Oxford University Press.

Hollingshead, A., & Redlich, F. (1958). *Social class and mental illness*. New York: Wiley.

Horwath, E., Cohen, R. S., & Weissman, M. M. (2000). Epidemiology of depressive and anxiety disorders. In M. T. Tsuang (Ed.), *Textbook of psychiatric epidemiology* (pp. 389–426). New York: Wiley-Liss.

Insel, T., & Quirion, R. (2005). Psychiatry as a clinical neuroscience discipline. *Journal of the American Medical Association*, *294*, 2221–2224.

Jaspers, K. (1997). *General psychopathology* (2 vols.). Baltimore: Johns Hopkins University Press. (Original work published 1913)

Kendler, K. S. (2006). Reflections on the relationship between psychiatric genetics and psychiatric nosology. *American Journal of Psychiatry*, *163*, 1138–1146.

Kendler, K. S., Walters, E. E., Neale, M., Kessler, R. C., Heath, A. C., & Eaves, L. J. (1995). The structure of the genetic and environmental risk factors for six major psychiatric disorders in women. *Archives of General Psychiatry*, *52*, 374–383.

Kraepelin, E. R. (1919). *Dementia praecox and paraphrenia*. Edinburgh, UK: Livingstone.

Kuhn, T. A. (1970). *The structure of scientific revolutions* (2nd ed.). Chicago: University of Chicago Press.

Marlatt, G. A. (1992). Substance abuse: Implications of a biopsychosocial model for prevention, treatment, and relapse prevention. In J. Grabowski & G. R. VandenBos (Eds.), *Psychopharmacology: Basic mechanisms and applied interventions* (pp. 131–162). Washington, DC: American Psychological Association.

McClain, T., O'Sullivan, P. S., & Clardy, J. A. (2004). Biopsychosocial formulation: Recognizing educational shortcomings. *Academic Psychiatry*, *28*, 88–94.

McLaren, N. (1998). A critical review of the biopsychosocial model. *Australian and New Zealand Journal of Psychiatry*, *32*, 86–92.

Meaney, M. J., & Szyf, M. (2005). Maternal care as a model for experience-dependent chromatin plasticity? *Trends in Neurosciences*, *28*, 456–463.

Meyer, A. (1952). *The collected papers of Adolf Meyer*. Baltimore: John Hopkins University Press.

Meyer, J. M. (2007). Strategies for the long-term treatment of schizophrenia: Real-world lessons from the CATIE trial. *Journal of Clinical Psychiatry*, *68*(Suppl. 1), 28–33.

Miresco, M. J., & Kirmayer, L. J. (2006). The persistence of mind–brain dualism in psychiatric reasoning about clinical scenarios. *American Journal of Psychiatry*, *163*, 913–916.

Moncrieff, J., Wessely, S., & Hardy, R. (2004). Active placebos versus antidepressants for depression. *Cochrane Database of Systematic Reviews*, Issue 1 (Article No. CD003012). DOI: 10.1002/14651858.CD003012.pub2.

Monroe, S. M., & Simons, A. D. (1991). Diathesis–stress theories in the context of life stress research. *Psychological Bulletin*, *110*, 406–425.

Morton, N. E. (2001). Complex inheritance: The 21st century. *Advances in Genetics*, *42*, 535–543.

Paris, J. (1994). The etiology of borderline personality disorder: A biopsychosocial model. *Psychiatry*, *57*, 300–307.

Paris, J. (1996). Antisocial personality disorder: A biopsychosocial model. *Canadian Journal of Psychiatry*, *41*, 75–80.

Paris, J. (1998). Psychopathy: A biopsychosocial model. In T. Millon, E. Simonsen, M. Birket-Smith, & R. D. Davis (Eds.), *Psychopathy* (pp. 277–290). New York: Guilford Press.

Paris, J. (1999). *Nature and nurture in psychiatry*. Washington, DC: American Psychiatric Press.

Paris, J. (2003). *Personality disorders over time*. Washington, Dc: American Psychiatric Press.

Paris, J. (2005). Borderline personality disorder. *Canadian Medical Association Journal*, *172*, 1579–1583.

Paris, J. (2008). *Prescriptions for the mind*. New York: Oxford University Press.

Parker, G. (2005). Beyond major depression. *Psychological Medicine*, *35*, 467–474.

Plomin, R., DeFries, J. C., McClearn, G. E., & Rutter, M. (2001). *Behavioral genetics* (4th ed.). New York: Freeman.

Richter, D. (1999). Chronic mental illness and the limits of the biopsychosocial model. *Medicine, Health Care and Philosophy*, *2*, 21–30.

Robins, L. N. (1966). *Deviant children grown up*. Baltimore: Williams & Wilkins.

Rubinow, D. R. (2006). Treatment strategies after SSRI failure: Good news and bad news. *New England Journal of Medicine*, *354*, 1305–1307.

Rutter, M., & Smith, D. J. (1995). *Psychosocial factors in young people: Time trends and their causes*. New York: Wiley.

Schuckit, M. A., & Smith, T. L. (1996). An 8 year follow-up of 450 sons of alcoholic and control subjects. *Archives of General Psychiatry*, *53*, 202–210.

Shorter, E. (1997). *A history of psychiatry*. New York: Wiley.

Torgersen, S., Lygren, S., Oien, P. A., Skre, I., Onstad, S., Edvardsen, J., et al. (2000). A twin study of personality disorders. *Comprehensive Psychiatry*, *41*, 416–425.

Turkington, D., Kingdon, D., & Weiden, P. J. (2006). Cognitive behavior therapy for schizophrenia. *American Journal of Psychiatry*, *163*, 365–373.

Tyers, M., & Mann, M. (2003). From genomics to proteomics. *Nature*, *22*, 193–197.

Westermeyer, J. (1991). Historical and social context of psychoactive substance disorders. In R. J. Frances & S. I. Miller (Eds.), *Clinical textbook of addictive disorders* (pp. 23–42). New York: Guilford Press.

Zimmerman, M., Rothschild, L., & Chelminski, I. (2005). The prevalence of DSM-IV personality disorders in psychiatric outpatients. *American Journal of Psychiatry*, *162*, 1911–1918.

CHAPTER 25

Reactivating the Psychodynamic Approach to the Classification of Psychopathology

SIDNEY J. BLATT
PATRICK LUYTEN

The present volume amply demonstrates that we are on the brink of a new era in the classification of psychopathology. Current approaches to classification are increasingly moving away from a descriptive categorical system toward a dimensionally organized diagnostic system; this move has major etiological as well as therapeutic implications.

In this chapter, we discuss psychoanalytic contributions to this trend. First, we discuss the limitations of the current DSM approach from a psychodynamic perspective. Second, we present a psychodynamically inspired view of psychiatric classification that uses personality theory to articulate how various forms of psychopathology can be seen as evolving out of disruptions of normal personality development. We compare this view with other current hierarchical and dimensional models of psychopathology, and we show how such a dialogue may lead to more encompassing models of psychopathology by focusing on clinically meaningful underlying principles of psychopathology, based on theories of normal and disrupted personality development.

Psychoanalysis and Classification: A Historical Perspective

Historically, two approaches to the classification of psychopathology have been in competition. The approach espoused more often in psychiatry emphasizes description and favors a categorical view of psychiatric disorders; it is more static and is mainly concerned with the search for the relatively unique (biological) etiopathogenesis of particular psychiatric disorders. The approach more popular in psychology (and particularly in psychoanalysis) is more theory-driven and dimensionally oriented; it emphasizes life history and personality dynamics, conceptualizing psychopathology as evolving out of the manifold interactions between biological endowment and responses to both the external environment and intrapsychic reality. Whereas the former approach is more disorder-centered, the latter is more person-oriented (Luyten, Viegen, Van Houdenhove, & Blatt, 2008). The first approach gained popularity through the work of Kraepelin (1904/1907), and the second through the

work of Freud (e.g., 1917/1963) and Meyer (1951–1952), but obviously both approaches have historical roots that go far beyond these three giants of modern psychiatry (Ellenberger, 1970).

In many respects, these two approaches are complementary and reflect many difficulties in categorizing psychiatric disorders, as psychiatric nosology constantly shifts between these two approaches. Furthermore, the Freud–Meyer approach led not only to insights into the dynamics of disorders and their interrelationships, but also to the delineation and description of such disorders as obsessive–compulsive (Freud, 1895/1957a) and depression (Freud, 1917/1957b) and borderline personality (Knight, 1940) disorders. Research within the Kraepelinian tradition, in turn, led to the identification of important psychosocial determinants of several disorders, such as the role of early trauma and life stress in depressive disorders (Hammen, 2005), again testifying to the fact that these two approaches are not mutually exclusive. Historically, this has led psychiatry to adopt a hybrid descriptive–dynamic point of view in categorizing and treating disorders—emphasizing the importance of the description of separate disorders, as well as the importance of life history and personality dynamics.

Although earlier editions of the DSM embraced such a hybrid approach, the dynamic, dimensional, and etiological points of view emphasizing life history and dynamics were dropped with the introduction of DSM-III (American Psychiatric Association [APA], 1980) in favor, with some important exceptions, of a purely descriptive, categorical, and atheoretical approach. In retrospect, this change made perfect sense because it reduced diagnostic confusion by providing a common language for the psychiatric community. Furthermore, this approach made broad-ranging and systematic research on psychiatric disorders possible, with the aim of facilitating the development of a new and more etiologically based classification system. In addition, at the time when DSM-II was introduced, psychosocial research often lacked methodological rigor and the neurosciences were only in their infancy, limiting the development of an etiologically based classification system (Luyten & Blatt, 2007). Theoretical, empirical, and methodological developments in both psychosocial research and the neurosciences, however, are now increasingly leading researchers to believe that a more etiologically based diagnostic system of psychiatric disorders is possible (e.g., Blatt, 2004, 2008; Blatt & Levy, 1998; Clark, 2005; Luyten & Blatt, 2007; McHugh, 2005; Parker, 2005; Watson, 2005). This belief has led to renewed interest in combining the descriptive/categorical and etiological/dimensional approaches (Westen, Shedler, & Bradley, 2006).

Thus one of the most important tasks psychiatry faces at present is to come to terms with the complexity of mental disorders, in terms of both etiopathogenesis and categorization. As this volume amply demonstrates, various competing theoretical models are currently being investigated. In this chapter, we describe a psychodynamic developmental psychopathology approach built on a broad and integrative theory of normal and pathological personality development. In brief, the proposed "two-configurations" model argues that the two fundamental psychological dimensions of relatedness and self-definition, or attachment and separation, provide a theoretical matrix for understanding processes of personality development, variations in normal personality organization, concepts of psychopathology, and mechanisms of therapeutic action. The continuity of these two primary dimensions in personality development and in psychopathology provides the basis for considering forms of psychopathology as distorted modes of adaptation that derive from variations and disruptions of normal psychological development.

In this two-configurations model, personality plays a central role. It is indeed our central assumption that personality is an important unifying concept in both the categorization and treatment of psychiatric disorders—linking normal and pathological development, as well as connecting research in developmental psychopathology with current neuroscientific investigations. Before introducing this approach, however, we first discuss limitations of the current DSM system from a psychodynamic perspective, and then contrast this approach with the proposed two-polarities model.

Limitations of DSM and Implications for Future Classification Systems

Like any classification system, DSM-III and its successors have been based on a number of explicit and implicit assumptions. Although some of these assumptions have often been incorrectly attributed to DSM, they have nevertheless had an important influence on research and clinical practice (Cuthbert, 2005). We discuss each of these assumptions and their rationale, followed by a discussion of relevant empirical findings.

1. The most central assumption of DSM-III and its successors is that clinical disorders are categorically distinct from subclinical disorders and normal functioning (APA, 1994, p. xxi), and that, with some exceptions, disorders can and should be diagnosed on the basis of symptoms alone (APA, 1994, pp. xvii–xviii). A major advantage of this approach for both research and clinical practice is that it implies a clear demarcation between cases and noncases; this approach is congruent with the preferences of clinicians, insurance companies, and the general public (APA, 1994; First, 2005). An additional advantage is that the emphasis on symptoms should lead to greater diagnostic reliability than a categorization system based on purported etiopathogenetic factors or considerations involving life history or personality dynamics (Blatt & Levy, 1998; Westen, Heim, Morrison, Patterson, & Campbell, 2002).

2. A second assumption is that Axis I and Axis II disorders are independent (Westen et al., 2002). Although this assumption may be incorrectly attributed to the designers of DSM, it has nevertheless influenced research, as various rationales have been suggested for this assumption. These include the suggestions (a) that Axis I disorders are more ego-dystonic and state-dependent, while personality disorders are presumed to be more ego-syntonic and trait-like; and (b) that biological factors may play a greater role in Axis I disorders, while personality disorders may be more psychosocial in origin. Such suggestions have led, somewhat paradoxically, to a burgeoning literature on comorbidity between Axis I and II disorders, but to surprisingly little research that has investigated possible causal relationships between disorders on both axes (Clark, 2005).

3. Finally, most research concerning psychiatric disorders based on DSM-III and its successors has been guided by the assumption that disorders have relatively distinct etiologies. Importantly, this has also led to attempts to develop specific treatments for each of these disorders (Frances, First, & Pincus, 1995; Kupfer, First, & Regier, 2002). Although, again, this may not have been the original intent of DSM's designers, Cuthbert (2005) has convincingly argued that this assumption has led to extensive research on the etiopathogenesis of specific disorders, to the neglect of research concerning common etiological pathways to various disorders from infancy to childhood (see also Luyten et al., 2008). As noted, part of the rationale for trying to identify distinct vulnerability factors may have been related to the hope that this would enable the development of specific treatments for each disorder (Kupfer et al., 2002). Whatever its inspiration may have been, it is hard to deny that most treatment studies and treatment guidelines tend to focus on the efficacy and effectiveness of disorder-specific treatments, and not on the effects of specific treatments for broader classes of disorders (Westen et al., 2002) or on the identification of treatment processes that may explain treatment outcome across a variety of disorders and different treatment modalities (Zuroff & Blatt, 2006). This focus on specific disorders and disorder-specific treatments may also partly result from the fact that funding has been easier to obtain for research on a specific DSM-defined disorder than for research on common etiological factors underlying several disorders (Cuthbert, 2005). Because of this "hegemony of the DSM categorical system" (First, 2005, p. 562), it is thus hardly surprising that most research has concentrated on the etiology and treatment of specific disorders, rather than focusing on shared etiological factors and the efficacy and effectiveness of treatments for a range of disorders (Cuthbert, 2005; van Praag, de Kloet, & van Os, 2004).

Research over the last decades has identified problems that have derived from each of

these assumptions (e.g., Blatt & Levy, 1998; Cuthbert, 2005; Luyten, Blatt, Van Houdenhove, & Corveleyn, 2006; Parker, 2005;Widiger & Samuel, 2005).

First, concerning the categorical approach, studies suggest that most disorders—including even severe psychiatric disorders, such as psychotic and bipolar disorders (Haslam, 2003; Ruscio & Ruscio, 2000; Tsuang, Stone, Tarbox, & Faraone, 2003)—are best understood as situated on a continuum from normality to subclinical pathology to manifest clinical disorders (Blatt, 1974; Haslam, 2003; Kendler & Gardner, 1998; Ruscio & Ruscio, 2000; Solomon, Haaga, & Arnow, 2001). This does not necessarily imply, however, that a dimensional approach should be adopted in clinical practice, or that there are no discrete categories or "taxa" (e.g., melancholic depression) (Parker, 2000). It does imply, however, that the current arbitrary, consensus-based cutoff criteria that define disorders are in need of reevaluation (Brown & Barlow, 2005; First, 2005). In this context, Widiger and Clark (2000, p. 949) have correctly pointed out that "The failure to conduct pilot studies of a criterion set is uncomfortably comparable to releasing a psychological test for publication in the absence of validation data." Like any psychological test, the psychometric properties of these cutoff criteria need to be investigated. For instance, specifiers such as "mild," "moderate," and "severe" may be used in the future to define meaningful (as opposed to consensus-based) cutoffs, and their validity could be investigated by studying their relationship to etiopathogenetic factors, as well as to clinical course and treatment response (Parker, 2005; Widiger & Clark, 2000). It is even possible that these cutoff criteria could be different for different purposes (e.g., for research vs. health insurance). Such studies could also shed more light on the issue of comorbidity. For instance, about 75% of patients with dysthymic disorder have a lifetime history of major depression (Keller et al., 1995), which makes it unreasonable to assume that individuals with dysthymic disorder also periodically suffer from another disorder (i.e., major depression). Rather, they seem to suffer from chronic and more episodic manifestations of the same disorder, much as someone with diabetes may have a periodic increase in symptoms (Widiger & Clark, 2000).

Likewise, the almost exclusive reliance on objective symptoms in the most recent editions of DSM may have resulted in poor validity (Blatt & Levy, 1998; Westen et al., 2006; Widiger & Samuel, 2005). Studies have amply demonstrated that patients with the same DSM diagnosis are often heterogeneous in terms of etiology and pathogenesis, which has hampered research on the identification of etiopathogenetic factors (Nemeroff et al., 2003; van Praag et al., 2004). Furthermore, this "count/cutoff" approach does not match the way clinicians intuitively think about patients and make diagnoses, which may explain why clinicians rarely follow the diagnostic procedures prescribed by DSM (Westen et al., 2006). Importantly, this has limited the clinical utility of DSM (First et al., 2004).

Together, these findings raise serious concerns about the current descriptive, symptom-based approach to mental disorders (Charney et al., 2002; Kupfer et al., 2002; McHugh, 2005). In particular, the high comorbidity between many disorders suggests the need to regroup DSM categories on the basis of common etiological and pathogenetic factors (see Widiger & Samuel, 2005, for an overview). The studies showing high rates of comorbidity among disorders have undermined "the hypothesis that the syndromes represent distinct etiologies" (Kupfer et al., 2002, p. xviii). In response to these findings, researchers are now increasingly moving toward the idea of spectra of disorders—such as an obsessive–compulsive spectrum (including obsessive–compulsive disorder, Tourette's and other tic disorders, hypochondriasis, trichotillomania, and body dysmorphic disorder); a spectrum of stress-related disorders (including posttraumatic stress disorder [PTSD] and acute stress disorder); and a spectrum of affective disorders (Hudson, Arnold, Keck, Auchenbach, & Pope, 2004; Raphael, Janal, Nayak, Schwartz, & Gallagher, 2004)—based on considerations concerning common etiological factors (Phillips, First, & Pincus, 2003). Another avenue has been to develop hierarchical models of mental disorders, with disorders in a particular cluster presumably sharing etiopathogenetic factors (Watson, 2005).

Second, regarding the assumption of orthogonality between Axis I and Axis II, studies have clearly shown that most Axis I disorders are not independent of personality disorders or of personality more generally. Evidence is accruing that temperament (e.g., Clark, 2005) and personality dimensions—both broad dimensions (e.g., neuroticism) (Kendler, Kuhn, & Prescott, 2004; Ormel, Oldehinkel, & Brilman, 2001) and more specific ones (e.g., interpersonal dependency and perfectionism) (Blatt, 2004; Cox, McWilliams, Enns, & Clara, 2004; Zuroff, Mongrain, & Santor, 2004)—are involved in the etiopathogenesis of many disorders. This may explain the high rates of comorbidity between specific clusters of Axis I and Axis II disorders (Westen, Novotny, & Thompson-Brenner, 2004). For example, personality factors such as neuroticism and self-critical perfectionism have been implicated not only in depression (Blatt, 2004; Kendler et al., 2004), but also in eating disorders (Westen & Harnden-Fisher, 2001), opiate addiction (Blatt, McDonald, Sugarman, & Wilber, 1984), cardiovascular and immunological disease (Blatt, Cornell & Eshkol, 1993), bipolar disorders (Lam, Wright, & Smith, 2004), and several anxiety disorders (Shafran & Mansell, 2001). In addition, increasing evidence suggests the role of temperamental dimensions, such as positive and negative affect and disinhibition, in a variety of mental disorders (Weinstock & Whisman, 2006). Recent research even suggests that causal relationships between Axis I and Axis II disorders may develop over time (Clark, 2005; Westen et al., 2004). These findings are also congruent with studies on gene–environment correlations, which strongly suggest that vulnerable individuals in part create their own (stressful) environments (Moffitt, Caspi, & Rutter, 2005).

Hence it is time to reconsider the distinction between Axis I and II, which may also have important implications for treatment. For instance, many patients with depressive disorders have comorbid borderline personality disorder; they may thus be more likely to benefit from treatments developed for personality disorders, such as dialectical behavior therapy (Linehan, 1993), mentalization-based treatment (Bateman & Fonagy, 2004), or transference-focused psychotherapy (Levy et al., 2006), than from more narrowly depression-focused treatments. Thus, in short, changing our perspective on the relationship between Axis I and Axis II may lead to considerable change in the way we think about treatment issues.

Finally, and in line with the previous set of findings, research has consistently shown that comorbidity between Axis I disorders (e.g., between mood and anxiety disorders) is the rule rather than the exception; this appears to contradict the assumption that Axis I disorders are relatively distinct from one another (Luyten, Blatt, & Corveleyn, 2005c; Nemeroff, 2002; Parker, 2005). Researchers have thus begun to consider the validity of mixed categories, such as a depressed–anxious disorder (e.g., Phillips et al., 2003), as well as to formulate more encompassing and often hierarchical models of psychopathology (Watson, 2005). For instance, studies have shown that both clinical and subclinical depressive disorders share many psychosocial (e.g., Ormel et al., 2001) and biological (Heim, Plotsky, & Nemeroff, 2005) etiological factors with other disorders, indicating that earlier research may have been overly concerned with identifying the "unique" etiological factors implied in depression and other disorders (van Praag et al., 2004) instead of looking for common mechanisms across different disorders (Nemeroff et al., 2003). The fact that various pharmacological agents are often equally effective in treating depression and anxiety disorders points in the same direction (Parker, 2005), and may in part explain why DSM diagnoses have little predictive power in regard to treatment response (Kupfer et al., 2002).

Taken together, these findings support the view that various DSM assumptions are unlikely to be accurate and productive (Krueger, Markon, Patrick, Benning, & Kramer, 2007). Hence a clear shift is occurring from the classification and treatment of specific forms of psychopathology toward the use of hierarchical and dimensional models. Yet it is our central contention that to be clinically meaningful, such models also need to be firmly rooted in developmental psychopathology; researchers need to appreciate the importance of equifinality and multifinality in developmental pathways toward clusters of disorders (Cicchetti & Rogosch, 1996; Luyten et al., 2008). "Equifinality" is the

assumption that there are several possible pathways toward one psychiatric disorder, rather than one distinct etiological pathway for each disorder. "Multifinality," in turn, is the view that the same etiological factors may result in a variety of disorders, depending on their interaction with other factors. Hence patients who present with similar symptoms may have very different etiological backgrounds, which is congruent with the findings that most DSM diagnoses represent etiologically heterogeneous categories (Luyten & Blatt, 2007; Parker, 2005). Likewise, patients who have similar etiological backgrounds, depending on other factors, may express their problems in different ways.

This shift from a focus on the study of the etiology of specific disorders to studying etiological pathways involved in different disorders has important implications for future research. As Cuthbert (2005, p. 567) has argued, "given the increasing realization that most DSM diagnoses do not represent homogeneous categories, reliable genetic associations or biomarkers are much more likely to be established when better definitions and delineation of disorders are achieved." The same is probably true for psychosocial factors as well. We return to this issue further below. In addition, a focus on equifinality and multifinality of developmental pathways instead of disorders implies a shift in research from disorder- and variable-centered approaches to person-centered approaches (Cicchetti & Rogosch, 1996). This may also enhance the clinical utility of a classification system and facilitate the translation of research findings to clinical practice (Luyten et al., 2008). Variable-centered studies often convey little relevant information for clinicians because there is always a gap between nomothetic findings based on group studies and the idiographic level (i.e., the individual patient; Luyten, Blatt, & Corveleyn, 2006). Person-oriented studies, in contrast, focus on identifying pathways from early childhood to later adulthood in clusters of individuals. This approach may be of more interest to clinicians because this may allow them to assess the extent to which an individual patient matches a particular cluster of individuals. Indeed, this approach fits with the prototype approach that clinicians tend to use in clinical practice, rather than the count/cutoff approach of the current DSM system (Ablon & Jones, 2005; Westen et al., 2006). Moreover, this implies that assessment should not be exclusively aimed at diagnosing a particular disorder, but should also include a developmental assessment of each patient's underlying vulnerabilities and strengths, which should then inform treatment (Luyten, Blatt, & Corveleyn, 2005a).

Reactivating the Psychodynamic Approach to Diagnosis: Basic Principles

The absence of an objective standard is a major impediment to the development of any classification of mental disorders. Indeed, there is no known set of "naturally occurring categories" against which to validate a classification system (Westen & Shedler, 2000). One approach to this issue is empirical—that is, delineating the criteria for a classification of psychopathology based on empirical research. Based on the findings reviewed earlier, the following principles can be stated.

1. Any categorization system must be based on empirical research. Although this may seem self-evident, DSM (as noted earlier) is fundamentally based on consensus, not on empirical research.

2. Yet, although any classification system should rest on solid empirical ground, it is our view that classification systems should also be firmly rooted in an encompassing developmental theory of normal and pathological development. Just as the almost sole reliance on consensus may have led to arbitrary criteria that lack empirical foundation, an overreliance on atheoretical multivariate approaches, which are currently much in vogue, may lead to a classification system that neither provides a theoretically consistent view of psychopathology nor appeals to clinicians' intuitive ways of conceptualizing psychiatric disorders. Research increasingly demonstrates the complex pathways and the multifactorial and often recursive causality involved in both normal and abnormal development from infancy through childhood to senescence. This implies that a classification system should ideally be rooted in such research and driven by an encompassing the-

ory concerning the nature of personality development and psychopathology. As Pincus (2005) has noted, one of the most challenging tasks for the future may be to bridge the gap between current causal/theoretical and practical/empirical approaches to the classification of psychopathology. In addition, we agree with Pincus that such attempts should not necessarily be anchored in the current DSM classification system or need to account for DSM diagnoses.

3. Although discrete, categorical disorders may exist, a dimensional view of psychopathology appears to fit the data for most disorders better. Yet some disorders may represent true "taxa," and clinicians and insurance companies will continue to find it more convenient to think and speak in terms of categories (e.g., "This patient 'has' PTSD"). The challenge for the future, then, will be to develop a classification system that capitalizes on the advantages of both the dimensional and categorical views (e.g., see Westen & Shedler, 2000).

4. Finally, a classification system should possess clinical utility. It should not only be "nature friendly but also user friendly" (Westen et al., 2002, p. 222). DSM includes distinctions that are not only to some extent arbitrary, but also often difficult to make and cumbersome (as anyone who has ever administered a structured clinical interview for DSM will endorse). As noted, its clinical utility is further limited in that most diagnoses offer little information concerning treatment and prognosis (Kupfer et al., 2002).

The Two-Configurations Model

Relatedness and Self-Definition as Basic Dimensions in Personality Development

As noted at the start of this chapter, the two-configurations model proposes relatedness and self-definition, or attachment and separation, as two fundamental psychological dimensions that provide a theoretical matrix for understanding processes of personality development, variations in normal personality organization, concepts of psychopathology, and mechanisms of therapeutic action. This continuity of two primary dimensions in personality development and in psychopathology provides the basis for considering various forms of psychopathology as distortions that stem from variations and disruptions of normal psychological development. In addition, the articulation of these basic processes in personality development facilitates further understanding of processes of psychological development that can occur in therapy, as well as of the relationship between psychosocial and biological factors in normal and abnormal development (Blatt, 2006, 2008).

Importantly, these two basic psychological dimensions of relatedness and self-definition in personality development are consistent with a wide range of personality theories, ranging from classical to neopsychoanalytic conceptualizations to empirically derived formulations. A number of psychoanalytic theorists beyond Freud (e.g., Abraham, Jung, Adler, Rank, Horney, Tausk, Bowlby, Balint, Shor and Sanville, Sullivan, Kohut, M. Slavin, and Kriegman), as well as many nonpsychoanalytic personality theorists (e.g., Angyal, Bakan, L. Benjamin, Carson, Deci and Ryan, U. Foa, Gilligan, Hogan, L. Horowitz, Leary, McClelland, McAdams, Winter, Hegelson, Markus et al., Maddi, Spiegel and Spiegel, White, and Wiggins), have made these two fundamental dimensions of relatedness and self-definition central to their formulations (see Blatt, 2008, for a detailed discussion).

Blatt and colleagues (e.g., Blatt, 1974, 1990, 1991a, 1995a, 1995b, 2006, 2008; Blatt & Blass, 1990, 1996; Blatt & Shichman, 1983) have further elaborated these theoretical formulations in several ways. First, they have proposed that personality development evolves, from infancy to senescence, through a complex dialectic transaction between these two fundamental psychological dimensions. They define "relatedness" as the development of increasingly mature, intimate, mutually satisfying, reciprocal, interpersonal relationships; they define "self-definition" as the development of an increasingly differentiated, integrated, realistic, essentially positive sense of self or identity. These two fundamental developmental processes evolve through a lifelong, complex, synergistic, hierarchical, dialectic transaction, such that progress in one developmental line usually facilitates progress in the other. An increasingly differentiated, integrated, and mature sense of self emerges

out of constructive interpersonal relationships; conversely, the continued development of increasingly mature interpersonal relationships is contingent on the development of a more differentiated and integrated self-definition and identity. Meaningful and satisfying relationships contribute to the evolving concept of self, and a revised sense of self leads in turn to more mature levels of interpersonal relatedness.

Second, Blatt and colleagues' extension of Erikson's epigenetic psychosocial model (e.g., Erikson, 1963) illustrates how these two fundamental developmental dimensions of interpersonal relatedness and self-definition evolve and are eventually integrated in a mature self-identity—a "self-in-relation" with others (see Figure 25.1). This grounds the two-polarities model in a broad developmental model of human development from birth to senescence. Blatt and Shichman (1983) expanded Erikson's developmental model by including an additional psychosocial phase, "cooperation versus alienation," at about the age of 4–6 years with the emergent awareness of the triadic structure of the family (the Oedipal phase), the development of operational thinking (e.g., Piaget, 1954), and the beginnings of cooperative peer play (e.g., Whiteside, Busch, & Horner, 1976). They placed this phase at the appropriate point in Erikson's developmental sequence between the stage of "initiative versus guilt" and the latency stage of "industry versus inferiority."

This dialectical rendering of Erikson's formulations is congruent with other recent developmental models (e.g., Beebe et al., 2007; Stern, 1985). One developmental dimension (self-definition or individuality) evolves from early experiences of separation and autonomy from the primary caregiver, to a capacity to initiate activity (first in opposition to another, and later, proactively, to industry with sustained goal-directed activity that has direction and purpose), and finally to the emergence of individuality and a "self-identity." The addition of an intermediate stage of cooperation defines phases in a developmental dimension of interpersonal relatedness that evolves from the sharing of affective experiences between mother and infant (e.g., Beebe & Lachmann, 1988; Stern, 1985) with a concomitant sense of basic trust, to a capacity for cooperation and collaboration with peers, to the evolution of a close friendship with a same-sex chum (Sullivan, 1953), and finally to the development of mutual, reciprocal, enduring intimacy.

This broadened Eriksonian model also articulates the developmental dialectic transaction between relatedness and self-definition (attachment–separation or communion–agency). These two personality dimensions develop reciprocally throughout the life cycle from infancy through the early developmental years until adolescence,[1] at which time the developmental task is to integrate these two developmental dimensions of relatedness and self-definition into the com-

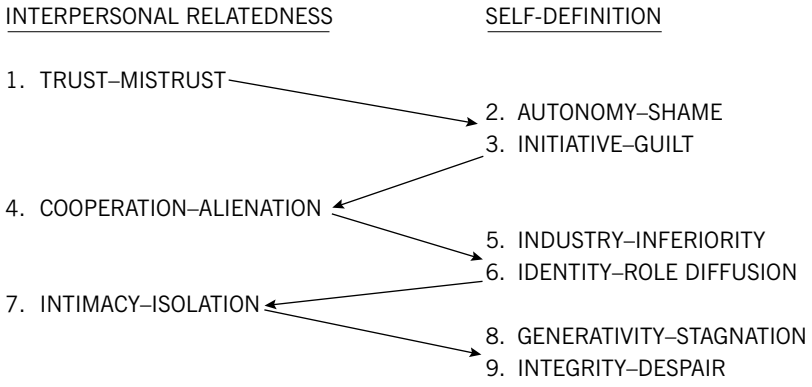


FIGURE 25.1. The dialectical interaction of interpersonal relatedness and self-definition implicit in Erikson's psychosocial model, as extended by Blatt and colleagues. From Blatt and Blass (1996). Copyright 1996 by Taylor & Francis. Reprinted by permission.

prehensive structure that Erikson called "self-identity" (Blatt & Blass, 1990, 1996) and that Blatt and colleagues call the "self-in-relation" (Blatt, 2006, 2008). Hence adolescence is a crucial time that can result in either the synthesis of a stable identity or the emergence of many forms of psychopathology, particularly the personality disorders, that are characterized by a partial or sometimes even complete failure to synthesize and integrate part-identities.

Relatedness and Self-Definition in Personality Organization

As Figure 25.1 illustrates, well-functioning personality organization involves an integration (or balance) in the development of interpersonal relatedness and of self-definition. However, each individual, even within the normal range, places a somewhat greater emphasis on one or the other of these dimensions. This relative emphasis delineates two basic personality or character styles, each with a particular experiential mode; preferred forms of cognition, defense, and adaptation; unique aspects of interpersonal relatedness; and specific forms of object and self-representation (Blatt, 2006, 2008; Luyten et al., 2005a). Blatt (1974) and Blatt and Shichman (1983) used the term "anaclitic" for the personality organization that focuses predominantly on interpersonal relatedness. This term was taken by Freud (1905/1963, 1915/1957b) from the Greek *anaklitas* ("to rest or lean on"), to characterize all interpersonal relationships that derive from dependency experienced in satisfying drives such as hunger in the context of the mother–child relationship (Laplanche & Pontalis, 1974). Blatt (1974) and Blatt and Shichman (1983) used the term "introjective" for the personality organization primarily focused on self-definition. This term was used by Freud (1917) to describe the processes whereby values, patterns of culture, motives, and restraints are assimilated into the self (e.g., made subjective), consciously or unconsciously, as guiding personal principles through learning and socialization.

Thinking in the anaclitic personality style is more figurative and focused primarily on affects and visual images. It is characterized by simultaneous rather than sequential processing, and by an emphasis on the reconciliation and synthesis of elements into an integrated cohesion rather than a critical analysis of separate elements and details (Szumotalska, 1992). The anaclitic personality style is characterized by a predominant tendency to seek fusion, harmony, integration, and synthesis. The focus is on personal experiences—on meanings, feelings, affects, and emotional reactions. These individuals are primarily field-dependent (Witkin, 1965) and are very aware of and influenced by environmental factors. Thinking in the introjective personality style, in contrast, is much more literal, sequential, linguistic, and critical. Concerns are focused on action, overt behavior, manifest form, logic, consistency, and causality. These individuals tend to place emphasis on analysis rather than on synthesis—on the critical dissection of details and part properties, rather than on achieving a total integration and an overall gestalt (Szumotalska, 1992). These individuals are predominantly field-independent (Witkin, 1965); thus their experiences and judgments are primarily influenced by internal, rather than environmental, factors.

Extensive research demonstrates the validity of the distinction between anaclitic and introjective personality styles in nonclinical samples (see summaries in Blatt, 2004, 2008; Blatt & Zuroff, 1992; Luyten et al., 2005a; Zuroff, Mongrain, & Santor, 2004).

Relatedness and Self-Definition in Psychopathology

The two-configurations model also provides a theoretical model for conceptualizing different forms of psychopathology as deviations from normal personality development. The two personality dimensions of relatedness and self-definition develop through the life cycle, each contributing to the shape and meaning given to psychological experiences. As discussed earlier, these developmental lines evolve in normal psychological development in a parallel and integrated form. Biological predispositions and severely disruptive environmental events, however, can interact in complex ways to disrupt this integrated developmental process and lead to defensive, markedly exaggerated emphasis on one developmental dimension at the

expense of the other. These deviations can be relatively mild in normal character variations, as discussed above, but they can also be quite extreme. The more extensive the deviation, the greater the exaggerated emphasis on one developmental line at the expense of the other, and thus the greater the possibility of psychopathology.

The two-configurations model is a dynamic structural developmental approach to personality development and psychopathology; as Fonagy (2008, p. xi) notes, it is the "first genuinely psychodynamic developmental psychopathology." It is supported by extensive research in which personality organization provides the basis for understanding the motivational organization and dynamic factors contributing to a wide range of symptomatic expressions of fundamental psychological disturbances. This hierarchical organization, in which different symptomatic expressions of psychological disturbances derive from more basic dimensions of personality organization, provides the basis for dealing with the complex and vexing problem of comorbidity that can occur in more conventional approaches to the diagnosis and classification of psychopathology.

A central assumption of the two-configurations model is that exaggerated distortion of one developmental line, to the neglect of the other, reflects compensatory or defensive maneuvers in response to developmental disruptions. Hence different forms of psychopathology are not static entities resulting from deficits in development, but dynamic, conflict–defense constellations that serve the purpose of maintaining a balance (however disturbed) between relatedness and self-definition. The differentiation of the anaclitic and introjective personality configurations thus provides the basis for considering different types of psychopathology in both Axis I and Axis II of DSM as exaggerated and distorted preoccupations, at different developmental levels, with either of the two fundamental dimensions of interpersonal relatedness and self-definition

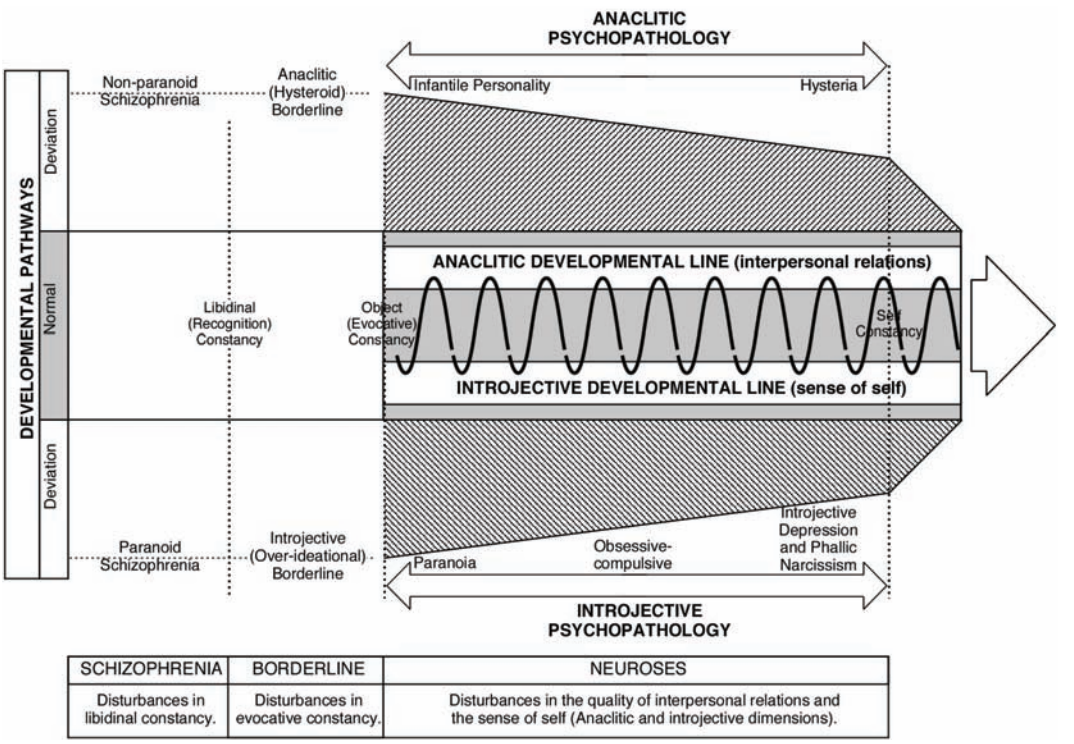


FIGURE 25.2. A model of normal and psychopathological development. From Blatt and Shichman (1983). Copyright 1983 by International Universities Press. Adapted by permission.

(Blatt & Shichman, 1983; see Figure 25.2). Anaclitic psychopathology is characterized by distorted and exaggerated attempts to maintain satisfying interpersonal experiences, whereas introjective psychopathology reflects distorted attempts to establish an effective sense of self. In addition, regression and progression on each of these developmental lines are possible, which may in part explain the high comorbidity and causal relationships between different disorders in the two configurations of psychopathology. We return to this issue below.

Psychopathologies within the anaclitic configuration share a basic preoccupation with libidinal (sensuous) issues, such as closeness and intimacy. These patients have a better capacity for affective bonding and a greater potential for developing meaningful interpersonal relations than patients with introjective psychopathologies. Psychopathologies within the anaclitic configuration also have similar defensive styles, with a predominant use of avoidant defenses (e.g., denial, repression, and displacement). Psychopathologies in the introjective configuration share a basic focus on aggression and themes of self-definition, self-control, and self-worth. They also exhibit similarity in defensive styles, with the predominant use of counteractive defenses (e.g., isolation, doing–undoing, intellectualization, reaction formation, introjection, identification with the aggressor, and overcompensation). Cognitive processes in the introjective configuration are more fully developed, with a greater potential for the development of logical thought.[2]

Anaclitic psychopathology involves exaggerated preoccupations with establishing and maintaining satisfying intimate relationships—with feeling loved and being able to love. Such patients are desperately concerned about trust, closeness, and the dependability of others, as well as with their capacity to receive and give love and affection. The development of the self is disrupted by these intense conflicts about feeling deprived of care, affection, and love. This excessive preoccupation with establishing and maintaining satisfying interpersonal relatedness can occur at several developmental levels—in a lack of differentiation between self and other, as in schizophrenia; in severe conflicts between dependency and the need for separateness, as in hysteroid borderline personality disorder; in intense dependent attachment, as in dependent or infantile personalities; and in difficulties in more mature, reciprocal types of relationships, as in hysterical personality organization. As noted above, patients with these disorders use primarily avoidant defenses (e.g., withdrawal, denial, repression) to cope with psychological conflict and stress and to avoid intense erotic longings and competitive strivings because these intense feelings potentially threaten their tenuous interpersonal relations.

Introjective psychopathology involves an excessive preoccupation with issues of self at varying developmental levels. These range from a basic sense of separation and differentiation from others, through concerns about autonomy and control of one's mind and body, to more internalized issues of self-worth, identity, and integrity. The development of interpersonal relations is interfered with by exaggerated struggles to establish and maintain a viable sense of self. Patients with introjective psychopathology are more ideational, and issues of anger and aggression, directed toward the self or others, are usually central to their difficulties. Introjective disturbances, ranging developmentally from more to less severe, include paranoid schizophrenia, overideational or schizoid borderline personality, paranoid personality, obsessive–compulsive personality, introjective (guilt-ridden) depression, and narcissistic or phallic narcissistic personality. As noted above, patients with these disorders use primarily counteractive defenses (e.g., projection, rationalization, negativism, isolation, intellectualization, doing and undoing, reaction formation, and overcompensation), such that the underlying impulse and conflict are partially expressed but in disguised form. The basic issue for such patients is to achieve separation, control, independence, and self-definition, and to be acknowledged, respected, and admired. Conflicts within the introjective configuration usually involve profound feelings of inadequacy, inferiority, worthlessness, guilt, and difficulty managing affect (especially anger and aggression) toward others and the self (Blatt, 1974, 1990, 1991a, 1995a, 1995b; Blatt & Shichman, 1983).

The relationship between anaclitic and introjective personality organization and

psychopathology and gender appears to be quite complex. Western society places more manifest emphasis on the need for self-definition for men, and greater emphasis for women on the capacity for relatedness (i.e., for care, affection, and love). Developmental disruptions, therefore, are often expressed in males and females along the predominant psychological tasks defined by cultural expectations. But this gender difference is also a function of fundamental developmental psychological processes. Both females and males have their initial bonding to the mother, and thus a primary normative developmental task for a young girl is to maintain her primary object of identification with her mother but to shift her primary object of affection to her father. Thus issues of relatedness are of central concern in the early development of women. The converse occurs with a young boy, who normatively maintains his primary object of affection with his mother but must normatively shift his primary object of identification to his father. Thus issues of identification or self-definition are of central importance in the early development of men (see Chevron, Quinlan, & Blatt, 1978; Golding & Singer, 1983).

Because Western society values self-definition in men and relatedness in women, Blatt (2004) also hypothesized that persons exhibiting gender incongruence (i.e., men with high levels of dependency and women with high levels of self-criticism) may be at increased risk for psychopathology. Hence anaclitic traits in men and introjective traits in women may be associated with implicit and explicit criticism by others, as well as with identity problems. Further research in this area is particularly needed because very few studies have examined the role of gender incongruence (Chevron et al., 1978; Golding & Singer, 1983; Sanfilipo, 1994; Smith, O'Keeffe, & Jenkins, 1988) in personality organization. Although these studies support the gender incongruence hypothesis, they have mainly been limited to investigations of the severity of depression. Chevron and colleagues (1978), for example, reported that valuing feminine characteristics (e.g., warmth/expressiveness) was positively related to severity of depression in men, as measured by the Zung Depression Scale (Zung, 1965). In contrast, in women, feminine traits were negatively related to severity of depression. Similarly, Silverstein, Clauson, Perdue, Carpman, and Cimarolli (1998) showed that anxious/somatic (anaclitic) depression was related to the thwarting of female achievement strivings, as well as to the perception that parents valued male attributes more than female attributes. Hence frustration of introjective issues may be associated with higher levels of depression in women. Luyten, Sabbe, and colleagues (2007), in turn, found that gender-incongruent personality organization in both women and men was associated with a higher risk of depression and other psychiatric disorders.

Despite the clear need for further research, particularly cross-cultural research, a considerable body of research suggests that gender differences in anaclitic and introjective psychopathology may be related to personality factors and attests to the fruitfulness of this approach. Disorders of the introjective configuration (e.g., antisocial personality disorder) occur with greater frequency in men, whereas disorders of the anaclitic configuration (e.g., borderline personality disorder, anaclitic depression) occur with greater frequency in women. Further research is needed to explore the biological, psychosocial, and cultural bases for these differences.

Relationship to DSM Disorders

Although we agree with Pincus (2005) that attempts to improve the current DSM classification system need not necessarily be anchored in DSM or have to account for current DSM diagnoses, we do think it important to investigate the relationship between the proposed two-polarities model and DSM nomenclature. We limit ourselves to studies that have focused on depression on the one hand, and personality disorders on the other. In particular, the differentiation of relatedness and of self-definition as two fundamental psychological dimensions has enabled investigators from several different theoretical orientations (e.g., Arieti & Bemporad, 1978, 1980; Beck, 1983; Blatt, 1974, 1998, 2004; Bowlby, 1988a, 1988b) to identify two fundamental dimensions in depression (Blatt & Maroudas, 1992): an anaclitic (dependent) dimension centered on feelings of loneliness, abandonment, and neglect, and an introjective (self-critical) di-

mension focused on issues of self-worth and feelings of failure and guilt (e.g., Blatt, 1974, 1998, 2004; Blatt, D'Afflitti & Quinlan, 1976; Blatt, Quinlan, & Chevron, 1990; Blatt, Quinlan, Chevron, McDonald, & Zuroff, 1982; Luyten et al., 2005a; Zuroff et al., 2004).

Research by Blatt and colleagues has shown that anaclitic or dependent depression is characterized by feelings of loneliness, helplessness, and weakness. These individuals have intense and chronic fears of being abandoned and left unprotected and uncared for. They have deep longings to be loved, nurtured, and protected. Because they have not internalized experiences of gratification or of qualities of the individuals who provided satisfaction, others are valued primarily for the immediate care, comfort, and satisfaction they provide. Separation from others and object loss create considerable fear and apprehension, and are often dealt with by denial and/or a desperate search for substitutes (Blatt, 1974). Anaclitically depressed individuals often express their depression in somatic complaints, frequently seeking the care and concern of others, including physicians (Blatt & Zuroff, 1992). Depression in these patients is often precipitated by object loss, and they often make suicidal gestures by overdosing on prescribed antidepressant medication (Blatt et al., 1982).

Introjective or self-critical depression, by contrast, is characterized by feelings of unworthiness, inferiority, failure, and guilt. These individuals engage in constant and harsh self-scrutiny and evaluation, and have a chronic fear of criticism and of losing the approval of significant others. They strive for excessive achievement and perfection, are often highly competitive and work hard, make many demands on themselves, and often achieve a great deal, but with little lasting satisfaction. Because of their intense competitiveness, they can also be critical and attacking toward others. Through overcompensation, they strive to achieve and maintain approval and recognition (Blatt, 1974, 2004). This focus on issues of self-worth, self-esteem, failure, and guilt can be particularly insidious, placing these individuals at considerable risk for serious suicide attempts (Beck, 1983; Blatt, 1974, 1995a, 1998; Blatt et al., 1982; Fazaa & Page, 2003). Numerous clinical reports, as well as accounts in the mass media, illustrate the considerable suicidal potential of highly talented, ambitious, and very successful individuals who are plagued by intense self-scrutiny, self-doubt, and self-criticism. Powerful needs to succeed and to avoid public criticism and the appearance of defect force such individuals to work incessantly to achieve and accomplish. But they are always profoundly vulnerable to the criticism of others and to their own self-scrutiny and judgment (Blatt, 1995a).

Arieti and Bemporad (1978, 1980), from a psychodynamic interpersonal perspective, distinguished two similar types of depression—a "dominant-other" and a "dominant-goal" type. When the dominant other is lost or the dominant goal is not achieved, depression can result. Arieti and Bemporad (1978) discussed two intense and basic wishes in depression: "to be passively gratified by the dominant other" and "to be reassured of one's own worth, and to be free of the burden of guilt" (p. 167). In the dominant-other type of depression, the individual desires to be passively gratified by developing a relationship that is clinging, demanding, dependent, and infantile. In the dominant-goal type, the individual seeks to be reassured of his or her worth and to be free of guilt by directing every effort toward a goal that has become an end in itself.

Congruent with these earlier psychoanalytic formulations of depression, Beck (1983), from a cognitive-behavioral perspective, has distinguished between "sociotropic" (socially dependent) and "autonomous" types of depression. Sociotropy, according to Beck, "refers to the person's investment in positive interchange with other people . . . including passive–receptive wishes (acceptance, intimacy, understanding, support, guidance)" (p. 273). Highly sociotropic individuals are "particularly concerned about the possibility of being disapproved of by others, and they often try to please others and maintain their attachments" (Robins & Block, 1988, p. 848). Depression is most likely to occur in these individuals in response to perceived loss or rejection in social relationships. Individuality (autonomy), according to Beck, refers to the person's "investment in preserving and increasing his independence, mobility, and personal rights; freedom of choice, action, and expression; protection

of his domain . . . and attaining meaningful goals" (p. 272). An autonomously depressed individual is "permeated with the theme of defeat or failure," blaming "himself continually for falling below his standards," and being "specifically self-critical for having 'defaulted' on his obligations" (p. 276). Highly autonomous, achievement-oriented individuals are very concerned about the possibility of personal failure and often try to maximize their control over the environment to reduce the probability of failure and criticism. Depression most often occurs in these individuals in response to a perceived failure to achieve or a lack of control over the environment.

Extensive empirical and clinical investigations (see Besser, Vliegen, Luyten, & Blatt, 2008; Blatt, 2004; Blatt & Zuroff, 1992; Luyten, Blatt, & Corveleyn, 2005b, 2005c) indicates consistent differences in the current and early life experiences of these two types of depressed individuals (Blatt & Homann, 1992), as well as major differences in their basic character style, their relational and attachment style (Luyten et al., 2005a), their clinical expression of depression (Blatt, 2004; Blatt & Zuroff, 2005), and their therapeutic response (Blatt, 2008; Blatt & Zuroff, 2005). Increasing evidence indicates that these differences can also be found in postpartum depression (Besser et al., 2008). Hence these findings indicate that it may be more fruitful to focus on underlying personality dynamics as a basis for classification than on manifest symptoms. We discuss this issue, and particularly its importance for treatment and treatment research, further below.

The differentiation between individuals preoccupied with issues of relatedness and those struggling with issues of self-definition has also enabled investigators to identify an empirically derived, replicated taxonomy for the diverse personality disorders described in Axis II of the DSM. Systematic empirical investigation of both inpatients and outpatients (Clark, Steer, Haslam, Beck, & Brown, 1997; Cogswell & Alloy, 2006; Levy et al., 1995; Morse, Robins, & Gittes-Fox, 2002; Nordahl & Stiles, 2000; Ouimette & Klein, 1993; Ouimette, Klein, Anderson, Riso, & Lizardi, 1994; Overholser & Freiheit, 1994; Pilkonis, 1988; Ryder, McBride & Bagby, 2008) has demonstrated that various Axis II personality disorders can be organized meaningfully and in theoretically expected ways into two primary configurations—one organized around issues of relatedness, and the other around issues of self-definition. Congruent with theoretical assumptions, these studies have generally found that individuals with dependent, histrionic, or borderline personality disorder have significantly greater concern with issues of interpersonal relatedness than with issues of self-definition, whereas individuals with paranoid, schizoid, schizotypal, antisocial, narcissistic, avoidant, obsessive–compulsive, or self-defeating personality disorder usually have significantly greater preoccupation with issues of self-definition than with issues of interpersonal relatedness. These findings are further supported by attachment research showing that personality disorders can be similarly organized in a two-dimensional space defined by "attachment anxiety" (reflecting anaclitic concerns) and "attachment avoidance" (reflecting introjective issues) (Meyer & Pilkonis, 2005). In addition, several studies have provided evidence for a distinction between "hysteroid" and "introjective" types of borderline personality disorder (Blatt & Auerbach, 1988; Levy, Edell, & McGlashan, 2007; Ryder et al., 2008; Westen et al., 1992; Wixom, Ludolph, & Westen, 1993; see also Southwick, Yehuda, & Giller, 1995).

Thus systematic empirical investigations from various perspectives indicate that the personality disorders in Axis II of DSM can be integrated parsimoniously into two configurations of anaclitic and introjective personality disorders. Yet, at the same time, these studies also show that there is no one-to-one relationship between the two-configurations model and the current DSM Axis II classification. For example, Ouimette and Klein (1993), who assessed two samples of students and depressed outpatients with five different measurement instruments of anaclitic and introjective features, found that not all of the predicted convergent and discriminant relationships between anaclitic and introjective features and DSM personality disorders were significant. Although a number of reasons may account for this—including the limitations of Axis II and the two-configurations model, as well as methodological limitations of current measurement instruments—one

should not expect a perfect relationship between the two categorization systems. As Levy and colleagues (2006) argue, current DSM criteria with their focus on symptoms may be unable to tap into the different underlying dynamics of patients who present with similar symptoms (see also Westen et al., 2006). For example, Levy and colleagues found that patients who received a DSM-IV diagnosis of borderline personality disorder showed marked differences in interpersonal distress, self-destructive behaviors, and impulsivity, but that these differences could be accounted for in theoretically predicted ways by the two-configurations model of a more anaclitic or hysteroid versus a more introjective or paranoid type of borderline personality disorder (Blatt, Ford, Berman, Cook, & Meyer, 1988).

In sum, the fundamental distinction between relatedness and self-definition has facilitated the differentiation of two primary types of psychopathology in both Axis I (depression) and Axis II (personality disorders) of the DSM, based on differences between an excessive preoccupation with issues of relatedness and an excessive focus on issues of self-definition. These findings have important implications not only for the classification of psychopathology, but also for the therapeutic process and for research on the relationship between psychosocial and neurobiological factors in the etiology of psychopathology.

Implications for the Classification of Psychiatric Disorders

First, a primary advantage of the two-configurations formulations of psychopathology is that they are based on the identification of continuities among processes in personality development, normal variations in personality organization, and various forms of psychopathology that are anchored in concepts of developmental psychopathology. These formulations thus avoid many of the pitfalls that have been discussed in frequent contemporary criticisms of the atheoretical DSM approach to diagnosis, including the problematic issue of extensive comorbidity (e.g., Blatt & Levy, 1998; Luyten, 2006; Luyten & Blatt, 2007; Luyten, Blatt, et al., 2006; Nemeroff, 2002; Parker, 2005; Widiger & Trull, 2007). In contrast to the atheoretical DSM diagnostic scheme, which is based primarily on differences in manifest symptoms, the anaclitic–introjective (or relational–self-definitional) distinctions of psychopathology derive from various dynamic considerations: differences in early life experiences, instinctual focus (libidinal vs. aggressive), types of defensive organization (avoidant vs. counteractive), relational style (approach vs. avoidance), and predominant character style (e.g., emphasis on orientation toward relationships vs. the self and on affects vs. cognition). We now have available a conceptual structure necessary to begin developing a classification system based on empirical research on the etiology and treatment response of the various disorders—a classification system that has important implications for both clinical practice and clinical research. The anaclitic and introjective configurations of personality development and psychopathology provide a comprehensive theoretical structure for identifying fundamental similarities among many forms of psychopathology, as well as for maintaining conceptual continuity across processes of psychological development and normal variations in character or personality organization.

Second, and related to the first issue, these formulations allow us to understand the relationships among different disorders in each of the two configurations as various levels of psychopathology within the anaclitic and the introjective configurations, thus defining different points of organization in the two developmental dimensions along which patients can progress or regress. An individual's difficulties can usually be located predominantly in one or the other configuration, at a particular developmental level, with a differential potential to regress or progress to other developmental levels within that configuration. Thus various forms of psychopathology are no longer seen as isolated, independent disorders. Instead, congruent with the developmental principles of equifinality and multifinality, different types of psychopathology can now be considered as interrelated modes of adaptation to difficult early and later life experiences in interaction with biological endowment, organized at different developmental levels within the two basic configurations. In this view, psychopathological disorders are com-

pensatory exaggerations and distortions in response to severe disruptions of the reciprocally balanced, normal, synergistic, dialectic development of interpersonal relatedness and self-definition. Severe disruptions of this developmental process result in exaggerated attempts to achieve equilibrium, through either an intense, distorted preoccupation with the quality of interpersonal relatedness or exaggerated defensive efforts to consolidate the sense of self.

Psychopathological disorders within the anaclitic configuration are interrelated and therefore show high comorbidity because they share a preoccupation with intense struggles to establish satisfying interpersonal relations (characterized by feelings of trust, intimacy, cooperation, and mutuality) at different levels of development. Because of an exaggerated emphasis upon interpersonal relatedness, the self is defined primarily in terms of the quality of interpersonal experiences. Psychopathological disorders within the introjective configuration, in contrast, are interrelated in their focus on struggles to achieve and maintain a sense of self-definition (characterized by feelings of separateness, autonomy, and self-worth) to the neglect of developing interpersonal relations. The primary preoccupation in these disorders with self-definition shapes and distorts the quality of interpersonal experiences.

Third, this emphasis on differences in personality organization in most forms of psychopathology is consistent with the recent emphasis on the necessity of establishing a dimensionally organized taxonomy of psychopathology—that is, based on a few broad overarching constructs or multiple dimensions of disordered thought, affect, behavior, temperament, or personality (e.g., Clark, 2005; Krueger, Watson, & Barlow, 2005; Widiger & Samuel, 2005; Widiger, Simonsen, Krueger, Livesley, & Verheul, 2005; Widiger & Trull, 2007). Such a taxonomy would "transcend a putative distinction between more normal and more abnormal psychological phenomena" and the "official nosologies such as the DSM" (Krueger, Watson, et al., 2005, p. 491). The anaclitic–introjective differentiation in personality organization and psychopathology stresses personality dimensions as the basis for establishing a coherent diagnostic classification system. Psychopathological disorders are therefore best understood on a continuum from normal personality organization to subclinical pathology to manifest clinical disorders (Blatt, 1974, 2004; Blatt & Shichman, 1983; Haslam, 2003; Luyten & Blatt, 2007; Ruscio & Ruscio, 2000; Tsuang et al., 2003; Widiger & Clark, 2000).

The formulation of two primary configurations of psychopathology not only provides a theoretical model for integrating clinical disorders in Axis I of DSM with the personality disorders of Axis II; it also suggests the possibility of identifying a hierarchical organization in which many symptom-based disorders can be subsumed within one of several major clinical disorders (Blatt, 2004; Blatt & Shichman, 1983; Clark, 2005; Krueger, Markon, Patrick, & Iacono, 2005; Watson, 2005). A hierarchical view of clinical disorders provides a parsimonious way of dealing with the problematic and vexing issue of comorbidity. Symptom-based diagnoses, such as conduct and antisocial personality disorders (e.g., Blatt, 2004; Blatt & Shichman, 1981), substance use disorders (e.g., Blatt, Rounsaville, Eyre, & Wilber, 1984; Lidz, Lidz, & Rubenstein, 1976), eating disorders (e.g., Bers, Blatt, & Dolinsky, 2004; Claes et al., 2006; Speranza et al., 2005; Thompson-Brenner & Westen, 2005; Westen & Harnden-Fischer, 2001), sleep disturbance (Norlander, Johansson, & Bood, 2005), PTSD (Gargurevich, 2006; Southwick et al., 1995), and chronic fatigue syndrome (e.g., Luyten, Van Houdenhove, Cosyns, & Van den Broeck, 2006; Luyten, Van Houdenhove, & Kempe, 2007; Van Houdenhove, Luyten, & Egle, 2009), for example, can often be considered as behavioral expressions of more primary disorders in either the anaclitic or introjective configuration. In addition, ample evidence suggests that the concept of anaclitic and introjective personality traits may explain the high comorbidity among many of these disorders (Shafran & Mansell, 2001).

These formulations have important implications for intervention. They indicate, for instance, that disruptive behavior in many symptom-based disorders, including conduct and antisocial personality disorders, are frequently defensive and distorted attempts to establish some form of interpersonal relatedness or some sense of self-worth (Blatt & Shichman, 1981)—issues that should be

a central focus of treatment, in addition to the more manifest symptomatic expressions of these disorders (Blatt & Shichman, 1981; First et al., 2004; Kupfer et al., 2002).

Implications for the Therapeutic Process

The utility and validity of the two-configurations model has been demonstrated in several investigations of the treatment process in both long-term intensive therapies and brief treatments, as well as in both inpatient and outpatient settings (Blatt, 1992; Blatt & Ford, 1994; Blatt & Shahar, 2004a, 2004b; Blatt & Zuroff, 2005; Vermote, 2005). These two groups of patients with different personality organizations and configurations of psychopathology had differential response in therapeutic process and outcome under the different conditions studied. Several prominent research methodologists (e.g., Cronbach, 1953) have long noted that much of the difficulty in identifying significant differences among different types of therapeutic intervention may be a function of the assumption of "homogeneity" among patients (Kiesler, 1966)—that is, the assumption that all patients are equivalent at the beginning of treatment (Blatt & Felsen, 1995). The failure to differentiate effectively among patients limits the potential to address more complex questions, such as whether certain treatments are more effective with certain kinds of patients, possibly resulting in different kinds of change (Blatt, Shahar, & Zuroff, 2001, 2002).

Blatt and colleagues introduced the anaclitic–introjective distinction into the evaluation of data from two major studies of patients receiving long-term, intensive, psychodynamically oriented treatment—outpatients in the Menninger Psychotherapy Research Project (MPRP), and inpatients at the Austen Riggs Center (the Riggs–Yale Project [R-YP]). They also incorporated it into analyses of data from an extensive study of the brief outpatient treatment of major depression: the National Institute of Mental Health (NIMH)–sponsored Treatment for Depression Collaborative Research Program (TDCRP). In the evaluation of long-term intensive treatment in the MPRP and the R-YP, experienced clinical judges were able to differentiate reliably between anaclitic and introjective patients on the basis of clinical case records prepared at the outset of treatment (see Blatt, 1992; Blatt & Ford, 1994; Blatt et al., 1988; Fertuck, Bucci, Blatt, & Ford, 2004). In the TDCRP, the anaclitic–introjective distinction was used dimensionally, as assessed by the Dysfunctional Attitudes Scale (DAS; Weissman & Beck, 1978). Findings from these studies of both long-term intensive therapies and brief treatments indicate that anaclitic and introjective patients responded differentially to different types of therapy and change in ways consistent with their personality organizations.

Anaclitic patients showed significantly greater therapeutic gains in supportive–expressive psychotherapy than in psychoanalysis in the MPRP; the reverse was true for the introjective patients, who made greater therapeutic gain in psychoanalysis (Blatt, 1992; Blatt & Shahar, 2004a, 2004b). These findings are consistent with findings from the study of long-term, intensive, psychodynamically oriented inpatient treatment in the R-YP (Blatt et al., 1988; Blatt & Ford, 1994), which indicated that seriously disturbed, treatment-resistant, introjective patients showed greater improvement than anaclitic patients, as evaluated via ratings of independently prepared clinical case records and psychological test protocols. Therapeutic change in the R-YP was most consistently expressed in introjective patients in changes in their manifest symptoms (as reliably rated from their clinical case records) and in the efficacy of their cognitive processes (as assessed via changes in intelligence and the level of thought disorder in the psychological test protocols). Therapeutic change in anaclitic patients, in contrast, was most consistently indicated by changes in their interpersonal relationships (as reported in the clinical case records) and in the quality of their representations of human figures on the Rorschach. Thus anaclitic and introjective patients expressed therapeutic change in the modalities most relevant to their types of psychopathology and their basic character structures.

These findings of constructive therapeutic gain, especially for introjective patients, in long-term psychodynamic treatment of inpatients in the R-YP and of outpatients in the MPRP are consistent with the findings

of Fonagy and colleagues (1996) and with the conclusions by Gabbard and colleagues (1994) about the constructive response of introjective patients with borderline personality disorder to long-term, insight-oriented, psychodynamic treatment. Thus findings from several studies indicate that such treatment is effective, especially with introjective patients.

The constructive response of introjective patients to long-term psychodynamic treatment in the MPRP and the R-YP stands in contrast to findings (e.g., Blatt, Quinlan, Pilkonis, & Shea, 1995; Blatt, Zuroff, Quinlan, & Pilkonis, 1996) from the extensive study of brief (16-week, once-weekly) outpatient treatment for severe depression in the NIMH-sponsored TDCRP. This study compared three manually directed brief outpatient treatments for depression—cognitive-behavioral therapy, interpersonal therapy, and imipramine with clinical management—to a double-blind, passive placebo with clinical management. As noted above, patients in the TDCRP had been administered the DAS (Weissman & Beck, 1978), which comprises two primary factors: Need for Approval and Perfectionism (e.g., Cane, Olinger, Gotlib, & Kuiper, 1986; Oliver & Baumgart, 1985). These factors are closely related to measures of anaclitic and introjective dimensions of depression, respectively (e.g., Blaney & Kutcher, 1991; Dunkley & Blankstein, 2000; Enns & Cox, 1999; Powers, Zuroff, & Topciu, 2004). Thus the anaclitic–introjective distinction was introduced into analyses of data from the TDCRP by using patients' pretreatment scores on the two factors of the DAS.

In contrast to the lack of significant differences in symptom reduction at termination and follow-up among the three active treatments in the TDCRP, analyses of the TDCRP data indicated that patients' personality characteristics had a major effect on treatment outcome and on aspects of the therapeutic process (Blatt et al., 1988, 1995, 2001; Blatt, Zuroff, et al., 1996; Shahar et al., 2003; Shahar, Blatt, Zuroff, Krupnick, & Sotsky, 2004; Zuroff et al., 2000). Specifically, patients' pretreatment levels of perfectionism or self-criticism (i.e., an introjective personality organization) resulted in poorer therapeutic outcome at termination and at follow-up in all three forms of brief treatment for depression evaluated in the TDCRP. In addition, these analyses indicated that the introjective personality dimension interfered with therapeutic progress primarily in the second half of the treatment process (in the last 8 weeks) by disrupting patients' development of interpersonal relationships both within and outside the treatment process (Shahar et al., 2004; Zuroff et al., 2000; Zuroff & Blatt, 2006). Thus introjective patients appear not to have benefited extensively from brief treatment in the TDCRP or from long-term supportive–expressive psychotherapy in the MPRP, but appear to have been particularly responsive to long-term, intensive, dynamically oriented treatment, including psychoanalysis, in the MPRP and the R-YP.

Taken together, these findings provide strong confirmation of Cronbach's (e.g., 1953) formulations that pretreatment characteristics of patients are important dimensions that influence therapeutic response (Blatt & Felsen, 1993). This mounting evidence for the crucial role of these characteristics reflects a major shift in psychotherapy research, in which data analyses are now going beyond the comparison of two forms of treatment for the reduction of a particular symptom (e.g., depression or anxiety) and are beginning to address more complex questions—such as what types of treatment are more effective, in what kinds of ways, with which types of patients (Blatt et al., 2002).

Affectively labile, emotionally overwhelmed, anaclitic patients, who usually have a preoccupied insecure attachment style, do better in supportive–expressive therapy because it contains their affective lability, possibly by reducing their associative activity. Introjective patients, who usually have an avoidant or dismissive attachment style, make significantly greater progress in treatment if they have more referential activity (Fertuck et al., 2004) and if they are in intensive, long-term, psychoanalytically oriented treatment (Blatt & Ford, 1994; Fonagy et al., 1996) that helps them overcome their interpersonal and emotional detachment (Eames & Roth, 2000; Mallinckrodt, Gantt, & Coble, 1995; Meyer, Pilkonis, Proietti, Heape, & Egan, 2001) through interpretations (Hardy et al., 1999). Emotionally and interpersonally detached introjective

patients seem to do better in psychoanalysis than in psychotherapy because psychoanalysis appears to liberate their associative processes (Blatt & Shahar, 2004a).

The two-configurations model of personality development and psychopathology thus has important implications for understanding some of the mechanisms that can lead to therapeutic change in the psychotherapeutic process (Blatt, 2008). Experiences of engagement and disengagement, of attachment and separation, of gratifying involvement with others and experiences of incompatibility with aspects of that involvement result in modifications and revisions in the representation of self and significant others in the treatment process, as in all of life (Blatt & Behrends, 1987). And these revised internalizations are expressed behaviorally and psychologically in more mature levels of self-definition and of interpersonal relatedness. Experiences of gratifying involvement and experienced incompatibility, which are central to the development of self-definition and interpersonal relatedness throughout life, are also central to personality development that can occur in the psychotherapeutic process. Hierarchical experiences of engagement and disengagement in the treatment process result in new and revised internalizations that lead to the development of more articulated, differentiated, and integrated representations of self, of others, and of their actual and potential relationships (Blatt, Auerbach, & Behrends, 2008; Blatt, Stayner, Auerbach, & Behrends, 1996; Diamond, Kaslow, Coonerty, & Blatt, 1990; Gruen & Blatt, 1990; Harpaz-Rotem & Blatt, 2005, 2009). Thus development in the psychotherapeutic process is similar in fundamental ways to the processes of normal psychological development.

The systematic study of these revisions in mental representations (or cognitive–affective schemas of self and of others) provides a method for assessing the extent and nature of therapeutic change—the reparative interpersonal therapeutic process in which individuals are able to move toward more mature levels of self-definition and more mature levels of interpersonal relatedness, with a capacity to find personal satisfaction in mutually enhancing and facilitating interpersonal relationships (e.g., Blatt et al., 1998; Blatt, Zuroff, et al., 1996; Calabrese, Farber, & Westen, 2005; Phillips, Wennberg, Werbart, & Schubert, 2006). This focus on changes in mental representation of self and of others in the treatment process provides links between treatment research and the newly emerging field of social-cognitive neuroscience (e.g., Lieberman, 2007), which seeks to use neuroscience research tools to examine social processes, including the understanding of oneself and others and the "processes that occur at the interface of self and others" (Lieberman, 2007, p. 259).

Implications for Research on Interactions between Psychosocial and Biological Factors

These formulations may furthermore provide the basis for studying the etiology, nature, and treatment of psychological disturbances within a biopsychosocial dynamic interactionism model (Luyten et al., 2006c), which seeks to identify recursive interactions among biological, psychological, and sociological factors in the etiology of psychological disturbances. In our opinion, recursive interactions among biological, psychological, and social context factors in the etiology of psychopathology are most effectively studied from the perspective of a theory-based, comprehensive model that specifies well-established developmental pathways from infancy to adulthood. This theory-based developmental approach is very different from the orientation of much contemporary psychiatric research, which is based on post hoc attempts to reconstruct etiological factors that could have contributed to the various disorders as they are identified in a clinical context. Such post hoc analyses are plagued by the fact that similar symptoms can emerge from different etiological pathways (equifinality) and, depending on a variety of factors and circumstances, can be expressed in different disorders (multifinality) (Luyten et al., 2008).

In contrast, the two-configurations model of personality development and psychopathology provides a prospective developmental approach to investigating the etiologies of various forms of psychopathology as variations and disruptions of clearly specified normal psychological development (Blatt, 2008). Research on the development of secure and insecure attachment patterns (e.g.,

Ainsworth, Blehar, Waters, & Wall, 1978; Fonagy, Steele, & Steele, 1991; Steele, Steele, & Fonagy, 1996), for example, clearly demonstrates the impact of a mother's attachment style and her relationship with her own mother on the development of her child's personality organization. Recent evidence (Besser & Priel, 2005) has demonstrated that these attachment patterns are in fact transmitted across at least three generations of women—from grandmother to mother to daughter. Substantial research demonstrates the impact of a mother's psychological disturbances on the child's development of different types of psychopathology, including depression (e.g., Blatt & Homann, 1992; Goodman & Gotlib, 2002; Kaminer, 1999).

Recent research (Beebe et al., 2007), in fact, demonstrates how a mother's personality organization influences her infant's development of self-regulation and interactive regulation as early as 4 months of age. Using the Depressive Experiences Questionnaire (DEQ; Blatt et al., 1976; Blatt, D'Afflitti, & Quinlan, 1979), Beebe and colleagues assessed 6 weeks after delivery the extent to which a normal, ethnically diverse, low-risk, well-educated sample of primiparous mothers of healthy first-born children experienced feelings of dependency or self-criticism. Next, they examined the impact of these feelings on the interactive play patterns in these mothers and their infants 4 months after the infants' birth. Using well-established split-screen analyses of mother–infant interaction, Beebe and colleagues (2007) found that elevated maternal scores on DEQ dependency and self-criticism 6 weeks postpartum significantly predicted lower infant self-regulation at 4 months of age. These two dimensions also predicted very different patterns of mother–infant interactive regulation at 4 months. More dependent (more anaclitic) mothers had heightened facial and vocal coordination with their infants—an "attentional vigilance" that was accompanied by heightened emotional activation in the infants. Infants of these dependent/anaclitic mothers showed a similar emotional vigilance and an intense reactivity to their mothers' affective shifts. This heightened mother–infant vigilance and dyadic symmetry indicate excessive maternal concern about the infants' availability that limits the infants' individuation and affect regulation.

In contrast, mothers with elevated scores on self-criticism (more introjective mothers) had difficulty sharing their infants' attentional focus and emotional variations. These mothers appeared to try to compensate for their disengagement with their infants by touching their infants more frequently—a more neutral type of engagement than sharing facial expressions, voice quality, or visual gaze. In response to the disengagement of these self-critical/introjective mothers, their infants seemed to disengage from their mothers by withdrawing vocal quality coordination. This distancing and disengagement between self-critical mothers and their infants appear to be the precursors of dismissive insecure attachment. The intense involvement of dependent mothers and their infants, in contrast, appears to be the precursor of preoccupied or anxious-ambivalent insecure attachment. It remains for subsequent research to examine the relationship of these early interpersonal interactive patterns observed at 4 months of age to attachment patterns observed in the second year of life and to the development of anaclitic and introjective forms of personality organization and psychopathology.

The research paradigm established by Beebe and her colleagues—that is, investigating the impact of personality variations in a nonclinical sample of first-time mothers on infants' early interpersonal engagement—provides a structure for systematically examining the impact of neurobiological and genetic dimensions on psychological development. Infants at 4 months of age begin to establish prerepresentational schemas of self and others that become increasingly consolidated in the symbolic representational structures associated with secure and insecure attachment patterns observed in the second year of life (e.g., Mikulincer & Shaver, 2007). The emergence of the behavioral, cognitive, and interpersonal expressions of these representational structures can be used to establish extensive research paradigms to evaluate the impact of mothers' neurobiological and genetic characteristics on their own caring patterns and on their infants' neurobiological development, particularly the maturation of the hypothalamic–pituitary–adrenal axis

(Claes & Nemeroff, 2005; Gutman & Nemeroff, 2003) and its role in their subsequent development. As noted recently by Gunnar and Quevedo (2007), individual differences in the social regulation of neurobiological reactions to stress observed in mother–infant interactions, and in the attachment representations that develop in this context, can provide a means for examining questions about the impact and management of stress throughout development. Rather than the post hoc searching for specific genetic and neurobiological markers of particular psychiatric diseases or disorders seen in the clinical context—an approach plagued by complex issues of the equifinality and multifinality of symptoms—the developmental approach to personality development and psychopathology, as exemplified by the research of Beebe and colleagues (2007), has considerable promise but will require extensive longitudinal studies. Such investigations, as the "developmental origins of health and disease" paradigm (e.g., Gluckman, Hanson, & Beedle, 2007), may also shed light on the complex interactions and relationships between life experiences and psychiatric as well as (functional) somatic syndromes (Luyten et al., 2008).

Relationship to Other Current Models of Psychopathology

Our proposed two-configurations model of personality development and psychopathology, like several other contemporary theoretical models (i.e., interpersonal and attachment models), offers an alternative that is very different from DSM's categorical, descriptive, symptom-based approach. An important future task is to investigate the relative merits and limitations of these alternative models, as well as their interrelationships. We believe that the proposed two-configurations model is conceptually and empirically linked to several of these other models in important ways.

In particular, the two-configurations model shares with contemporary interpersonal models an emphasis on agency and communion as the basis for understanding both normal and pathological development. The two-configurations model, as noted by Pincus (2005), may complement the interpersonal models with an emphasis on mental representations or cognitive–affective schemas of self and other. Conversely, the mathematical sophistication of contemporary interpersonal models and the testing of complex mathematical models may enrich the two-configurations model. The emphasis on issues of interpersonal relatedness and self-definition in the two-configurations model and in interpersonal theory is also congruent with studies showing that the two fundamental developmental dimensions underlie attachment styles (e.g., Mikulincer & Shaver, 2007). Autonomy (or self-definition) is closely related to attachment avoidance, and interpersonal relatedness (or sociotropy) to attachment anxiety (e.g., Sibley, 2007). The two-configurations model, with its emphasis on structural or developmental levels of psychopathology, may also extend and enrich contemporary attachment theory—for instance, by introducing further differentiations within types of insecure attachment (e.g., Blatt & Levy, 2003; Levy & Blatt, 1999), including subtypes of disorganized attachment (e.g., Blatt, 2004; Lyons-Ruth & Block, 1996; Lyons-Ruth, Zeanah, & Benoit, 2003).

By contrast, the two-configurations model has less in common with other, sometimes more symptom-based, and frequently theory-neutral classification systems that are derived from multivariate analyses, including the tripartite model (Clark, 2005), the five-factor model (FFM; Widiger & Trull, 2007), and formulations of externalizing and internalizing dimensions (Krueger et al., 2007; Krueger, Markon, et al., 2005). Although these approaches are clearly of merit, as both Pincus (2005) and Fonagy (2008) have pointed out, they risk having little clinical utility. In addition, these models fail to note and may even obscure important dynamic relationships between various disorders. For instance, studies based on the internalizing–externalizing formulations have often found that several disorders, such as depression and anxiety disorders, show high loadings on both the internalizing and externalizing dimensions (Krueger et al., 2007; Krueger, Markon, et al., 2005; Lahey et al., 2008; McGlinchey & Zimmerman, 2007).

Although such findings are difficult to accommodate within the internalizing–externalizing models, issues of relatedness and autonomy or self-definition in the two-configurations model can be expressed in both internalizing and externalizing forms, depending on other dynamic factors. Antisocial (externalizing) behaviors, for example, are often a defense against underlying (internalizing) feelings of depression and hence can be expected to show substantial comorbidity (Blatt, 2004; Blatt & Shichman, 1981). Thus any classification system based primarily on symptomatic expressions alone may lead to the same problems as experienced with the current DSM approach. Similarly, an atheoretical model such as the FFM may lack clinical utility, not only because of its emphasis on broad higher-order factors, but because of its lack of a developmental perspective. In addition, the FFM has been criticized for being biased toward normal personality functioning, resulting in a lack of sophistication for understanding psychopathology and its relationships to normal development and the treatment process (Shedler & Westen, 2004). However, only research directly comparing these different theoretical models will be able to demonstrate the relative merits and limitations of these competing approaches to the classification of psychopathology. Such research, therefore, is urgently needed.

Conclusions

This chapter has provided an overview of a contemporary psychodynamic approach to the conceptualization and classification of mental disorders. The proposed model argues for a developmental approach to psychopathology that emphasizes continuity between normal and pathological personality development and organization. Disorders are not static, relatively distinct diseases, each with unique causes. Rather, they are dynamic, hierarchically organized entities that are exaggerated distortions of normal developmental processes—exaggerations at different developmental levels that attempt to maintain an experience of interpersonal relatedness at the expense of a sense of self, or an exaggerated and distorted sense of self at the expense of interpersonal relatedness. The two-configurations conceptualization of psychopathology may also facilitate further clarification of the mutative factors in treatments across different therapeutic schools, as well as the interactions between biological and psychosocial factors in both normal and pathological development across developmental stages. The psychodynamically inspired two-configurations model thus contributes to the shift from a disorder- to a person-centered approach to psychological development and psychopathology—a shift that is restoring the importance of considering the role of the individual in psychiatry and psychology.

Note

1. The evolving capacities for autonomy, initiative, and industry in the self-definitional developmental dimension progress in an alternating sequence with the growth of relational capacities. For example, one needs a sense of basic trust to venture in opposition to the need-gratifying other in asserting one's autonomy and independence; later, one needs a sense of autonomy and initiative to establish cooperative and collaborative relationships with peers. Development begins with a focus on interpersonal relatedness—specifically, with the stage of trust versus mistrust—before proceeding to two early self-definitional stages: autonomy versus shame and initiative versus guilt. These early expressions of self-definition are then followed by the newly identified stage of interpersonal relatedness (cooperation vs. alienation), and then by two later stages of self-definition (industry vs. inferiority and identity vs. role diffusion). These more mature expressions of self-definition are followed by the more advanced stage of interpersonal relatedness (intimacy vs. isolation), before development proceeds to two mature stages of self-definition (generativity vs. stagnation and integrity vs. despair) (Blatt & Shichman, 1983).
2. Although most forms of psychopathology are organized primarily around one configuration or the other, some patients may have predominant features from both the anaclitic and introjective dimensions, and their psychopathology may thus derive from both configurations (see Shahar, Blatt, & Ford, 2003, for an investigation of patients with mixed anaclitic and introjective characteristics).

References

Ablon, J. S., & Jones, E. E. (2005). On analytic process. *Journal of the American Psychoanalytic Association*, *53*, 541–568.

Ainsworth, M. D. S., Blehar, M. E., Waters, E., & Wall, S. (1978). *Patterns of attachment: A psychological study of the Strange Situation*. Hillsdale, NJ: Erlbaum.

American Psychiatric Association (APA). (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

American Psychiatric Association (APA). (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Arieti, S., & Bemporad, J. R. (1978). *Severe and mild depression: The therapeutic approach*. New York: Basic Books.

Arieti, S., & Bemporad, J. R. (1980). The psychological organization of depression. *American Journal of Psychiatry*, *137*, 1360–1365.

Bateman, A., & Fonagy, P. (2004). *Psychotherapy for borderline personality disorder: Mentalization-based treatment*. Oxford, UK: Oxford University Press.

Beck, A. T. (1983). Cognitive therapy of depression: New perspectives. In P. J. Clayton & J. E. Barrett (Eds.), *Treatment of depression: Old controversies and new approaches* (pp. 265–290), New York: Raven Press.

Beebe, B., Jaffe, J., Buck, K., Chen, H., Cohen, P., Blatt, S. J., et al. (2007). Six-week postpartum maternal self-criticism and dependency predict 4-month mother–infant self- and interactive regulation. *Developmental Psychology*, *43*, 1360–1376.

Beebe, B., & Lachmann, F. (1988). The contribution of the mother–infant mutual influence to the origins of self- and object representations. *Psychoanalytic Psychology*, *5*, 305–338.

Bers, S. A., Blatt, S. J., & Dolinsky, A. (2004). The sense of self in anorexia-nervosa patients: A psychoanalytically informed method for studying self-representation. *Psychoanalytic Study of the Child*, *59*, 294–315.

Besser, A., & Priel, B. (2005). The apple does not fall from the tree: Attachment styles and personality vulnerabilities to depression in three generations of women. *Personality and Social Psychology Bulletin*, *31*, 1052–1073.

Besser, A., Vliegen, N., Luyten, P., & Blatt, S. J. (2008). Systematic empirical investigation of vulnerability to postpartum depression from a psychodynamic perspective. *Psychoanalytic Psychology*, *25*, 392–410.

Blaney, P. H., & Kutcher, G. S. (1991). Measures of depressive dimensions: Are they interchangeable? *Journal of Personality Assessment*, *56*, 502–512.

Blatt, S. J. (1974). Levels of object representation in anaclitic and introjective depression. *Psychoanalytic Study of the Child*, *29*, 107–157.

Blatt, S. J. (1990). Interpersonal relatedness and self-definition: Two personality configurations and their implications for psychopathology and psychotherapy. In J. L. Singer (Ed.), *Repression and dissociation: Implications for personality theory, psychopathology and health* (pp. 299–335). Chicago: University of Chicago Press.

Blatt, S. J. (1991a). A cognitive morphology of psychopathology. *Journal of Nervous and Mental Disease*, *179*, 449–458.

Blatt, S. J. (1991b). Depression and destructive risk-taking behavior in adolescence. In L. P. Lipsitt & L. L. Mitnick (Eds.), *Self-regulatory behavior and risk-taking: Causes and consequences* (pp. 285–309). Norwood, NJ: Ablex.

Blatt, S. J. (1992). The differential effect of psychotherapy and psychoanalysis on anaclitic and introjective patients: The Menninger Psychotherapy Research Project revisited. *Journal of the American Psychoanalytic Association*, *40*, 691–724.

Blatt, S. J. (1995a). The destructiveness of perfectionism: Implications for the treatment of depression. *American Psychologist*, *50*, 1003–1020.

Blatt, S. J. (1995b). Representational structures in psychopathology. In D. Cicchetti & S. Toth (Eds.), *Rochester Symposium on Developmental Psychopathology: Vol. 6. Emotion, cognition, and representation* (pp. 1–33). Rochester, NY: University of Rochester Press.

Blatt, S. J. (1998). Contributions of psychoanalysis to the understanding and treatment of depression. *Journal of the American Psychoanalytic Association*, *46*, 723–752.

Blatt, S. J. (2004). *Experiences of depression: Theoretical, clinical, and research perspectives*. Washington, DC: American Psychological Association.

Blatt, S. J. (2006). A fundamental polarity in psychoanalysis: Implications for personality development, psychopathology, and the therapeutic process. *Psychoanalytic Inquiry*, *26*, 492–518.

Blatt, S. J. (2008). *Polarities of experience: Relatedness and self-definition in personality*

development, psychopathology, and the therapeutic process. Washington, DC: American Psychological Association.

Blatt, S. J., & Auerbach, J. S. (1988). Differential cognitive disturbances in three types of "borderline" patients. *Journal of Personality Disorders*, *2*, 198–211.

Blatt, S. J., Auerbach, J. S., & Behrends, R. S. (2008). Changes in the representation of self and significant others in the treatment process: Links between representation, internalization, and mentalization. In A. Slade, E. Jurist, & S. Bergner (Eds.), *Mind to mind: Infant research, neuroscience and psychoanalysis* (pp. 225–263). New York: Other Press.

Blatt, S. J., & Behrends, R. S. (1987). Internalization, separation–individuation, and the nature of therapeutic action. *International Journal of Psycho-Analysis*, *68*, 279–297.

Blatt, S. J., & Blass, R. B. (1990). Attachment and separateness: A dialectic model of the products and processes of psychological development. *Psychoanalytic Study of the Child*, *45*, 107–127.

Blatt, S. J., & Blass, R. (1996). Relatedness and self definition: A dialectic model of personality development. In G. G. Noam & K. W. Fischer (Eds.), *Development and vulnerability in close relationships* (pp. 309–338). Mahwah, NJ: Erlbaum.

Blatt, S. J., Cornell, C. E., & Eshkol, E. (1993). Personality style, differential vulnerability and clinical course in immunological and cardiovascular disease. *Clinical Psychology Review*, *13*, 421–450.

Blatt, S. J., D'Afflitti, J. P., & Quinlan, D. M. (1976). Experiences of depression in normal young adults. *Journal of Abnormal Psychology*, *85*, 383–389.

Blatt, S. J., D'Afflitti, J., & Quinlan, D. M. (1979). *Depressive Experiences Questionnaire (DEQ)*. Unpublished research manual, Yale University.

Blatt, S. J., & Felsen, I. (1993). "Different kinds of folks may need different kinds of strokes": The effect of patients' characteristics on therapeutic process and outcome. *Psychotherapy Research*, *3*, 245–259.

Blatt, S. J., & Ford, R. (1994). *Therapeutic change: An object relations perspective*. New York: Plenum Press.

Blatt, S. J., Ford, R. Q., Berman, W., Cook, B., & Meyer, R. (1988). The assessment of change during the intensive treatment of borderline and schizophrenic young adults. *Psychoanalytic Psychology*, *5*, 127–158.

Blatt, S. J., & Homann, E. (1992). Parent–child interaction in the etiology of dependent and self-critical depression. *Clinical Psychology Review*, *12*, 47–91.

Blatt, S. J., & Levy, K. N. (1998). A psychodynamic approach to the diagnosis of psychopathology. In J. W. Barron (Ed.), *Making diagnosis meaningful* (pp. 73–109). Washington, DC: American Psychological Association.

Blatt, S. J., & Levy, K. N. (2003). Attachment theory, psychoanalysis, personality development, and psychopathology. *Psychoanalytic Inquiry*, *23*, 102–150.

Blatt, S. J., & Maroudas, C. (1992). Convergence of psychoanalytic and cognitive behavioral theories of depression. *Psychoanalytic Psychology*, *9*, 157–190.

Blatt, S. J., McDonald, C., Sugarman, A., & Wilber, C. (1984). Psychodynamic theory of opiate addiction: New directions for research. *Clinical Psychological Review*, *4*, 159–189.

Blatt, S. J., Quinlan, D. M., & Chevron, E. (1990). Empirical investigations of a psychoanalytic theory of depression. In J. Masling (Ed.), *Empirical studies of psychoanalytic theories* (Vol. 3, pp. 89–147). Hillsdale, NJ: Analytic Press.

Blatt, S. J., Quinlan, D. M., Chevron, E. S. McDonald, C., & Zuroff, D. (1982). Dependency and self-criticism: Psychological dimensions of depression. *Journal of Consulting and Clinical Psychology*, *50*, 113–124.

Blatt, S. J., Quinlan, D. M., Pilkonis, P. A., & Shea, T. (1995). Impact of perfectionism and need for approval on the brief treatment of depression: The National Institute of Mental Health Treatment of Depression Collaborative Research Program revisited. *Journal of Consulting and Clinical Psychology*, *63*, 125–132.

Blatt, S. J., Rounsaville, B. J., Eyre, S., & Wilber, C. (1984). The psycho-dynamics of opiate addiction. *Journal of Nervous and Mental Disease*, *172*, 342–352.

Blatt, S. J., & Shahar, G. (2004a). Psychoanalysis: For what, with whom, and how: A comparison with psychotherapy. *Journal of the American Psychoanalytic Association*, *52*, 393–447.

Blatt, S. J., & Shahar, G. (2004b). Stability of the patient-by-treatment interaction in the Menninger Psychotherapy Research Project. *Bulletin of the Menninger Clinic*, *68*, 23–36.

Blatt, S. J., Shahar, G., & Zuroff, D. C. (2001). Anaclitic (sociotropic) and introjective (autonomous) dimensions. *Psychotherapy*, *38*, 449–454.

Blatt, S. J., Shahar, G., & Zuroff, D. C. (2002). Anaclitic (sociotropic) and introjective (autonomous) dimensions. In J. C. Norcross (Ed.), *Psychotherapy relationships that work: Therapist contributions and responsiveness to patients* (pp. 306–324). New York: Oxford University Press.

Blatt, S. J., & Shichman, S. (1981). Antisocial behavior and personality organization. In S. Tuttman, C. Kaye, & M. Zimmerman (Eds.), *Object and self: A developmental approach: Essays in honor of Edith Jacobson* (pp. 325–367). Madison, CT: International Universities Press.

Blatt, S. J., & Shichman, S. (1983). Two primary configurations of psychopathology. *Psychoanalysis and Contemporary Thought*, *6*, 187–254.

Blatt, S. J., Stayner, D., Auerbach, J., & Behrends, R. S. (1996). Change in object and self representations in long-term, intensive, inpatient treatment of seriously disturbed adolescents and young adults. *Psychiatry: Interpersonal and Biological Processes*, *59*, 82–107.

Blatt, S. J., & Zuroff, D. C. (1992). Interpersonal relatedness and self-definition: Two prototypes for depression. *Clinical Psychology Review*, *12*, 527–562.

Blatt, S. J., & Zuroff, D. C. (2005). Empirical evaluation of the assumptions in identifying evidence based treatments in mental health. *Clinical Psychology Review*, *25*, 459–486.

Blatt, S. J., Zuroff, D. C., Quinlan, D. M., & Pilkonis, P. A. (1996). Interpersonal factors in brief treatment of depression: Further analyses of the National Institute of Mental Health Treatment of Depression Collaborative Research Program. *Journal of Consulting and Clinical Psychology*, *64*, 162–171.

Bornstein, R. F. (1992). Parental perceptions and psychopathology. *Journal of Nervous and Mental Disease*, *180*, 475–483.

Bornstein, R. F., & Masling, J. M. (2005). The Rorschach Oral Dependency scale. In R. F. Bornstein & J. M. Masling (Eds.), *Scoring the Rorschach: Seven validated systems* (pp. 135–157). Mahwah, NJ: Erlbaum.

Bowlby, J. (1988a). Developmental psychology comes of age. *American Journal of Psychiatry*, *145*, 1–10.

Bowlby, J. (1988b). *A secure base: Clinical applications of attachment theory*. London: Routledge & Kegan Paul.

Brown, T. A., & Barlow, D. H. (2005). Dimensional versus categorical classification of mental disorders in the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* and beyond: Comments on the special section. *Journal of Abnormal Psychology*, *114*, 551–556.

Calabrese, M. L., Farber, B. A., & Westen, D. (2005). The relationship of adult attachment constructs to object relational patterns of representing self and others. *Journal of the American Academy of Psychoanalysis and Dynamic Psychiatry*, *33*, 513–530.

Cane, D. B., Olinger, L. J., Gotlib, I. H., & Kuiper, N. A. (1986). Factor structure of the Dysfunctional Attitude Scale in a student population. *Journal of Clinical Psychology*, *42*, 307–309.

Charney, D. S., Barlow, D. H., Botteron, K., Cohen, J., Goldman, D., Gur, J. D., et al. (2002). Neuroscience research agenda to guide development of a pathophysiologically based classification system. In D. J. Kupfer, M. B. First, & D. A. Regier (Eds.), *A research agenda for DSM-V* (pp. 31–83). Washington, DC: American Psychiatric Association.

Chevron, E. S., Quinlan, D. M., & Blatt, S. J. (1978). Sex roles and gender differences in the experience of depression. *Journal of Abnormal Psychology*, *87*, 680–683.

Cicchetti, D., & Rogosch, F. A. (1996). Equifinality and multifinality in developmental psychopathology. *Development and Psychopathology*, *8*, 597–600.

Claes, L., Vandereycken, W., Luyten, P., Soenes, B., Pieters, G., & Vertommen, H. (2006). Personality prototypes in eating disorders based on the Big Five model. *Journal of Personality Disorders*, *20*, 401–416.

Claes, S. J., & Nemeroff, C. B. (2005). Corticotropin releasing factor (CRF) and major depression: Towards an integration of psychology and neurobiology in depression research. In J. Corveleyn, P. Luyten, & S. J. Blatt (Eds.), *The theory and treatment of depression: Towards a dynamic interactionism model* (pp. 227–252). Leuven, Belgium: University of Leuven Press.

Clark, D. A., Steer, R. A., Haslam, N., Beck, A. T., & Brown, G. K. (1997). Personality vulnerability, psychiatric diagnoses, and symp-

toms: Cluster analyses of the Sociotropy and Autonomy subscales. *Cognitive Therapy and Research*, *21*, 267–283.

Clark, L. A. (2005). Temperament as a unifying base for personality and psychopathology. *Journal of Abnormal Psychology*, *114*, 505–521.

Cogswell, A., & Alloy, L. B. (2006). The relation of neediness and Axis II pathology. *Journal of Personality Disorders*, *20*, 16–21.

Cox, B. J., McWilliams, L. A., Enns, M. W., & Clara, I. P. (2004). Broad and specific personality dimensions associated with major depression in a nationally representative sample. *Comprehensive Psychiatry*, *45*, 246–253.

Cronbach, L. J. (1953). Correlation between persons as a research tool. In O. H. Mowrer (Ed.), *Psychotherapy: Theory and research* (pp. 376–389). New York: Ronald Press.

Cuthbert, B. N. (2005). Dimensional models of psychopathology: Research agenda and clinical utility. *Journal of Abnormal Psychology*, *114*, 565–569.

Diamond, D., Kaslow, N., Coonerty, S., & Blatt, S. J. (1990). Change in separation–individuation and intersubjectivity in long-term treatment. *Psychoanalytic Psychology*, *7*, 363–397.

Dunkley, D. M., & Blankstein, K. R. (2000). Self-critical perfectionism, coping, hassles, and current distress: A structural equation modeling approach. *Cognitive Therapy and Research*, *24*, 713–730.

Eames, V., & Roth, A. (2000). Patient attachment orientation and the early working alliance: A study of patient and therapist reports of alliance quality and ruptures. *Psychotherapy Research*, *10*, 421–434.

Ellenberger, H. F. (1970). *The discovery of the unconscious*. London: Allen Lane.

Enns, M. W., & Cox, B. J. (1999). Perfectionism and depressive symptom severity in major depressive disorder. *Behaviour Research and Therapy*, *37*, 783–794.

Erikson, E. H. (1963). *Childhood and society* (2nd ed.). New York: Norton.

Fazaa, N., & Page, S. (2003). Dependency and self-criticism as predictors of suicidal behavior. *Suicide and Life-Threatening Behavior*, *33*, 172–185.

Fertuck, E., Bucci, W., Blatt, S. J., & Ford, R. Q. (2004). Verbal representation and therapeutic change in anaclitic and introjective patients. *Psychotherapy: Theory, Research, Practice, Training*, *41*, 13–25.

First, M. B. (2005). Clinical utility: A prerequisite for the adoption of a dimensional approach in DSM. *Journal of Abnormal Psychology*, *114*, 560–564.

First, M. B., Pincus, H. A., Levine, J. B., Williams, J. B., Ustun, B., & Peele, R. (2004). Clinical utility as a criterion for revision psychiatric diagnoses. *American Journal of Psychiatry*, *161*, 946–954.

Fonagy, P. (2008). Foreword. In S. J. Blatt, *Polarities of experience: Relatedness and self definition in personality development, psychopathology, and the therapeutic process* (pp. ix–xiii). Washington, DC: American Psychological Association.

Fonagy, P., Leigh, T., Steele, M., Steele, H., Kennedy, R., Mattoon, G., et al. (1996). The relation of attachment status, psychiatric classification, and response to psychotherapy. *Journal of Consulting and Clinical Psychology*, *64*, 22–31.

Fonagy, P., Steele, H., & Steele, M. (1991). Maternal representations of attachment during pregnancy predict the organization of infant–mother attachment at one year of age. *Child Development*, *62*, 891–905.

Frances, A. J., First, M. B., & Pincus, H. A. (1995). *DSM-IV guidebook*. Washington, DC: American Psychiatric Press.

Freud, S. (1957a). Draft G: On melancholia. In J. Strachey (Ed. & Trans.), *The standard edition of the complete psychological works of Sigmund Freud* (Vol. 18, pp. 7–64). London: Hogarth Press. (Original work published 1895)

Freud, S. (1957b). Mourning and melancholia. In J. Strachey (Ed. & Trans.), *The standard edition of the complete psychological works of Sigmund Freud* (Vol. 14, pp. 243–258). London: Hogarth Press. (Original work published 1917)

Freud, S. (1963). Introductory lectures in psychoanalysis. Part III: General theory of the neuroses. In J. Strachey (Ed. & Trans.), *The standard edition of the complete psychological works of Sigmund Freud* (Vol. 16, pp. 339–377). London: Hogarth Press. (Original work published 1917)

Gabbard, G. O., Horowitz, L., Allen, J. G., Frieswyk, S., Newson, G., Colson, D. B., et al. (1994). Transference interpretation in the psychotherapy of borderline patients: A high-risk, high-gain phenomenon. *Harvard Review of Psychiatry*, *2*, 59–69.

Gargurevich, R. (2006). *Post-traumatic stress disorder and disasters in Peru*. Unpublished doctoral dissertation, University of Leuven, Leuven, Belgium.

Gluckman, P. D., Hanson, M. A., & Beedle, A. S. (2007). Early life events and their consequences for later disease: A life history and evolutionary perspective. *American Journal of Human Biology*, *19*, 1–19.

Golding, J. M., & Singer, J. L. (1983). Patterns of inner experience: Daydreaming styles, depressive moods, and sex roles. *Journal of Personality and Social Psychology*, *45*, 663–675.

Goodman, S. H., & Gotlib, I. (2002). Risk for psychopathology in the children of depressed mothers: A developmental model for understanding the mechanisms of transmission. *Psychological Bulletin*, *106*, 458–490.

Gruen, R., & Blatt, S. J. (1990). Change in self and object representation during long-term dynamically oriented treatment. *Psychoanalytic Psychology*, *7*, 399–422.

Gunnar, M., & Quevedo, K. (2007). The neurobiology of stress and development. *Annula Review of Psychology*, *58*, 145–173.

Gutman, D. A., & Nemeroff, C. E. (2003). Persistent central nervous system effects of an adverse early environment: Clinical and preclinical studies. *Physiology and Behavior*, *79*, 471–478.

Hammen, C. (2005). Stress and depression. *Annual Review of Clinical Psychology*, *1*, 293–339.

Hardy, G. E., Aldridge, J., Davidson, C., Rowe, C., Reilly, S., & Shapiro, D. (1999). Therapist responsiveness to patient attachment styles and issues observed inpatient-identified significant events in psychodynamic–interpersonal psychotherapy. *Psychotherapy Research*, *9*, 36–53.

Harpaz-Rotem, I., & Blatt, S. J. (2005). Changes in representations of a self designated significant other in long term intensive inpatient treatment of seriously disturbed adolescents and young adults. *Psychiatry: Interpersonal and Biological Processes*, *68*, 266–282.

Harpaz-Rotem, I., & Blatt, S. J. (2009). A pathway to therapeutic change: Changes in self-representation in the treatment of adolescents and young adults. *Psychiatry: Interpersonal and Biological Processes*, *72*, 32–49.

Haslam, N. (2003). Categorical versus dimensional models of mental disorders: The taxonometric evidence. *Australian and New Zealand Journal of Psychiatry*, *37*, 696–704.

Heim, C., Plotsky, P. M., & Nemeroff, C. B. (2005). Importance of studying the contributions of early adverse experiences to neurobiological findings in depression. *Neuropsychopharmacology*, *29*, 641–648.

Hudson, J. I., Arnold L. M., Keck, P. E., Jr., Auchenbach, M. B., & Pope, H.G., Jr. (2004). Family study of fibromyalgia and affective spectrum disorder. *Biological Psychiatry*, *56*, 884–891.

Kaminer, T. (1999). *Maternal depression, maternal speech, and infant gaze at 4 months*. Unpublished doctoral dissertation, St. John's University, New York.

Keller, M. B., Klein, D. N., Hirschfeld, R. M. A., Kocsis, J. H., McCullough, J. P., Miller, I., et al. (1995). Results of the DSM-IV mood disorders trial. *American Journal of Psychiatry*, *152*, 843–849.

Kendler, K. S., & Gardner, C. O. (1998). Boundaries of major depression: An evaluation of DSM-IV criteria. *American Journal of Psychiatry*, *155*, 172–177.

Kendler, K. S., Kuhn, J., & Prescott, C. A. (2004). The interrelationship of neuroticism, sex, and stressful life events in the prediction of episodes of major depression. *American Journal of Psychiatry*, *161*, 631–636.

Kiesler, D. J. (1966). Some myths of psychotherapy research and the search for a paradigm. *Psychological Bulletin*, *65*, 110–136.

Knight, R. P. (1940). Introjection, projection, and identification. *Psychoanalytical Quarterly*, *9*, 334–341.

Kraepelin, E. (1907). *Clinical psychiatry* (A. R. Diefendorf, Ed. & Trans.). New York: Macmillan. (Original work published 1904)

Krueger, R. F., Markon, K. E., Patrick, C. J., Benning, S. D., & Kramer, M. D. (2007). Linking antisocial behavior, substance use, and personality: An integrative quantitative model of the adult externalizing spectrum. *Journal of Abnormal Psychology*, *116*, 645–666.

Krueger, R. F., Markon, K. E., Patrick, C. J., & Iacono, W. G. (2005). Externalizing psychopathology in adulthood: A dimensional-spectrum conceptualization and its implications for DSM-V. *Journal of Abnormal Psychology*, *114*, 537–550.

Krueger, R. F., Watson, D., & Barlow, D. H. (2005). Introduction to the special section: Toward a dimensionally based taxonomy of

psychopathology. *Journal of Abnormal Psychology*, *114*, 491–493.

Kupfer, D. J., First, M. B., & Regier, D. A. (2002). Introduction. In D. J. Kupfer, M. B. First, & D. E. Regier (Eds.), *A research agenda for DSM-V* (pp. xv–xxiii). Washington, DC: American Psychiatric Association.

Lahey, B. B., Rathouz, P. J., Van Hulle, C., Urbano, R. C., Krueger, R. F., Applegate, B., et al. (2008). Testing structural models of DSM-IV symptoms of common forms of child and adolescent psychopathology. *Journal of Abnormal Child Psychology*, *36*, 187–206.

Lam, D. H., Wright, K., & Smith, N. (2004). Dysfunctional assumptions in bipolar disorder. *Journal of Affective Disorders*, *79*, 193–199.

Laplanche, J., & Pontalis, J. B. (1974). *The language of psycho-analysis* (D. Micholson-Smith, Trans.). New York: Norton.

Levy, K. N., & Blatt, S. J. (1999). Attachment theory and psychoanalysis: Further differentiation within insecure attachment patterns. *Psychoanalytic Inquiry*, *19*, 541–575.

Levy, K. N., Clarkin, J. F., Yeomans, F. E., Scott, L. N., Wasserman, R. H., & Kernberg, O. (2006). The mechanisms of change in the treatment of borderline personality disorder with transference focused psychotherapy. *Journal of Clinical Psychology*, *62*, 481–501.

Levy, K. N., Edell, W. S., Blatt, S. J., Becker, D. F., Quinlan, D. M., Kolligan, J., et al. (1995). *Two configurations of psychopathology: The relationship of dependency, anaclitic neediness, and self-criticism to personality pathology*. Unpublished manuscript.

Levy, K. N., Edell, W. S., & McGlashan, T. H. (2007). Depressive experiences in inpatients with borderline personality disorder. *Psychiatric Quarterly*, *78*, 129–143.

Lidz, T., Lidz, R. W., & Rubenstein, R. (1976). An anaclitic syndrome in adolescent amphetamine addicts. *Psychoanalytic Study of the Child*, *31*, 317–348.

Lieberman, M. D. (2007). Social cognitive neuroscience: A review of core processes. *Annual Review of Psychology*, *58*, 259–289.

Linehan, M. M. (1993). *Cognitive-behavioral treatment of borderline personality disorder*. New York: Guilford Press.

Luyten, P. (2006). Psychopathology: A simple twist of fate or a meaningful distortion of normal development? Toward an etiologically based alternative to the DSM approach. *Psychoanalytic Inquiry*, *26*, 521–535.

Luyten, P., & Blatt, S. J. (2007). Looking back towards the future: Is it time to change the DSM approach to psychiatric disorders? The case of depression. *Psychiatry: Interpersonal and Biological Processes*, *70*, 85–99.

Luyten, P., Blatt, S. J., & Corveleyn, J. (2005a). The convergence among psychodynamic and cognitive-behavioral theories of depression: Theoretical overview. In J. Corveleyn, P. Luyten, & S. J. Blatt (Eds.), *The theory and treatment of depression: Towards a dynamic interactionism model* (pp. 95–136). Leuven, Belgium: University of Leuven Press.

Luyten, P., Blatt, S. J., & Corveleyn, J. (2005b). Introduction. In J. Corveleyn, P. Luyten, & S. J. Blatt (Eds.), *The theory and treatment of depression: Towards a dynamic interactionism model* (pp. 5–15). Leuven, Belgium: University of Leuven Press.

Luyten, P., Blatt, S. J., & Corveleyn, J. (2005c). Towards integration in the theory and treatment of depression. In J. Corvelyen, P. Luyten, & S. J. Blatt (Eds.), *The theory and treatment of depression: Towards a dynamic interactionism model* (pp. 253–284). Leuven, Belgium: University of Leuven Press.

Luyten, P., Blatt, S. J., & Corvelyen, J. (2006). Mind the gap between positivism and hermeneutics in psychoanalytic research. *Journal of the American Psychoanalytic Association*, *54*, 572–609.

Luyten, P., Blatt, S. J., Van Houdenhove, B., & Corveleyn, J. (2006). Depression research and treatment: Are we skating to where the puck is going to be? *Clinical Psychology Review*, *26*, 985–999.

Luyten, P., Sabbe, B., Blatt, S. J., Meganck, S., Jansen, B., DeGrave, C., et al. (2007). Dependency and self-criticism: Relationship with major depressive disorder, severity of depression and clinical presentation. *Depression and Anxiety*, *24*, 586–596.

Luyten, P., Van Houdenhove, B., Cosyns, N., & Van den Broeck, A. (2006). Are patients with chronic fatigue syndrome perfectionistic—or were they: A case–control study. *Personality and Individual Differences*, *40*, 1473–1483.

Luyten, P., Van Houdenhove, B., & Kempe, S. (in press). Putting the psychodynamics back into chronic fatigue syndrome and fibromyalgia: From clinical observation to neurobiology and back. *Journal of Aggression Maltreatment and Trauma*.

Luyten, P., Viegen, N., Van Houdenhove, B., & Blatt, S. J. (2008). Equifinality, multifinality

and the rediscovery of the importance of early experiences: Pathways from early adversity to psychiatric and (functional) somatic disorders. *Psychoanalytic Study of the Child*, *63*, 27–60.

Lyons-Ruth, K., & Block, D. (1996). The disturbed caregiving system: Relations among childhood trauma, maternal caregiving, and infant affect and attachment. *Infant Mental Health Journal*, *17*, 257–275.

Lyons-Ruth, K., Zeanah, C. H., & Benoit, D. (2003). Disorder and risk for disorder during infancy and toddlerhood. In E. J. Mash & R. A. Barkley (Eds.), *Child psychopathology* (2nd ed., pp. 589–631). New York: Guilford Press.

Mallinckrodt, B., Gantt, D. L., & Coble, H. M. (1995). Attachment patterns in the psychotherapy relationship: Development of the Patient Attachment to Therapist Scale. *Journal of Counseling Psychology*, *42*, 307–317.

McGlinchey, J. B., & Zimmerman, M. (2007). Examining a dimensional representation of depression and anxiety disorders' comorbidity in psychiatric outpatients with item response modelling. *Journal of Abnormal Psychology*, *116*, 464–474.

McHugh, P. R. (2005). Striving for coherence: Psychiatry's effort over classification. *Journal of the American Medical Association*, *293*, 2526–2528.

Meyer, A. (1951–1952). *Collected papers of Adolf Meyer*. Baltimore: John Hopkins University Press.

Meyer, B., & Pilkonis, P. A. (2005). An attachment model of personality disorders. In M. F. Lenzenweger & J. F. Clarkin (Eds.), *Major theories of personality disorder* (2nd ed., pp. 231–281). New York: Guilford Press.

Meyer, B., Pilkonis, P. A., Proietti, J. M., Heape, C. L., & Egan, M. (2001). Attachment styles, personality disorders, and response to treatment. *Journal of Personality Disorder*, *15*, 371–389.

Mikulincer, M., & Shaver, P. R. (2007). *Attachment in adulthood: Structure, dynamics, and change*. New York: Guilford Press.

Moffitt, T. E., Caspi, A., & Rutter, M. (2005). Strategy for investigating interactions between measured genes and measured environments. *Archives of General Psychiatry*, *62*, 473–481.

Morse, J., Robins, C. J., & Gittes-Fox, M. (2002). Sociotropy, autonomy, and personality disorder criteria in psychiatric patients. *Journal of Personality Disorders*, *16*, 549–560.

Nemeroff, C. B. (2002). Editorial: Comorbidity of mood and anxiety disorders: The rule not the exception. *American Journal of Psychology*, *159*, 3–4.

Nemeroff, C. B., Heim, C. M., Thase, M. E., Klein, D. N., Rush, J. A., Schatzberg, A. F., et al. (2003). Differential responses to psychotherapy versus pharmacotherapy in patients with chronic forms of major depression and childhood trauma. *Proceedings of the National Academy of Sciences USA*, *100*, 14239–14296.

Nordahl, H. M., & Stiles, T. C. (2000). The specificity of cognitive personality dimensions in Cluster C personality disorders. *Behavioral and Cognitive Psychotherapy*, *28*, 235–246.

Norlander, T., Johansson, A., & Bood, S. A. (2005). The affective personality: Its relation to quality of sleep, well-being, and stress. *Social Behavior and Personality*, *33*, 709–722.

Oliver, J. M., & Baumgart, B. P. (1985). The Dysfunctional Attitude Scale: Psychometric properties in an unselected adult population. *Cognitive Theory and Research*, *9*, 161–169.

Ormel, J., Oldehinkel, A. J., & Brilman, E. I. (2001). The interplay and etiological continuity of neuroticism, difficulties, and life events in the etiology of major and subsyndromal, first and recurrent depressive episodes in later life. *American Journal of Psychiatry*, *158*, 885–891.

Ouimette, P. C., & Klein, D. N. (1993). Convergence of psychoanalytic and cognitive-behavioral theories of depression: An empirical review and new data on Blatt's and Beck's models. In J. M. Masling & R. F. Bornstein (Eds.), *Psychoanalytic perspectives on psychopathology* (pp. 191–223). Washington, DC: American Psychological Association.

Ouimette, P. C., Klein, D. N., Anderson, R., Riso, L. P., & Lizardi, H. (1994). Relationship of sociotropy/autonomy and dependency/self-criticism to DSM-III-R personality disorders. *Journal of Abnormal Psychology*, *103*, 743–749.

Overholser, J. C., & Freiheit, S. R. (1994). Assessment of interpersonal dependency using the Millon Clinical Multiaxial Inventory II (MCMI-II) and the Depressive Experiences Questionnaire. *Personality and Individual Differences*, *17*, 71–78.

Parker, G. (2000). Classifying depression: Should paradigms lost be regained? *American Journal of Psychiatry*, *157*, 1195–1203.

Parker, G. (2005). Beyond major depression. *Psychological Medicine*, *35*, 467–474.

Phillips, B., Wennberg, P., Werbart, A., & Schubert, J. (2006). Young adults in psychoanalytic psychotherapy: Patient characteristics and treatment outcome. *Psychology and Psychotherapy*, *79*, 89–106.

Phillips, K. A., First, M. B., & Pincus, H. A. (Eds.). (2003). *Advancing DSM: Dilemmas in psychiatric diagnosis*. Washington, DC: American Psychiatric Association.

Piaget, J. (1954). *The construction of reality in the child* (M. Cook, Trans.). New York: Basic Books.

Pilkonis, P. A. (1988). Personality prototypes among depressives: Themes of dependency and autonomy. *Journal of Personality Disorders*, *2*, 144–152.

Pincus, A. L. (2005). A contemporary integrative interpersonal theory of personality disorders. In M. F. Lenzenweger & J. F. Clarkin (Eds.), *Major theories of personality disorder* (2nd ed., pp. 282–331). New York: Guilford Press.

Powers, T. A., Zuroff, D. C., & Topciu, R. (2004). Covert and overt expressions of self-criticism and perfectionism and their relation to depression. *European Journal of Personality*, *18*(1), 61–72.

Raphael, K. G., Janal, M. N., Nayak, S., Schwartz, J. E., & Gallagher, R. M. (2004). Familial aggregation of depression in fibromyalgia: A community-based test of alternate hypotheses. *Pain*, *110*, 449–460.

Robins, C. J., & Block, P. (1988). Personal vulnerability, life events, and depressive symptoms: A test of a specific interactional model. *Journal of Personality and Social Psychology*, *54*, 847–852.

Ruscio, J., & Ruscio, A. M. (2000). Informing the continuity controversy: A taxometric analysis of depression. *Journal of Abnormal Psychology*, *109*, 473–487.

Ryder, A. G., McBride, C., & Bagby, R. M. (2008). The association of affiliation and achievement personality styles with DSM-IV personality disorders. *Journal of Personality Disorder*, *22*, 208–216.

Sanfilipo, M. P. (1994). Masculinity, femininity, and subjective experiences of depression. *Journal of Clinical Psychology*, *50*, 144–157.

Shafran, R., & Mansell, W. (2001). Perfectionism and psychopathology: A review of research and treatment. *Clinical Psychology Review*, *21*, 879–906.

Shahar, G., Blatt, S. J., & Ford, R. Q. (2003). The identification of mixed anaclitic–introjective psychopathology in young adult inpatients. *Psychoanalytic Psychology*, *20*, 84–102.

Shahar, G., Blatt, S. J., Zuroff, D. C., Krupnick, J., & Sotsky, S. M. (2004). Perfectionism impedes social relations and response to brief treatment of depression. *Journal of Social and Clinical Psychology*, *23*, 140–154.

Shedler, J., & Westen, D. (2004). Refining personality disorder diagnosis: Integrating science and practice. *American Journal of Psychiatry*, *161*, 1350–1365.

Sibley, C. G. (2007). The association between working models of attachment and personality: Toward an integrative framework operationalizing global relational models. *Journal of Research in Personality*, *41*, 90–109.

Silverstein, B., Clauson, J., Perdue, L., Carpman, S., & Cimarolli, V. (1998). The association between female college students reports of depression and their perceptions of parental attitudes regarding gender. *Journal of Applied Social Psychology*, *28*, 537–549.

Smith, T. W., O'Keeffe, J. C., & Jenkins, M. (1988). Dependency and self-criticism: Correlates of depression or moderators of the effects of stressful events? *Journal of Personality Disorders*, *2*, 160–169.

Solomon, A., Haaga, D. A. F., & Arnow, B. A. (2001). Is clinical depression distinct from subthreshold depressive symptoms?: A review of the continuity issue in depression research. *Journal of Nervous and Mental Disease*, *189*, 498–506.

Southwick, S. M., Yehuda, R., & Giller, E. L. (1995). Psychological dimensions of depression in borderline personality disorder. *American Journal of Psychiatry*, *152*, 789–791.

Speranza, M., Corcos, M., Loas, G., Stephan, P., Guilbaud, O., Perez-Diaz, F., et al. (2005). Depressive personality dimensions and alexithymia in eating disorders. *Psychiatric Research*, *135*, 153–163.

Steele, H., Steele, M., & Fonagy, P. (1996). Associations among attachment classifications of mothers, fathers, and their infants. *Child Development*, *67*, 541–555.

Stern, D. N. (1985). *The interpersonal world of the infant: A view from psychoanalysis and developmental psychology*. New York: Basic Books.

Sullivan, H. S. (1953). *The interpersonal theory of psychiatry*. New York: Norton.

Szumotalska, E. (1992). *Severity and type of de-*

pressive affect as related to perceptual styles: Relationship of anaclitic versus introjective depressive configuration to holistic versus analytic similarity judgment. Unpublished doctoral dissertation, New School for Social Research, New York.

Thompson-Brenner, H., & Westen, D. (2005). Personality subtypes in eating disorders: Validation of a classification in a naturalistic sample. *British Journal of Psychiatry*, *186*, 516–524.

Tsuang, M. T., Stone, W. S., Tarbox, S. I., & Faraone, S. V. (2003). Implications of schizotaxia for the prevention of schizophrenia. In A. Grispini (Ed.), *Preventive strategies for schizophrenic disorders: Basic principles, opportunities and limits* (pp. 225–243). Rome: Giovanni Fioriti Editore.

Van Houdenhove, B., Luyten, P., & Egle, U. T. (2009). The role of childhood trauma in chronic pain and fatigue. In V. L. Banyard, V. J. Edwards, & K. A. Kendall-Tackett (Eds.), *Trauma and physical health: Understanding the effects of extreme stress and of psychological harm* (pp. 37–64). London: Routledge.

Van Houdenhove, B., Luyten, P., & Egle, U. (in press). Stress as a key concept in chronic pain and fatigue disorders. *Journal of Musculoskeletal Disorders*.

van Praag, H. M., de Kloet, R., & van Os, J. (2004). *Stress, the brain and depression*. Cambridge, UK: Cambridge University Press.

Vermote, R. (2005). *Touching inner change: Psychoanalytically informed hospitalization-based treatment of personality disorders. A process–outcome study*. Unpublished doctoral dissertation, Katholieke Universiteit, Leuven, Belgium.

Watson, D. (2005). Rethinking the mood and anxiety disorders: A quantitative hierarchical model for DSM-V. *Journal of Abnormal Psychology*, *114*, 522–532.

Weinstock, L. M., & Whisman, M. A. (2006). Neuroticism as a common feature of the depressive and anxiety disorders: A test of the revised integrative hierarchical model in a national sample. *Journal of Abnormal Psychology*, *115*, 68–74.

Weissman, A. N., & Beck, A. T. (1978, August–September). *Development and validation of the Dysfunctional Attitudes Scale: A preliminary investigation*. Paper presented at the 86th Annual Convention of the American Psychological Association, Toronto.

Westen, D., & Harnden-Fischer, T. (2001). Personality profiles in eating disorders: Rethinking the distinction between Axis I and Axis II. *American Journal of Psychiatry*, *158*, 547–562.

Westen, D., Heim, A. K., Morrison, K., Patterson, M., & Campbell, L. (2002). Simplifying diagnosis using a prototype-matching approach: Implications for the next edition of the DSM. In L. Beutler & M. Malik (Eds.), *Rethinking the DSM: A psychological perspective* (pp. 221–250). Washington, DC: American Psychological Association.

Westen, D., Novotny, C. M., & Thompson-Brenner, H. (2004). The empirical status of empirically supported psychotherapies: Assumptions, findings, and reporting in controlled clinical trials. *Psychological Bulletin*, *130*, 631–663.

Westen, D., & Shedler, J. (2000). A prototype matching approach to diagnosing personality disorders: Toward DSM-V. *Journal of Personality Disorders*, *14*, 109–126.

Westen, D., Shedler, J., & Bradley, R. (2006). A prototype approach to personality disorder diagnosis. *American Journal of Psychiatry*, *163*, 846–856.

Whiteside, M. F., Busch, F., & Horner, T. (1976). From egocentric to cooperative play in young children: A normative job study. *Journal of the American Academy of Child Psychiatry*, *15*, 294–313.

Widiger, T. A., & Clark, A. (2000). Toward DSM-V and the classification of psychopathology. *Psychological Bulletin*, *126*, 946–963.

Widiger, T. A., & Samuel, D. B. (2005). Diagnostic categories of dimensions?: A question for the *Diagnostic and Statistical Manual of Mental Disorders—Fifth Edition*. *Journal of Abnormal Psychology*, *114*, 494–504.

Widiger, T. A., Simonsen, E., Krueger, R. F., Livesley, W. J., & Verheul, R. (2005). Personality disorder research agenda for the DSM-V. *Journal of Personality Disorders*, *19*, 315–330.

Widiger, T. A., & Trull, T. J. (2007). Plate tectonics in the classification of personality disorder: Shifting to a dimensional model. *American Psychologist*, *62*, 71–83.

Witkin, H. A. (1965). Psychological differentiation and forms of pathology. *Journal of Abnormal Psychology*, *70*, 317–336.

Wixom, J., Ludolph, P., & Westen, D. (1993). The quality of depression in adolescents with

borderline personality disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, *32*, 1172–1177.

Zung, W. W. (1965). Self-rating depression scale. *Archives of General Psychiatry*, *12*, 63–70.

Zuroff, D. C., & Blatt, S. J. (2006). The therapeutic relationship in the brief treatment of depression: Contributions to clinical improvement and enhanced adaptive capacities. *Journal of Consulting and Clinical Psychology*, *47*, 130–140.

Zuroff, D. C., Blatt, S. J., Sotsky, S. M., Krupnick, J. L., Martin, D. J., Sanislow, C. A., et al. (2000). Relation of therapeutic alliance and perfectionism to outcome in brief outpatient treatment of depression. *Journal of Consulting and Clinical Psychology*, *68*, 114–124.

Zuroff, D. C., Mongrain, M., & Santor, D. A. (2004). Conceptualizing and measuring personality vulnerability to depression: Commentary on Coyne and Whiffen (1995). *Psychological Bulletin*, *130*, 489–511.

CHAPTER 26

A Life Course Approach to Psychoses

Outcome and Cultural Variation

RINA DUTTA
ROBIN M. MURRAY

The diagnosis of schizophrenia in both the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV; American Psychiatric Association, 1994) and the 10th revision of the *International Classification of Diseases* (ICD-10; World Health Organization, 1992) is based on the premise that it is a discrete illness entity, in particular distinct from the affective psychoses. This assumption has persisted for more than a century, even though schizophrenia has a wide diversity of lifetime outcomes, considerable cultural variation in phenomenology, and no known biological or psychological feature that is pathognomonic of the disorder. Furthermore, the degree of overlap between schizophrenia and primary mood disorder with psychotic features has led to development of a less than satisfactory intermediate category—schizoaffective disorder.

Writing about the nosology of schizophrenia almost two decades ago, one of Britain's most sophisticated phenomenologists, Ian Brockington (1992), enjoined:

> It is important to loosen the grip which the concept of "schizophrenia" has on the minds of psychiatrists. Schizophrenia is an idea whose very essence is equivocal, a nosological category without natural boundaries, a barren hypothesis. Such a blurred concept is "not a valid object of scientific enquiry."

By describing psychotic disorders from a life course perspective, encompassing cultural variation and differences in prognosis, we consider whether the concept of schizophrenia "versus" affective psychoses should be abandoned by deconstructing psychosis into its component dimensions for DSM-V and ICD-11.

The Historical Context of the Concept of Validity in Psychoses

For a categorical diagnosis of schizophrenia to be valid in a scientific sense, it should be a syndrome with specific risk factors, psychopathology, treatment responses, and outcomes, as well as clear symptom boundaries separating it from such conditions as the affective psychoses (Robins & Guze, 1970). The idea that such a distinction could be made between "dementia praecox" and "manic–depressive insanity" (i.e., schizophrenia and affective psychosis) has pervaded psychiatric research since Kraepelin's

original proposal of his eponymous dichotomy at the turn of the last century. This is despite the fact that in 1920 Kraepelin came to doubt his own approach and suggested replacing his defining principle with a dimensional–hierarchical model more appropriate to the heterogeneity of clinical presentations (Kraepelin, 1920/1992).

The 1960s saw a sustained attack on the validity of psychiatric diagnosis from the so-called "antipsychiatrists," such as R. D. Laing and Thomas Szasz (curiously, both psychiatrists). In response, from the late 1960s onward, a large number of competing operational diagnostic systems were proposed in an attempt to improve the reliability of psychiatric diagnoses in general and of schizophrenia in particular. Robins and Guze (1970) suggested five criteria to establish the validity of a categorical diagnosis of schizophrenia: clinical description, laboratory studies, delimitation from other disorders, follow-up studies, and family studies. Kendler (1980) developed this approach by distinguishing among antecedent, concurrent, and predictive validators.

Originally designed for research purposes, competing diagnostic systems (such as Feighner's, Taylor's, Schneider's, Langfeldt's, Spitzer's, Carpenter's, Astrachan's, two from Forrest & Hay, and CATEGO) were compared with respect to their reliability, concordance, and prediction of outcome (American Psychiatric Association, 1980) and showed wide disparity. The first attempt to provide diagnostic rules for clinicians as well as researchers was made in DSM-III (American Psychiatric Association, 1980).

When the earlier systems and DSM-III were compared, they varied by as many as sevenfold in their rates of diagnosing schizophrenia (Endicott, 1982). Like the Feighner criteria, the DSM-III definition of schizophrenia was narrow, requiring 6 months of illness before the diagnosis could be made. Both criteria sets also had a high degree of predictive specificity, with one study using these criteria showing no change in diagnosis over an average of 6.5 years' follow-up (Helzer, Brockington, & Kendell, 1981). These operational definitions were generally shown to be relatively reliable as well, once psychiatrists were trained in their use. However, reliability does not necessarily imply validity, and no single classification system proved to have superior validity over others. Indeed, attempts to study validity as opposed to reliability were limited.

A different solution was to adopt a polydiagnostic approach, in which several sets of criteria were applied to the same patients (Berner, Katschnig, & Lenz, 1982; Jansson & Parnas, 2007). One tool was the Operation Criteria Checklist for psychotic illness (Farmer et al., 1992). This approach uses a suite of computer programs to generate diagnoses according to 13 different classification systems. It has been a useful adjunct to research methodology, in view of the absence of clearly defined boundaries for schizophrenia and the wide variety of presentations.

An alternative solution was to speculate that schizophrenia comprised several discrete subtypes. There followed in the 1980s and 1990s attempts to account for diagnostic heterogeneity by probing for subtypes of schizophrenia—for example, negative, positive, and mixed schizophrenia (Andreasen & Olsen, 1982); hebephrenic and paranoid subtypes (Farmer, McGuffin, & Gottesman, 1984); and familial and sporadic schizophrenia (Murray, Lewis, & Reveley, 1985). Murray and colleagues (1992) later sought to discriminate developmental from adult-onset forms. Support for the hypothesis came from latent-class analyses, but there remained the problem of intermediate forms (Castle et al., 1994; Sham et al., 1996). Furthermore, genetic and environmental risk factors were seen to operate across diagnostic categories (Done et al., 1994; van Os et al., 1997). Revising the diagnostic concept to take this into account might suggest a return to the concept of a psychotic spectrum (the unitary hypothesis); however, this has not been adopted.

Does a Life Course Perspective Help in Classifying Psychoses?

Genetics, Neurodevelopment, and Symptomatology

Schizophrenia and bipolar I disorder cosegregate in the same families, owing to common underlying genetic predispositions. In a twin study using "blinded" diagnostic assessments, Cardno, Rijsdijk, and colleagues (2002) showed that if one member of a monozygotic twin pair has schizophrenia,

there is a more than 40% chance that the co-twin will meet criteria for schizophrenia; yet the co-twin also has an 8.2% chance of being diagnosed with schizoaffective disorder and an 8.2% chance of being diagnosed with mania instead. However, children who later exhibit schizophrenia are characterized by impairments in cognitive and neuromotor development and by intellectual decline (Reichenberg et al., 2005), which are not features of those who later develop bipolar I disorder (Cannon et al., 2002).

Confirmation that patients with bipolar I disorder do not have general neurocognitive impairment is provided by the Israeli Draft Board Registry study (Reichenberg et al., 2002), which showed that 68 individuals hospitalized with this disorder did not differ from their healthy matched counterparts on any test of intellectual, language, or behavioral functioning conducted routinely when they were adolescents. A more recent cohort study, using national registers to follow all Swedish children who completed compulsory education, showed that no students with excellent school performance developed schizophrenia or schizoaffective disorder. By contrast, achieving outstanding grades in certain school subjects was a significant predictor of later bipolar I disorder (MacCabe et al., 2006, 2008).

Further evidence that schizophrenia and bipolar I disorder are at least partially distinct in etiology comes from studying complications of pregnancy and delivery. The risk-increasing effect of obstetrical complications has been shown for schizophrenia (Geddes et al., 1999), but not bipolar I disorder. There is also some suggestion that small size for gestational age and bleeding during pregnancy are associated with increased risk of early-onset schizophrenia among males and could reflect placental insufficiency (Hultman et al., 1999).

The similarities and differences between schizophrenia and bipolar I disorder begin to suggest a model (Figure 26.1) in which, given a shared background of genetic predisposition to psychosis, additional specific genetic or early environmental insults interact to impair neurodevelopment, leaving individuals vulnerable to schizophrenia. By contrast, in bipolar I disorder, developmental impairment is absent, but syndrome-specific genes and environmental interactions may render individuals susceptible to social adversity (Dutta et al., 2007).

Traditionally, first-rank symptoms are given particular emphasis for making a diagnosis of schizophrenia rather than bipolar I disorder. However, although Cardno, Sham, and colleagues (2002) showed that a syndrome characterized by the presence of one or more first-rank symptoms has considerable heritability (71%; 95% confidence interval = 57–82%, compatible with a genetic contribution to variance in liability), it remains somewhat lower than that for schizophrenia as defined by established classifications, including DSM criteria.

An alternative to considering syndrome-based approaches to psychopathology is to

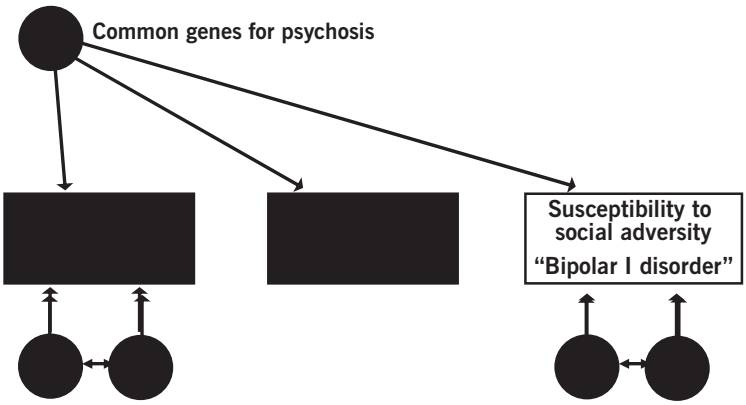


FIGURE 26.1. Gene–environment interactions to explain the overlap and distinctions between schizophrenia and bipolar I disorder. Based on Cardno, Rijsdijk, et al. (2002) and Murray et al. (2004).

study the validity of using identified groups of correlated symptoms (symptom dimensions) in patient populations that include a range of diagnostic groups (van Os et al., 1999) (shown schematically in Figure 26.2). Different research teams have usually extracted four or five different factors or dimensions (e.g., depressive, manic, positive, negative, and disorganized symptoms), and these broad factors have been remarkably consistent among studies of different patient cohorts. Recently it has been shown that using such symptom dimensions explains more about disease characteristics (such as premorbid impairment, the existence of stressors before disease onset, poor remissions or no recovery between episodes and exacerbations, response to neuroleptics, and deterioration) than diagnoses alone, and thus adds substantial information to diagnostic categories (Dikeos et al., 2006).

Although the intention in devising DSM-III was to use "research evidence relevant to various kinds of diagnostic validity" (Spitzer, Williams, & Skodol, 1980), as well as "the largest reliability study ever done" (Spitzer, 1985), the DSM-III Task Force chairman, Robert Spitzer (1985), acknowledged that "the subjective judgment of the members of the task force . . . played a crucial role in the development of DSM-III, and differences of opinion could only rarely be resolved by appeal to objective data." To date, neither the DSM nor the ICD review process has used external validators (such as quantitative biological measurements or psychological testing) to assist in the evaluation of current diagnostic criteria or to judge whether changes are improving clinical validity. A radical shift to a dimensional model has been debated, but so far such a model has been dismissed as less useful than the categorical model, perhaps owing to unfamiliarity with dimensional descriptions and perhaps because they do not evoke vivid clinical pictures. However, introducing a hybrid categorical–dimensional model to see whether clinicians do find dimensions useful as a means of conveying clinical information—and, indeed, of stimulating research, by encouraging them to look at disorders from a different perspective—might be a compromise and advance for both DSM-V and ICD-11 (Dutta et al., 2007).

How Does Cultural Variation Assist Us in Understanding the Psychoses?

The experience, content, and understanding of psychotic symptoms are embedded in a network of local meanings (Maslowski, van Jansen, & Mthoko, 1998) that vary from country to country, within different subcultural groups in a single nation (Barrio et al., 2003), and over time as communities undergo social changes. The "homogenization" of world culture may decrease these differences, but will not eliminate them. The nosological paradigms developed to categorize different types of psychotic symptoms are also embedded in specific professional cultures; these paradigms change as professional cultures evolve.

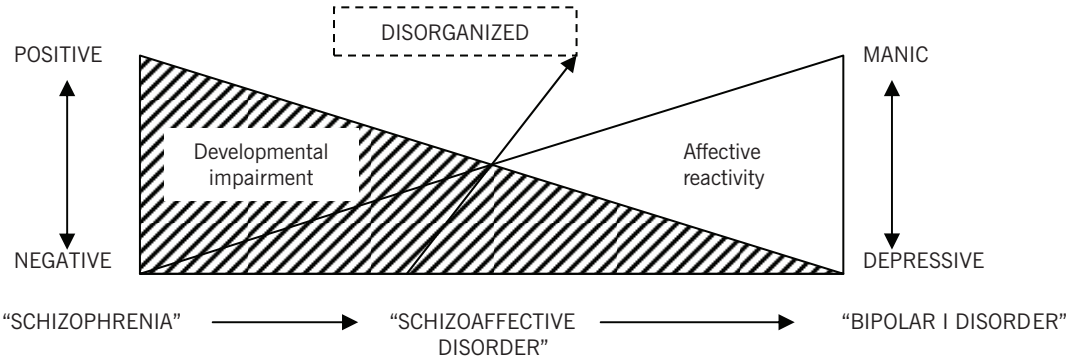


FIGURE 26.2. Schema incorporating five dimensions explaining the "spectrum" of syndromes from schizophrenia to bipolar I disorder.

The section "Ethnic and Cultural Considerations" in the introduction to DSM-IV explains why changes were introduced to facilitate the manual's use with culturally diverse individuals: "A clinician who is unfamiliar with the nuances of an individual's cultural frame of reference may incorrectly judge as psychopathology those normal variations in behavior, belief, or experience that are particular to the individual's culture" (American Psychiatric Association, 1994, p. xxiv). Awareness of cultural variation has made it clear that what is considered delusional ideation or bizarre behavior in one culture may be accepted as normal in another (Lu, Lim, & Mezzich, 1995). For example, in some communities, being "visited" by or "seeing" a recently deceased person is not unusual among family members, and experiencing "voices" or "visions" of religious figures are part of normal religious experience for some cultural groups. Therefore, interpreting an experience as pathological can be a subtle process, and a clinician with a different cultural perspective from the patient might well make errors in this process.

There has been discussion about whether the higher rate of psychosis in the U.K. African Caribbean population than in the general U.K. population reflects a genuinely increased rate of illness or whether it indicates misdiagnosis. One reason why mainstream psychiatrists may have difficulty in placing African Caribbeans with psychosis in the correct diagnostic categories is that their own cultural background may influence their perception of the quality and severity of the patients' psychiatric symptoms (Mackin et al., 2006). For example, it has been suggested that the frequency with which African Caribbean patients present with mood-incongruent delusions may contribute to the high rates of schizophrenia diagnosed in this population (Kirov & Murray, 1999).

In one study, a Jamaican psychiatrist was asked to make diagnoses on African Caribbean inpatients at a London teaching hospital. The U.K. doctors diagnosed schizophrenia in 52% of patients and the Jamaican psychiatrist diagnosed schizophrenia in 55% of patients, but there was agreement on the diagnosis of schizophrenia in just 55% of patients (Hickling et al., 1999). The results were no different whether ICD or DSM was used. This suggests problems in the reliability of diagnosing schizophrenia, rather than racial bias in the application of diagnostic criteria (Sharpley et al., 2001).

One of the major problems in international psychiatry is that the substantial differences in the onset, course, and treatment response of psychotic disorders between developed and less developed countries identified in the International Pilot Study of Schizophrenia (Sartorius et al., 1996) have had little effect on the dominant theories of psychosis, which have all been developed in Western countries and based on data from developed countries. Although the majority of individuals with psychotic conditions live in lower-income countries, research from such places either is unknown or has been dismissed as methodologically weak by nosologists from higher-income countries. Furthermore, studies that identify acute remitting psychosis (Susser & Wanderling, 1994) in poorer countries have been largely disregarded by Western nosologists. It is often assumed that methodological problems produce such "aberrant" findings, and so no attempt is made to identify other, more complex explanations.

It is possible that transient functional psychoses with complete recovery are 10 times more common in non-Western cultures than in Western cultures (Castillo, 2003). Some cultural psychologists attribute this to different levels of tolerance and expectations in the different societies. In an egocentric, "developed" society, where people are seen as responsible for themselves, family and community members may withdraw from a patient or express criticism or hostility when the person does not live up to societal expectations. The patient is also treated as having a "biogenetic" and therefore "incurable" brain disease. On the other hand, support from extended families and closer community ties in sociocentric, "developing" societies may lead to greater tolerance and acceptance of a spiritual explanation for psychosis, which in turn may lead to a better prognosis for such patients. Surely these potential differences are worthy of attention in future research?

Studies have consistently shown differences in the manifestations of schizophrenia in developing and developed countries. For example, so far there has been no satisfactory explanation for the marked differences in catatonia and hebephrenia observed in both

the International Pilot Study of Schizophrenia (Sartorius et al., 1996) and the Outcome of Severe Mental Disorders study (Jablensky et al., 1992). Catatonia was found to be 10 times as common in developing countries, and hebephrenia more than 3 times as common. Despite these and other documented differences across nations, cross-cultural research remains biased toward inferring "universals" in mental disorder, as Kleinman (1987) noted more than 20 years ago. Perhaps such inferences are oversimplifications, and the true diversity of phenomenology should be acknowledged and given more credence in diagnostic classifications.

Conclusion

By taking a life course approach to the study of psychoses (including genetic factors, neurodevelopmental distinctions, and symptomatology), we have seen in this chapter that the current methods of classification used in DSM-IV and ICD-10 are not strictly scientifically valid, although they have some utility for practicing clinicians and researchers (Kendell & Jablensky, 2003). It might therefore be useful to retain the categorical structure as a useful working concept, but to hybridize it with a dimensional approach in DSM-V and ICD-11; this would introduce the benefit of increased explanatory power of clinical characteristics, without completely dismissing the traditional paradigm of the Kraepelinian dichotomy. Similarly, including a rating of developmental impairment may aid clinicians in understanding the longitudinal course of illness evolution, rather than considering a diagnosis as a cross-sectional perspective based only on the current clinical picture. Anything more radical than these proposed changes is likely to be premature, given the expectation of further advances in genetic, neurobiological, environmental, psychosocial, and cross-cultural research in the coming years.

References

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Andreasen, N. C., & Olsen, S. (1982). Negative v. positive schizophrenia: Definition and validation. *Archives of General Psychiatry*, *39*, 789–794.

Barrio, C., Yamada, A. M., Atuel, H., et al. (2003). A tri-ethnic examination of symptom expression on the Positive and Negative Syndrome Scale in schizophrenia spectrum disorders. *Schizophrenia Research*, *60*, 259–269.

Berner, P., Katschnig, H., & Lenz, G. (1982). Poly-diagnostic approach: A method to clarify incongruences among the classification of the functional psychoses. *Psychiatric Journal of the University of Ottawa*, *7*, 244–248.

Brockington, I. F. (1992). Schizophrenia: Yesterday's concept. *European Psychiatry*, *7*, 203–207.

Cannon, M., Caspi, A., Moffitt, T. E., et al. (2002). Evidence for early-childhood, pandevelopmental impairment specific to schizophreniform disorder: Results from a longitudinal birth cohort. *Archives of General Psychiatry*, *59*, 449–456.

Cardno, A. G., Rijsdijk, F. V., Sham, P. C., et al. (2002). A twin study of genetic relationships between psychotic symptoms. *American Journal of Psychiatry*, *159*, 539–545.

Cardno, A. G., Sham, P. C., Farmer, A. E., et al. (2002). Heritability of Schneider's first-rank symptoms. *British Journal of Psychiatry*, *180*, 35–38.

Castillo, R. J. (2003). Trance, functional psychosis, and culture. *Psychiatry*, *66*, 9–21.

Castle, D. J., Sham, P. C., Wessely, S., et al. (1994). The subtyping of schizophrenia in men and women: A latent class analysis. *Psychological Medicine*, *24*, 41–51.

Dikeos, D. G., Wickham, H., McDonald, C., et al. (2006). Distribution of symptom dimensions across Kraepelinian divisions. *British Journal of Psychiatry*, *189*, 346–353.

Done, D. J., Crow, T. J., Johnstone, E. C., et al. (1994). Childhood antecedents of schizophrenia and affective illness: Social adjustment at ages 7 and 11. *British Medical Journal*, *309*, 699–703.

Dutta, R., Greene, T., Addington, J., et al. (2007). Biological, life course, and cross-cultural studies all point toward the value of dimensional and developmental ratings in the classification of psychosis. *Schizophrenia Bulletin*, *33*, 868–876.

Endicott, J. (1982). Diagnostic criteria for schizo-

phrenia: Reliabilities and agreement between systems. *Archives of General Psychiatry*, *39*, 884–889.

Farmer, A. E., McGuffin, P., & Gottesman, I. I. (1984). Searching for the split in schizophrenia: A twin study perspective. *Psychiatry Research*, *13*, 109–118.

Farmer, A. E., Wessely, S., Castle, D., et al. (1992). Methodological issues in using a polydiagnostic approach to define psychotic illness. *British Journal of Psychiatry*, *161*, 824–830.

Geddes, J. R., Verdoux, H., Takei, N., et al. (1999). Schizophrenia and complications of pregnancy and labor: An individual patient data meta-analysis. *Schizophrenia Bulletin, 25*, 413–423.

Helzer, J. E., Brockington, I. F., & Kendell, R. E. (1981). Predictive validity of DSM-III and Feighner definitions of schizophrenia: A comparison with Research Diagnostic Criteria and CATEGO. *Archives of General Psychiatry*, *38*, 791–797.

Hickling, F. W., McKenzie, K., Mullen, R., et al. (1999). A Jamaican psychiatrist evaluates diagnoses at a London psychiatric hospital. *British Journal of Psychiatry*, *175*, 283–285.

Hultman, C. M., Sparen, P., Takei, N., et al. (1999). Prenatal and perinatal risk factors for schizophrenia, affective psychosis, and reactive psychosis of early onset: Case–control study. *British Medical Journal*, *318*, 421–426.

Jablensky, A., Sartorius, N., Ernberg, G., et al. (1992). Schizophrenia: Manifestations, incidence and course in different cultures. A World Health Organization ten-country study. *Psychological Medicine* (Monograph Suppl.), *20*, 1–97.

Jansson, L. B., & Parnas, J. (2007). Competing definitions of schizophrenia: What can be learned from polydiagnostic studies? *Schizophrenia Bulletin*, *33*, 1178–1200.

Kendell, R., & Jablensky, A. (2003). Distinguishing between the validity and utility of psychiatric diagnoses. *American Journal of Psychiatry*, *160*, 4–12.

Kendler, K. S. (1980). The nosologic validity of paranoia (simple delusional disorder): A review. *Archives of General Psychiatry*, *37*, 699–706.

Kirov, G., & Murray, R. M. (1999). Ethnic differences in the presentation of bipolar affective disorder. *European Psychiatry*, *14*, 199–204.

Kleinman, A. (1987). Anthropology and psychiatry: The role of culture in cross-cultural research on illness. *British Journal of Psychiatry*, *151*, 447–454.

Kraepelin, E. (1992). Die Erscheinungsformen des Irreseins [The manifestations of insanity]. *History of Psychiatry*, *3*, 509–529. (Original work published 1920)

Lu, F. H., Lim, R. F., & Mezzich, J. E. (1995). Issues in the assessment and diagnosis of culturally diverse individuals. In J. M. Oldham & M. B. Riba (Eds.), *Review of psychiatry* (pp. 477–510). Washington, DC: American Psychiatric Press.

MacCabe, J. H., Lambe, M., Cnattingius, S., et al. (2006). Academic achievement at age 16 has contrasting effects on risk of later bipolar disorder and schizophrenia. *Schizophrenia Research*, *81*(Suppl.), 4–5.

MacCabe, J. H., Lambe, M. P., Cnattingius, S., et al. (2007). Scholastic achievement at age 16 and risk of schizophrenia and other psychoses: A national cohort study. *Psychological Medicine*, *38*(8), 1–8.

Mackin, P., Targum, S. D., Kalali, A., et al. (2006). Culture and assessment of manic symptoms. *British Journal of Psychiatry*, *189*, 379–380.

Maslowski, J., van Jansen, R. D., & Mthoko, N. (1998). A polydiagnostic approach to the differences in the symptoms of schizophrenia in different cultural and ethnic populations. *Acta Psychiatrica Scandinavica*, *98*, 41–46.

Murray, R. M., Lewis, S. W., & Reveley, A. M. (1985). Towards an aetiological classification of schizophrenia. *Lancet*, *i*, 1023–1026.

Murray, R. M., O'Callaghan, E., Castle, D. J., et al. (1992). A neurodevelopmental approach to the classification of schizophrenia. *Schizophrenia Bulletin*, *18*, 319–332.

Murray, R. M., Sham, P., Zanelli, J., et al. (2004). A developmental model for similarities and dissimilarities between schizophrenia and bipolar disorder. *Schizophrenia Research*, *71*, 405–416.

Reichenberg, A., Weiser, M., Rabinowitz, J., et al. (2002). A population-based cohort study of premorbid intellectual, language, and behavioral functioning in patients with schizophrenia, schizoaffective disorder, and nonpsychotic bipolar disorder. *American Journal of Psychiatry*, *159*, 2027–2035.

Reichenberg, A., Weiser, M., Rapp, M. A., et al. (2005). Elaboration on premorbid intellectual performance in schizophrenia: Premorbid intellectual decline and risk for schizophrenia. *Archives of General Psychiatry*, *62*, 1297–1304.

Robins, E., & Guze, S. B. (1970). Establishment of diagnostic validity in psychiatric illness: Its application to schizophrenia. *American Journal of Psychiatry*, *126*, 983–987.

Sartorius, N., Gulbinat, W., Harrison, G., et al. (1996). Long-term follow-up of schizophrenia in 16 countries: A description of the International Study of Schizophrenia conducted by the World Health Organization. *Social Psychiatry and Psychiatric Epidemiology*, *31*, 249–258.

Sham, P. C., Castle, D. J., Wessely, S., et al. (1996). Further exploration of a latent class typology of schizophrenia. *Schizophrenia Research*, *20*, 105–115.

Sharpley, M., Hutchinson, G., McKenzie, K., et al. (2001). Understanding the excess of psychosis among the African-Caribbean population in England: Review of current hypotheses. *British Journal of Psychiatry*, Suppl. *40*, s60–s68.

Spitzer, R. L. (1985). DSM-III and the politics–science dichotomy syndrome: A response to Thomas E. Schacht's "DSM-III and the politics of truth." *American Psychologist*, *40*, 522–526.

Spitzer, R. L., Williams, J. B., & Skodol, A. E. (1980). DSM-III: The major achievements and an overview. *American Journal of Psychiatry*, *137*, 151–164.

Susser, E., & Wanderling, J. (1994). Epidemiology of nonaffective acute remitting psychosis vs. schizophrenia: Sex and sociocultural setting. *Archives of General Psychiatry*, *51*, 294–301.

van Os, J., Gilvarry, C., Bale, R., et al. (1999). A comparison of the utility of dimensional and categorical representations of psychosis: UK700 Group. *Psychological Medicine*, *29*, 595–606.

van Os, J., Jones, P., Lewis, G., et al. (1997). Developmental precursors of affective illness in a general population birth cohort. *Archives of General Psychiatry*, *54*, 625–631.

World Health Organization. (1992). *The ICD-10 classification of mental and behavioural disorders: Clinical descriptions and diagnostic guidelines*. Geneva: Author.

CHAPTER 27

The Interpersonal Nexus of Personality and Psychopathology

Aaron L. Pincus
Mark R. Lukowitsky
Aidan G. C. Wright

If personology and psychopathology are to become a full-fledged science and profession, rather than a piecemeal potpourri of miscellaneous observations and ideas, the overall and ultimate architecture of the science must be refashioned, that is, given a scaffold or framework within which its elements can be properly located and ultimately coordinated. For example, our official diagnostic system should not stand alone, unconnected to other relevant realms of our clinical or scientific discourse; that is, it should be anchored to a foundation of an empirically supportable theory.

—Millon (2005, pp. 529–530)

Clinical science requires greater coordination of its definitional theories of normality–abnormality and its systems for describing variation in expression of psychopathology. Earlier work has articulated how the "interpersonal tradition" (Pincus & Gurtman, 2006) in clinical psychology and psychiatry can serve as an integrative nexus for the definition and description of personality disorders, providing a common scientific basis for understanding the nature of normality and abnormality and for the practical tasks of clinical identification and communication (Pincus, 2005a, 2005b). To serve the goals of this book and begin to fulfill Millon's (2005) blueprint for a clinical science, the present chapter demonstrates how the interpersonal tradition also provides an integrative nexus for the study of personality and psychopathology more generally.

To begin, let us briefly consider the nature of the criteria sets found in DSM-IV and ICD-10. Consistent with the distinctions above, a set of diagnostic criteria can be interpreted as defining what a disorder is, or as providing a set of fallible indicators for determining when the disorder is present (Regier et al., 1998; Widiger & Trull, 1991). Unfortunately, DSM-IV criteria sets do not adequately serve either function (Clark, Livesley, & Morey, 1997; Widiger & Clark, 2000). For example, Widiger (1991) suggested that DSM Axis II diagnostic criteria sets tend to describe the phenomenology of individual differences in personality disorders, rather than fundamentally defining the pathology of personality disorders in relation to normal personality functioning. Similarly, Parker and colleagues (2002) suggested that DSM-IV Axis II criteria sets mix

and confuse aspects of personality dysfunction (which could serve to define and explain personality disorder) with descriptors of personality style (which serve to distinguish and portray individual differences in personality disorder phenomenology). McGlashan and colleagues (2005) found that DSM-IV criteria for borderline, obsessive–compulsive, avoidant, and schizotypal personality disorders included a mix of traits, attitudes, cognitions, and behaviors with differential levels of stability. Problems such as these are alleviated in a current proposal for personality disorders in DSM-V: Krueger, Skodol, Livesley, Shrout, and Huang (2008) argue for the inclusion of core descriptive elements of personality to be rated for each patient, independently of a diagnosis of personality disorder. Like the interpersonal nexus of personality disorders (Pincus, 2005b), such a system decouples definition of disorder and phenomenological description of disorder. We would argue that such a system could be appropriate for both personality disorders and Axis I symptom syndromes, and that the clinical science of psychopathology will benefit from the recognition that diagnostic criteria sets for all disorders are best viewed in the context of personality.

In this chapter, we outline the interpersonal tradition and in so doing present the interpersonal nexus of personality and psychopathology. Because phenomenological description of psychopathology is an important component of diagnosis, assessment, and treatment of psychopathology (Cain, Pincus, & Ansell, 2008), and because this volume strives to inform the DSM and ICD diagnostic systems, we have chosen to focus on advances in the interpersonal description of psychopathology and to embed extended discussions of interpersonal pathoplasticity and new developments in the description of personality consistency (i.e., intraindividual variability, behavioral signatures) within the overview of the interpersonal tradition that follows.

The Interpersonal Tradition: Origins and Scope

The origins of the interpersonal tradition in personality and clinical psychology are found in Sullivan's (1953a, 1953b, 1954, 1956, 1962, 1964) highly generative interpersonal theory of psychiatry, which considered interpersonal relations and their impact on the self-concept to be core emphases in understanding personality, psychopathology, and psychotherapy. The interpersonal legacy that emerged from Sullivan's work is now in its fourth generation and has dramatically evolved over a nearly 60-year history, increasing in level of theoretical integration, methodological sophistication, and scope. Whether referred to as the "interpersonal paradigm" (Wiggins, 2003), the "interpersonal system" (LaForge, 2004), or the "interpersonal tradition" (Pincus & Gurtman, 2006), this vital work is notably broad; yet it provides a coherent nomological net for the integrative study of personality (e.g., Wiggins & Broughton, 1985; Wright, Pincus, Conroy, & Elliot, 2009), personality assessment (e.g., Pincus & Gurtman, 2003), psychotherapy (e.g., Anchin & Pincus, in press; Pincus & Cain, 2008), symptom syndromes (e.g., Barrett & Barber, 2007; Hopwood, Clarke, & Perez, 2007; Horowitz, 2004), personality disorders (e.g., Benjamin, 1996; Horowitz & Wilson, 2005; Pincus, 2005a, 2005b), and health psychology and behavioral medicine (e.g., Gallo, Smith, & Cox, 2006; Lackner & Gurtman, 2004, 2005). Although this evolution is vital and ongoing, Sullivan's commitment to the study of interpersonal phenomena remains at the forefront of these developments. This allows the diverse efforts in the interpersonal tradition to be interconnected and reciprocally influential.

The history of the interpersonal tradition is itself a representation of the coordination of explanatory theory and descriptive taxonomy we have discussed, as the interpersonal theory of personality has a long and reciprocally influential history in research programs that have culminated in well-validated, empirically derived models and methods to describe individual differences in interpersonal functioning (Pincus, 1994; Pincus & Gurtman, 2006). Thus the interpersonal nexus of personality and psychopathology includes multiple methods to assess the fundamental interpersonal dimensions of "agency" and "communion" (Wiggins, 1991) and associated circumplex structural models (Benjamin, 1974; Wiggins, 1996), and it ties operational definitions of reciprocal interpersonal pro-

cesses (Benjamin, 1996; Kiesler, 1983) and patterns of intraindividual variability in interpersonal behavior (Moskowitz & Zuroff, 2004a, 2004b) directly to these models. An overview of the interpersonal nexus of personality and psychopathology is presented in Figure 27.1.

The interpersonal nexus can also enhance the explanatory implications of descriptive features of psychopathology through the application of contemporary interpersonal theory (Benjamin, 2003; Horowitz, 2004; Pincus, 2005a), which assumes that the interpersonal dimensions underlying its descriptive models and methods are continuous with normal and disordered functioning, and emphasizes the "interpersonal situation" as an integrative theoretical concept. To fully satisfy the theoretical and personological needs of definition, the interpersonal nexus must also articulate the developmental, motivational, and regulatory factors associated with disordered self-concepts and maladaptive patterns of relating to others, and account for the fluctuating severity of symptomatology. In our view, the interpersonal nexus of personality and psychopathology provides the architecture for coordination of psychological theory with the practical/empirical description of the phenomenology of mental disorders. Such coordination is needed for scientifically grounded and clinically useful classification, diagnosis, professional communication, and treatment planning.

The Interpersonal Tradition: Contemporary Assumptions

The interpersonal tradition is a nomological net (Pincus, 2005a; Pincus & Ansell, 2003; Pincus & Cain, 2008) that incorporates explicit efforts toward integration of interpersonal theory and cognitive theories (e.g., Locke & Sadler, 2007; Safran, 1990a, 1990b), attachment theory (e.g., Bartholomew & Horowitz, 1991; Florsheim, Henry, & Benjamin, 1996), psychodynamic theories (e.g., Benjamin, 1995; Benjamin & Friedrich, 1991; Heck & Pincus, 2001; Mitchell, 1988; Pincus, 2005a), evolutionary theory (e.g., Fournier, Zuroff, & Moskowitz, 2007; Hoyenga, Hoyenga, Walters, & Schmidt, 1998; Zuroff, Moskowitz, & Côté, 1999), and even psychophysiology and neurobiology (e.g., aan het Rot, Moskowitz, & Young, 2008; D'Antono, Moskowitz, Miners, & Archambault, 2005; Moskowitz, Pinard, Zuroff, Annable, & Young, 2001). This integrative nature was best described by Horowitz and colleagues (2006):

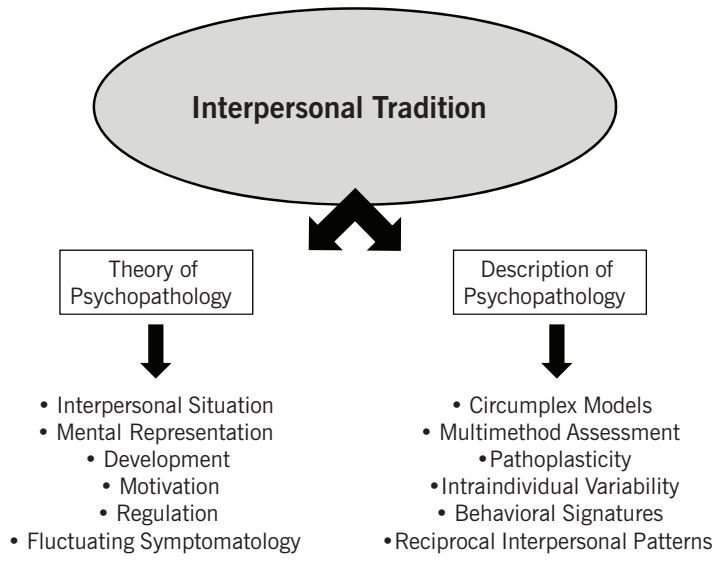


FIGURE 27.1. The interpersonal tradition provides an integrative nexus to coordinate psychological theory and description of personality and psychopathology.

> Because the interpersonal approach harmonizes so well with all of these theoretical approaches, it is integrative: It draws from the wisdom of all major approaches to systematize our understanding of interpersonal phenomena. Although it is integrative, however, it is also unique, posing characteristic questions of its own. (p. 82)

Virtually all theories of psychopathology touch upon interpersonal functioning, and contemporary interpersonal theory simply asserts that when we look at a domain of personality or its substrates in relation to psychopathology, our best bet may be to look at it in relation to interpersonal functioning. Thus contemporary interpersonal theory is also a nexus for bringing together elements across the theoretical spectrum, and it can serve as an integrative theoretical framework via consideration of the "interpersonal situation."

The Interpersonal Situation

> I had come to feel over the years that there was an acute need for a discipline that was determined to study not the individual organism or the social heritage, but the interpersonal situations through which persons manifest mental health or mental disorder. (Sullivan, 1953b, p. 18)

> Personality is the relatively enduring pattern of recurrent interpersonal situations which characterize a human life. (Sullivan, 1953b, pp. 110–111)

Sullivan's emphasis on the interpersonal situation as the focus for understanding both personality and psychopathology set an elemental course for psychiatry and clinical psychology. Contemporary interpersonal theory thus begins with the assumption that the most important expressions of personality and psychopathology occur in phenomena involving more than one person (see Table 27.1). Sullivan (1953a, 1953b) suggested that individuals express integrating tendencies that bring them together in the mutual pursuit of satisfactions (generally a large class of biologically grounded needs), security (i.e., anxiety-free functioning), and self-esteem. These integrating tendencies develop into increasingly complex "dynamisms," or patterns of interpersonal experience. From infancy throughout the lifespan, these dynamisms are encoded in memory via age-appropriate social learning. According to Sullivan, interpersonal learning of self-concept and social behavior is based on an anxiety gradient associated with interpersonal situations. All interpersonal situations range from rewarding (highly secure, esteem-promoting) through various degrees of anxiety (insecurity, low self-esteem), and end in a class of situations associated with such severe anxiety that they are dissociated from experience. The interpersonal situa-

TABLE 27.1. Contemporary Assumptions and Corollaries of the Interpersonal Tradition

Assumption 1

The most important expressions of personality and psychopathology occur in phenomena involving more than one person (i.e., interpersonal situations).

- An "interpersonal situation" can be defined as "the experience of a pattern of relating self with other associated with varying levels of anxiety (or security) in which learning takes place that influences the development of self-concept and social behavior" (Pincus & Ansell, 2003, p. 210).

Assumption 2

Interpersonal situations occur between proximal interactants *and* within the minds of those interactants via the capacity for perception, mental representation, memory, fantasy, and expectancy.

Assumption 3

Agency and communion provide an integrative metastructure for conceptualizing interpersonal situations.

- Explicatory systems derived from agency and communion can be used to describe, measure, and explain normal and pathological interpersonal motives, traits, and behaviors.
- Such systems can be applied to both proximal interpersonal situations *and* internal interpersonal situations.

Assumption 4

Interpersonal complementarity is most helpful if considered a common baseline for the field-regulatory pulls and invitations of interpersonal behavior.

- Chronic deviations from complementarity may be indicative of psychopathology.

tion underlies genesis, development, maintenance, and mutability of personality and psychopathology through the continuous patterning and repatterning of interpersonal experience in an effort to increase security and self-esteem (positively reinforcing) while avoiding anxiety (negatively reinforcing). Over time, this process gives rise to schematic representations of self and others (Sullivan's "personifications"), as well as to enduring patterns of adaptive or disturbed interpersonal relating.

Individual variation in learning occurs due to the interaction between a developing person's level of cognitive maturation and the characteristics of the interpersonal situations encountered. Interpersonal experience is understood differently, depending on the developing person's grasp of cause-and-effect logic and the use of consensual symbols such as language. This affects how the person makes sense of the qualities of significant others (including their "reflected appraisals," which communicate approval or disapproval of the person), as well as the ultimate outcomes of interpersonal situations characterizing a human life. Pincus and Ansell (2003) have summarized Sullivan's concept of the interpersonal situation as "the experience of a pattern of relating self with other associated with varying levels of anxiety (or security) in which learning takes place that influences the development of self-concept and social behavior" (p. 210). In one way or another, all perspectives on personality, psychopathology, and psychotherapy within the interpersonal tradition address elements of the interpersonal situation. These elements include individual differences, reciprocal interpersonal patterns of behavior, internal psychological processes, and the transactional and contextual frameworks for understanding interpersonal relations that we review in this chapter.

Proximal and Internal Interpersonal Situations

A potential misinterpretation of the term "interpersonal" is to assume that it refers to a limited class of phenomena that can be observed only in the immediate interaction between two proximal people. A review of Sullivan's body of work clearly reveals that this dichotomous conception of the interpersonal and the intrapsychic as two sets of distinct phenomena—one residing between people and one residing within a person—is an incorrect interpretation (Mitchell, 1988; Pincus, 2005a, 2005b; Pincus & Ansell, 2003; Pincus & Gurtman, 2006). From his emphasis on the interpersonal sources of the self-concept to his conceptions of personifications and parataxic distortions, Sullivan clearly viewed the interpersonal situation as equally likely to be found within the mind of the person as it is to be found in the observable interactions between two people. In fact, Sullivan (1964) defined psychiatry as the "study of the phenomena that occur in configurations made up of two or more people, all but one of whom may be more or less completely illusory" (p. 33). In contemporary interpersonal theory,

> The term *interpersonal* is meant to convey a sense of primacy, a set of fundamental phenomena important for personality development, structuralization, function, and pathology. It is not a geographic indicator of locale: It is not meant to generate a dichotomy between what is inside the person and what is outside the person. (Pincus & Ansell, 2003, p. 212)

This quotation makes it clear that interpersonal functioning occurs not only between people, but also inside people via the capacity for mental representation of self and others (e.g., Blatt, Auerbach, & Levy, 1997; Heck & Pincus, 2001). It also allows the contemporary interpersonal tradition to incorporate important pantheoretical representational constructs, such as cognitive interpersonal schemas, internalized object relations, and internal working models. Contemporary interpersonal theory does suggest that the most important personality and psychopathological phenomena are relational in nature, but it does not suggest that such phenomena are limited to contemporaneous, observable behavior. Interpersonal situations as defined by Pincus and Ansell (2003) occur both between proximal interactants and within the minds of those interactants. They occur in perceptions of contemporaneous events, memories of past experiences, and fantasies or expectations of future experiences. Regardless of the level of distortion or accuracy in these perceptions, memories, and fantasies, the ability to link

internal interpersonal situations and proximal interpersonal situations is crucial to the maturation and pantheoretical implications of the contemporary interpersonal tradition (Safran, 1992). Much of the field is moving in this direction, as the relationship between the interpersonal and the intrapsychic is a convergent focus of recent theoretical advances (Benjamin, 2003; Horowitz, 2004; Pincus, 2005a). Ultimately, both proximal and internal interpersonal situations continuously influence an individual's learned relational strategies and self-concept. Psychopathology is therefore inherently expressed via disturbed interpersonal relations (Sullivan, 1953b).

Agency and Communion: Integrative Metaconcepts

A major influence on the expansion and evolution of interpersonal perspectives on personality and psychopathology is Wiggins's (1991, 1997a, 2003) seminal review and integration of the interpersonal nature and relevance of Bakan's (1966) metaconcepts of "agency" and "communion." Wiggins argued that these two superordinate dimensions have propaedeutic explanatory power across fields as diverse as philosophy, linguistics, anthropology, sociology, psychiatry, and gender studies, as well as the subdisciplines of personality psychology, evolutionary psychology, cross-cultural psychology, social psychology, and clinical psychology. "Agency" refers to the condition of being a differentiated individual, and it is manifested in strivings for power and mastery, which can enhance and protect one's differentiation. "Communion" refers to the condition of being part of a larger social or spiritual entity, and is manifested in strivings for intimacy, union, and solidarity with the larger entity. Bakan (1966) noted that a key issue for understanding human existence is to comprehend how the tensions of this duality in the human condition are managed.

Wiggins (2003) proposed that agency and communion are most directly related to Sullivan's theory in terms of the goals of human relationship: security (communion) and self-esteem (agency). As can be seen in Figure 27.2, these "metaconcepts" (concepts about concepts) form a superordinate structure that can be used to derive explanatory and descriptive concepts at different levels of specificity. At the broadest and most interdisciplinary level, agency and communion serve to classify the interpersonal motives, strivings, and values of human relations (Horowitz, 2004). When motivation is considered in interpersonal situations, we may consider the agentic and communal nature of each individual's personal strivings or current concerns, or the more specific agentic and communal goals (e.g., to be in control, to be close) that specific behaviors are enacted to achieve (Horowitz & Wilson, 2005; Horowitz et al., 2006).

At more specific levels, the structure provides conceptual coordinates for describing and measuring interpersonal traits and behaviors (Wiggins, 1991). The intermediate level of traits includes an evolving set of interpersonal taxonomies of individual differences (Locke, 2006; Pincus & Gurtman, 2006). Agentic and communal traits and problems imply enduring patterns of perceiving, thinking, feeling, and behaving that are probabilistic in nature, and that describe an individual's interpersonal tendencies aggregated across time, place, and relationships. At the most specific level, the structure can be used to classify the nature and intensity of specific interpersonal behaviors (Moskowitz, 1994, 2005). Wiggins's theoretical analysis simultaneously allows for the integration of descriptive levels within the interpersonal tradition, as well as for expansion of the conceptual scope and meaning of interpersonal functioning. The interpersonal tradition in personality proposes that (1) agency and communion are the fundamental metaconcepts of personality, providing a superordinate structure for conceptualizing interpersonal situations; (2) explicatory systems derived from agency and communion can be used to understand, describe, and measure interpersonal traits and behaviors; and (3) such systems can be applied equally well to the objective description of contemporaneous interactions between two or more proximal individuals (e.g., Markey, Funder, & Ozer, 2003) and to interpersonal situations within the mind evoked via perception, memory, fantasy, and mental representation (e.g., Heck & Pincus, 2001).

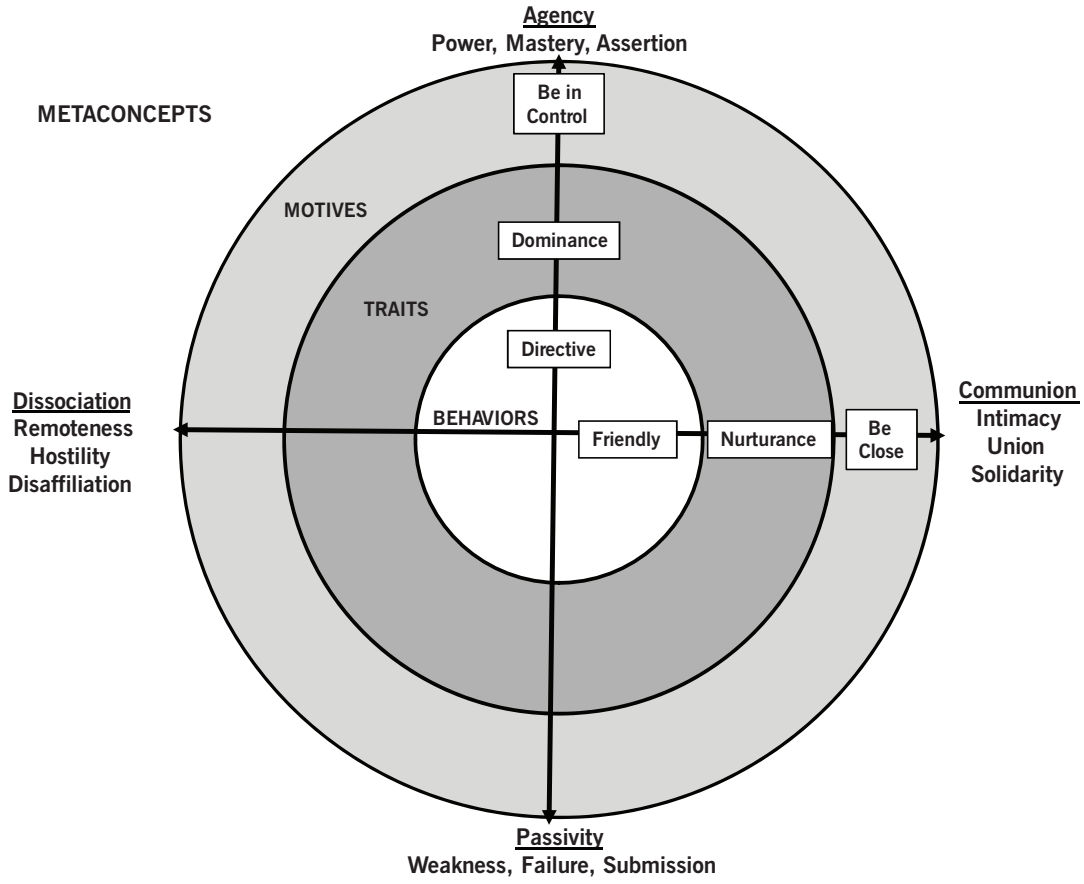


FIGURE 27.2. Agency and communion: Metaconcepts for the integration of interpersonal motives, dispositions, and behaviors.

Interpersonal Description of Individual Differences in Personality and Psychopathology

The emphasis on interpersonal functioning in Sullivan's work led to efforts to develop orderly and lawful conceptual and empirical models describing interpersonal behavior (for reviews of these developments, see LaForge, 2004; LaForge, Freedman, & Wiggins, 1985; Leary, 1957; Pincus, 1994; Wiggins, 1982, 1996). The goal of such work was to obtain an interpersonal taxonomy of dispositions and behaviors—that is, "to obtain categories of increasing generality that permit description of behaviors according to their natural relationships" (Schaefer, 1961, p. 126). In contemporary terms, these systems are referred to as "structural models," which can be used to conceptually systematize observation and covariation of variables of interest. When seen in relation to the metaconcepts of agency and communion, such models become part of an illuminating nomological net.

Empirical research into diverse interpersonal taxa—including traits (Wiggins, 1979), problems (Alden, Wiggins, & Pincus, 1990); values (Locke, 2000), impact messages (Kiesler, Schmidt, & Wagner, 1997), and verbal and nonverbal behaviors (Benjamin, 1974; Gifford, 1991; Kiesler, Goldston, & Schmidt, 1991; Moskowitz, 1994; Trobst, 2000)—converge in suggesting that the structure of interpersonal behavior takes the form of a circle or "circumplex" (Gurt-

man & Pincus, 2000; Locke, 2006; Wiggins & Trobst, 1997). An exemplar of this form, based on the two underlying dimensions of dominance–submission (agency) on the vertical axis and nurturance–coldness (communion) on the horizontal axis, is often referred to as the "Interpersonal Circle" (IPC; Kiesler, 1983; Pincus, 1994; Wiggins, 1996) (see Figure 27.3). The geometric properties of circumplex models give rise to unique computational methods for assessment and research (Gurtman, 1994, 1997, 2001; Gurtman & Balakrishnan, 1998; Gurtman & Pincus, 2003; Wright, Pincus, Conroy, & Hilsenroth, 2009) that are not reviewed here. In the present chapter, the IPC is used to coordinate phenomenological descriptions and theoretical concepts.

The IPC model is a geometric representation of individual differences in a variety of interpersonal domains; thus all qualities of individual differences within these domains can be described as blends of the circle's two underlying dimensions. Blends of dominance and nurturance can be located along the 360° perimeter of the circle. Interpersonal qualities close to one another on the perimeter are conceptually and statistically similar; qualities at 90° are conceptually and statistically independent; and qualities 180° apart are conceptual and statistical opposites. Whereas the circular model itself is a continuum without beginning or end (Carson, 1996; Gurtman & Pincus, 2000), any segmentalization of the IPC perimeter to identify lower-order taxa is potentially useful within the limits of reliable discriminability. The IPC has been segmentalized into sixteenths (Kiesler, 1983), octants (Wiggins, Trapnell, & Phillips, 1988), and quadrants (Carson, 1969).

Intermediate-level structural models derived from agency and communion focus on the description of distinctive consistencies of the individual (e.g., personality traits, interpersonal problems, interpersonal values), which, when understood in relation to their motives and goals, are assumed to give rise to adaptive and maladaptive behavior that is generally consistent across interpersonal situations (Horowitz & Wilson, 2005; Wiggins, 1997b). Thus we can use circumplex models to describe a person's typical ways of relating to others and refer to his or her

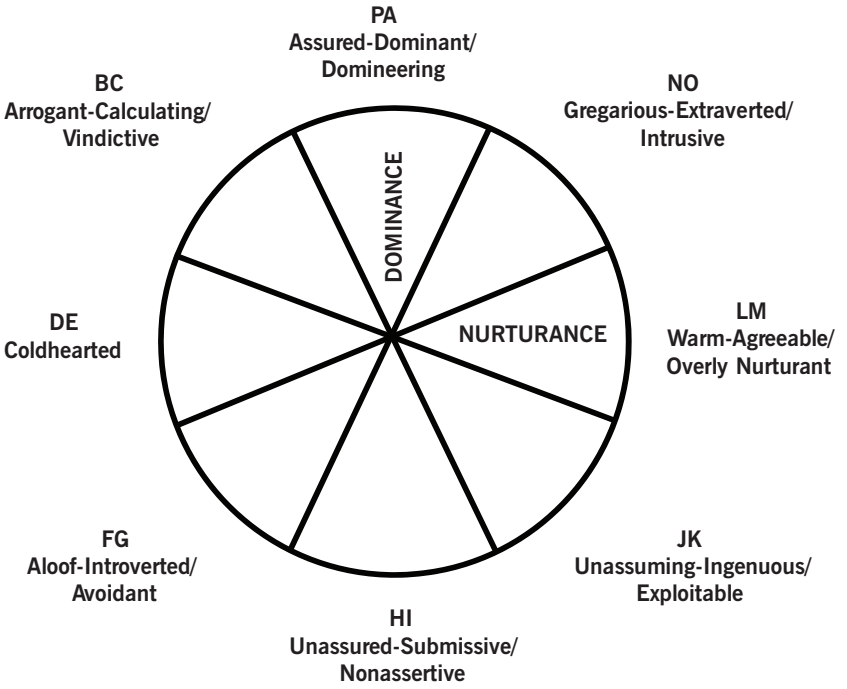


FIGURE 27.3. Circumplex model of interpersonal traits and interpersonal problems.

"interpersonal style." At the level of behavior, interpersonal description permits microanalytic, or transactional, analyses of interpersonal situations. Because interpersonal situations also occur within the mind, these models can also describe the person's typical ways of encoding new interpersonal information, as well as his or her consistent mental representations of self and others. Using circumplex models to classify individuals in terms of their agentic and communal characteristics is often referred to as "interpersonal diagnosis" (Benjamin, 1996; Leary, 1957; McLemore & Benjamin, 1979; Pincus & Wright, in press; Wiggins, Phillips, & Trapnell, 1989).

The central individual difference variable here is the "personality trait"—as typically defined, an enduring, dispositional attribute of the individual expressed in distinctive patterns of thought, behavior, and feeling. As McAdams (1995) points out, traits typically describe individual differences at a fairly broad or general level; they are inherently "decontextualized" and relatively "nonconditional" (p. 365). Hence the variables of interest here are assumed to reflect general features of the person's tendencies (e.g., "I am aggressive") that would presumably be relatively stable in time and found in an aggregate of interpersonal situations. Importantly, however, there are not one-to-one relationships between traits and behaviors; this means that the interpersonal meaning of a given behavior is ambiguous without consideration of the person's motive or goal in that interpersonal situation (Horowitz et al., 2006). Thus a certain trait or behavior (whether adaptive or maladaptive) may not necessarily be expressed in a particular interpersonal situation, relationship, or episode, or may not dictate a particular emergent process. For this level of specificity, contemporary interpersonal theory relies on additional theoretical constructs.

Interpersonal Extremity and Rigidity

Implications of the IPC model for individual differences in pathological functioning were initially based on the concepts of "behavioral intensity" (i.e., enacting behaviors in extreme forms) and "interpersonal rigidity" (i.e., displaying a limited repertoire of interpersonal behaviors). Disordered individuals tend to enact or rely on a limited or restricted range of behaviors, failing to adapt or conform their behaviors to the particular demands of a given interpersonal situation. From a circumplex perspective, they tend to draw from a small segment of the interpersonal circle, rather than to draw broadly as the situation requires. In a sense, rigidity can be construed as a kind of global interpersonal skill deficit (Gurtman, 1999). For example, research linking interpersonal traits and problems to particular personality disorder diagnoses (e.g., Pincus & Wiggins, 1990; Wiggins & Pincus, 1989) has implied that personality disorders, consistent with the DSM definition, are reflected in overly extreme and rigid agentic and communal expressions that cause impairment and/or subjective distress. Histrionic personality disorder is consistently associated with IPC octant NO, implying extreme extraversion that, when rigidly enacted, leads to intrusive interpersonal problems (e.g., "I want to be noticed too much"). In contrast, avoidant personality disorder is consistently associated with IPC octants FG and HI, implying extreme introversion and submissiveness that, when rigidly enacted, leads to avoidant and nonassertive interpersonal problems (e.g., "I find it hard to socialize with other people," "I find it hard to be self-confident when I am with other people"). In contrast, adaptivity has been assumed to be reflected in flexible expression of agentic and communal behavior around the circle at moderate levels of intensity, as called for by the particular interpersonal situation encountered. Interpersonally flexible individuals are capable of adjusting their behaviors appropriately to the cues of others in order to act effectively (see, e.g., Carson, 1991; Paulhus & Martin, 1987, 1988). Hence they are more likely to engage in mutually satisfying relationships (e.g., Kiesler, 1996).

However, we have found that these conceptions of adaptive and disordered interpersonal functioning provide only limited explanatory power, and they do not appear to be sufficient in scope to base an interpersonal *definition* of disorder. We believe that these concepts are better suited to description of individual differences in disordered behavior that may be observed in connection with various forms of psychopathology. This is because trait-like consistency is

probabilistic, and clearly even individuals with severe personality disorders vary in how consistently they behave and in what ways consistency is exhibited (e.g., Lenzenweger, Johnson, & Willett, 2004; McGlashan et al., 2005; Sansone & Sansone, 2008). Research suggests that the core phenomenology of only a subset of personality disorders may be substantially and uniquely interpersonal, and can be described by relatively extreme and rigid interpersonal styles (Horowitz et al., 2006). Specifically, the paranoid (BC—vindictive), schizoid (DE/FG—cold, avoidant), avoidant (FG/HI—avoidant, nonassertive), dependent (JK—exploitable), histrionic (NO—intrusive), and narcissistic (PA/BC—domineering, vindictive) personality disorders may be best described via their relatively unique interpersonal characteristics (see Figure 27.4). Beyond this, however, other personality disorders (e.g., borderline) and most symptom syndromes do not appear to present consistently with a single, prototypic interpersonal phenomenology. Thus, to take full advantage of the interpersonal nomological net, we must move beyond the overlapping associations between psychopathology and interpersonal characteristics implied by intensity and rigidity, to include both pathoplastic and dynamic associations. We turn to these associations next.

Advances in Interpersonal Description

Pathoplasticity

The interpersonal tradition asserts that maladaptive self-concepts and disturbed interpersonal relations are elements of the phenotypic presentation of all psychopathology. We suggest that using the architecture of the interpersonal nexus to account systematically for these elements provides additional valuable information beyond diagnosis itself for both planning treatment (e.g., Benjamin, 2005; Benjamin & Pugh, 2001; Pincus & Cain, 2008) and developing testable hypotheses regarding etiology and maintenance of psychopathology (Horowitz, 2004; Shechtman & Horowitz, 2006). A promising advance in this regard is the concept of

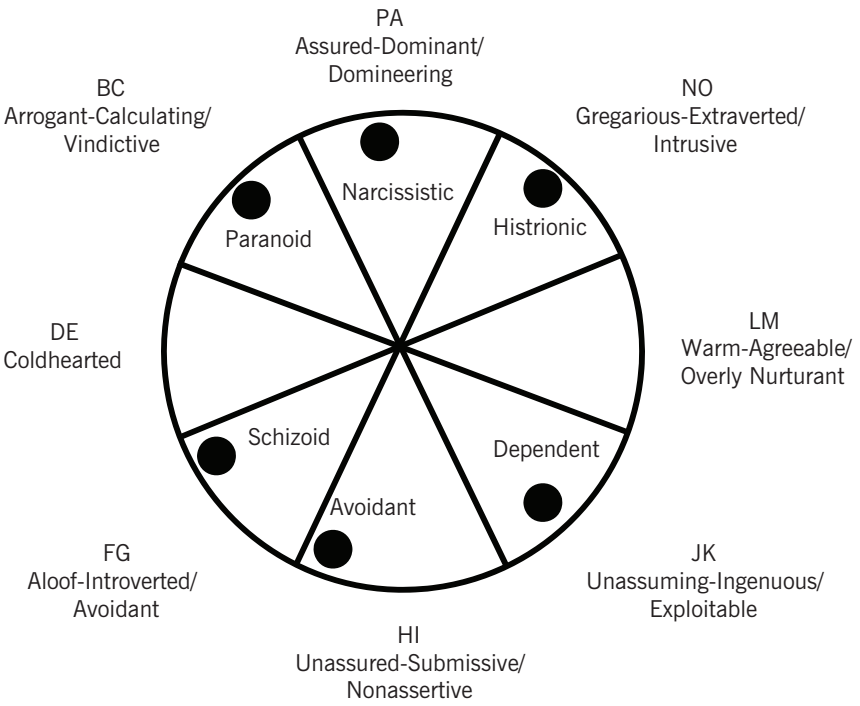


FIGURE 27.4. Interpersonal styles of six personality disorders.

interpersonal "pathoplasticity" in psychopathology.

The contemporary interpersonal tradition assumes a pathoplastic relationship between interpersonal functioning and some forms of psychopathology. Pathoplasticity is characterized by a mutually influencing nonetiological relationship between psychopathology and another psychological system (Klein, Wonderlich, & Shea, 1993; Widiger, Verheul, & van den Brink, 1999). Although it was initially conceptualized as a model relating personality and depression, its scope has been broadened to personality and psychopathology in general. Pathoplasticity recognizes that the expressions of certain maladaptive behaviors, symptoms, and mental disorders all occur in the larger context of an individual's personality (Millon, 1996, 2005), and points out that it would be unreasonable to assume that these expressions of pathology would not be influenced by the individual's characteristic manner of perceiving, thinking, feeling, behaving, and relating to the environment. Personality also has the potential for influencing the content and focus of a disorder, and is likely to shape the responses and coping strategies individuals employ when presented with a psychological stressor (Millon, 2000).[1]

Perhaps the best-known pathoplastic model in the clinical literature today, although it is not generally labeled as such, links differences in depression to individual differences in personality. Theorists from multiple theoretical perspectives have suggested similar models based on two "subtypes" of depression: "dependent/sociotropic/anaclitic" versus "self-critical/autonomous/introjective" (e.g., Beck, 1983; Blatt, 2004; see also Blatt & Luyten, Chapter 25, this volume). Regardless of labels, the two putative subtypes put forth by these theorists and others are remarkably similar to the basic interpersonal dimensions of communion and agency. The notion of two subtypes of depression proposes that the phenotype and phenomenology of the disorder differ as a function of an individual's personality. Specifically, there are those who suffer from a communal (dependent/sociotropic/anaclictic) type of depression characterized by feelings of loss and abandonment, in contrast to those who suffer from an agentic (self-critical/autonomous/introjective) type of depression characterized by feelings of failure, self-criticism, and worthlessness (Blatt & Zuroff, 1992). The differences in the way individuals of each personality type organize experience leave them vulnerable to particular psychological insults. Those who strongly value communal motives are most vulnerable to loss of significant others and relationships to death or abandonment, whereas those who strongly value agentic motives are most vulnerable to disapproval from others and failure (Horowitz, 2004). Blatt (1974) has also suggested that individuals of each personality type ward off the pain associated with their particular brand of depression in ways colored by their interpersonal strategies. A person with communal depression desperately pursues substitute relationships, while a person with agentic depression may become self-critical, or strive to maintain self-esteem by criticizing others.

Interpersonal pathoplasticity can describe the observed heterogeneity in phenotypic expression of psychopathology (e.g., Barrett & Barber, 2007), predict variability in response to psychotherapy within a disorder (e.g., Alden & Capreol, 1993; Borkovec, Newman, Pincus, & Lytle, 2002; Maling, Gurtman, & Howard, 1995), and account for a lack of uniformity in regulatory strategies displayed by those who otherwise are struggling with similar symptoms (e.g., Wright, Pincus, Conroy, & Elliot, 2009). Differences in interpersonal diagnosis will affect the manner in which patients express their distress and make bids for the type of interpersonal situation they feel is needed to regulate their self, affect, and relationships.

A number of investigations have found that individual differences in interpersonal problems exhibit pathoplastic relationships with pathological symptoms and mental disorders. For example, a series of studies examining generalized anxiety disorder (GAD) in Germany and the United States (*N*'s = 47, 83, and 78) have replicated evidence supporting interpersonal pathoplasticity, consistently identifying four distinct and prototypical interpersonal clusters within patients with DSM-IV-diagnosed GAD (Kasoff, 2002; Pincus et al., 2005; Salzer et al., 2008). These clusters, or subtypes, were labeled "nonassertive," "cold," "exploitable," and "intrusive" (see Figure 27.5), Patients with these subtypes of GAD

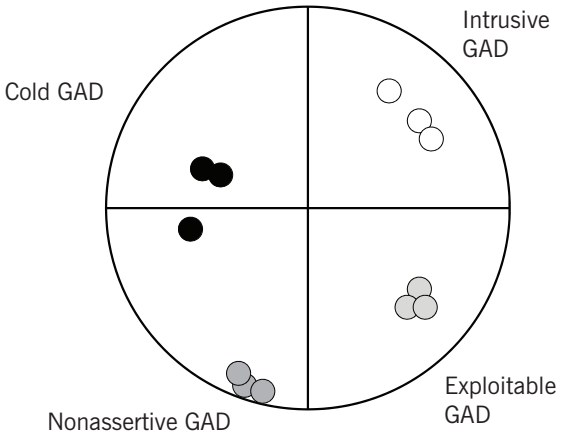


FIGURE 27.5. Interpersonal subtypes of generalized anxiety disorder (GAD), replicated in three samples.

reported distinctly different patterns of interpersonal problems, but exhibited no significant differences in symptom severity or psychiatric comorbidity. In the U.S. samples, individuals with these interpersonal subtypes exhibited no differences in attachment style, but they did vary in domains of worry content and controllability (Sibrava et al., 2007). Examination of treatment response in patients with these GAD subtypes has been limited to molar outcome measures for one sample ($N = 47$) of patients (Kasoff, 2002). Kasoff (2002) found that end-state functioning immediately after cognitive-behavioral treatment indicated that patients with nonassertive and exploitable GAD exhibited better adjustment than patients with cold and intrusive GAD. At 6-month follow-up, the functioning of patients with nonassertive and exploitable GAD continued to improve, whereas the functioning of patients with cold and intrusive GAD declined. Kasoff suggested that those with the submissive GAD subtypes had a better therapy outcome than those with the dominant GAD subtypes because of their personality compatibility with the patient–therapist role relationships in cognitive-behavioral therapy (Borkovec et al., 2002; Horowitz, Rosenberg, & Bartholomew, 1993; for divergent results, see also Puschner, Kraft, & Bauer, 2004). Thus these interpersonal subtypes of GAD seem to provide information beyond what is included in symptom measures. These subtypes may be useful not only for differential indication and individual case formulation, but also for explaining differences in therapy outcome.

DSM-IV social phobia is a disorder characterized by intense fear of social situations. Distinct groups of patients with social phobia have been identified, based on unique sets of interpersonal problems (Kachin, Newman, & Pincus, 2001). These groupings are associated with "vindictive" and "exploitable" interpersonal problems, respectively, and are not accounted for by symptom severity or psychiatric comorbidity. This suggests that these different groups, though diagnostically homogeneous, may react to the perceived threat of social situations in entirely different fashions. Maladaptive interpersonal response styles to threatening social situations may play a role in perpetuating the disorder by ensuring an unpleasant outcome (Carson, 1982). Pathoplasticity has implications for treatment planning with these patients beyond their primary Axis I diagnosis. Cain (2008) found that patients with exploitable social phobia showed significantly greater symptomatic improvement and satisfaction following Grawe's (2004) research-informed therapy than their colder counterparts did. In a related study, Alden and Capreol (1993) found that the effective treatment components for social anxiety in patients with avoidant personality disorder also differed, depending on their level of communion.

Whereas all patients exhibited significant nonassertive interpersonal problems, those whose nonassertiveness was colored by higher communion benefited most from intimacy-focused skills training, whereas those patients with lower communion benefited only from graduated exposure. Thus interpersonal pathoplasticity informs treatment planning beyond diagnosis by identifying different maladaptive behavior patterns in social situations that perpetuate negative outcomes (e.g., Benjamin, 2003, 2005).

This research has recently been extended beyond anxiety disorders. Disordered eating has also demonstrated a pathoplastic relationship with interpersonal problems (Hopwood et al., 2007). Women with bulimic symptoms could not be defined by one interpersonal style; instead, they varied broadly, suggesting that bulimia nervosa may be reciprocally related to a range of interpersonal difficulties. Even maladaptive traits can be more distinctly understood when personality features are considered. Slaney, Pincus, Uliaszek, and Wang (2006) found that maladaptive perfectionism is associated with interpersonal problems, whereas adaptive perfectionism is not. The interpersonal problems experienced by maladaptive perfectionists were not unitary, but were better described by two distinct clusters of perfectionists with prominently cold or exploitable interpersonal problems (Slaney et al., 2006). Finally, Wright, Pincus, Conroy, and Elliot (2009) demonstrated interpersonal pathoplasticity in individuals with high levels of fear of failure (FF). Conceptualized as an avoidance (vs. approach) achievement motive, FF is activated in situations where a failure to perform adequately is perceived to threaten an individual's ability to accomplish personally meaningful goals (Conroy, Willow, & Metzler, 2002), and sensitivity to shame motivates the individual to avoid failure (Atkinson, 1957; McGregor & Elliot, 2005). Two interpersonal subtypes of high FF were found to be associated with vindictive and nonassertive interpersonal problems, respectively, consistent with hypotheses derived from two distinct strategies for coping with elicited shame: rage versus appeasement (Wright, Pincus, Conroy, & Elliot, 2009).

Finally, some personality disorders may exhibit interpersonal pathoplasticity, although research is only beginning in this area. Leihener and colleagues (2003) found two interpersonal clusters of patients with borderline personality disorder (BPD): a primary cluster with dependency problems (JK—exploitable) and a secondary group with autonomy problems (PA—domineering). These clusters were replicated in a student sample exhibiting strong borderline features (Ryan & Shean, 2007). Leichsenring, Kunst, and Hoyer (2003) examined associations between interpersonal problems and borderline symptoms that may inform the interpersonal pathoplasticity of BPD. They found that primitive defenses and object relations were associated with controlling, vindictive, and cold interpersonal problems, whereas identity diffusion was associated with overly affiliative interpersonal problems. Narcissisitic personality disorder may also exhibit interpersonal pathoplasticity. A review of the phenotypic description of pathological narcissism across clinical theory, social and personality psychology, and psychiatric diagnosis revealed two themes of dysfunction: narcissistic grandiosity and narcissistic vulnerability (Cain et al., 2008; Pincus & Lukowitsky, in press). Grandiose narcissism is similar to the diagnostic criteria enumerated in DSM, and includes overt arrogance, exploitativeness, and inflated self-importance. In contrast, vulnerable narcissism is characterized by a depressive or shy presentation on the exterior, a heightened sensitivity to self-esteem wounds, and a covert grandiosity played out in fantasy. Therefore, these two very different interpersonal presentations share the same core narcissistic pathology, and even involve similar defenses at the core (e.g., grandiose fantasy), but vary in the interpersonal expression of their wounds and defensive mechanisms (Dickinson & Pincus, 2003; Miller & Campbell, 2008; Pincus et al., 2009; Zeigler-Hill, Clark, & Pickard, 2008).

Employing models of pathoplasticity in future DSMs and ICDs would require the diagnosis of disorders based on their defining features, followed by personality ratings for a given diagnosis (Krueger et al., 2008; Pincus, 2005b). We would argue strongly that such a system could be appropriate for both personality disorders and symptom syndromes, and should include assessment of agentic and communal personality features.

Intraindividual Variability and Behavioral Signatures

The addition of pathoplasticity greatly extends the clinical utility of interpersonal description of individual differences in personality and psychopathology. However, describing psychopathology in the context of varying dispositional personality concepts implying marked consistency of relational functioning is still insufficient and does not exhaust contemporary interpersonal descriptive approaches. Even patients described by a particular interpersonal style do not robotically emit the same behaviors without variation. Recent advances in the measurement and analysis of intraindividual variability (e.g., Eizenman, Nesselroade, Featherman, & Rowe, 1997; Heller, Watson, Komar, Min, & Perunovic, 2007; Moskowitz, 2005; Shoda, Mischel, & Wright, 1994) converge to warrant the assessment of temporal intraindividual variability of behavior and support a reconceptualization of personality consistency. This accumulating body of research suggests that individuals are characterized not only by their stable individual differences in trait levels of behavior, but also by stable differences in their variability in psychological states (Fleeson, 2001; Kernis, 2005), behaviors (Moskowitz & Zuroff, 2004a), and affect (Côté & Moskowitz, 1998; Eid & Diener, 1999) across time and situations.

Whereas evidence of variability was first interpreted as support of situational influences, contemporary views propose a comfortable coexistence of large within-person variability and large between-person stability in the study of personality (Fleeson, 2001; Fleeson & Leicht, 2006; Funder, 2006). That is, personality consistency is reflected in stability of behavior within situations and variability of behavior across situations. This increases the salience of contextual factors without losing the essence of personality itself. Thus variability in behavior across situations is no longer seen as error, but rather as meaningful information that characterizes individual differences in human behavior. The identification of stable "if . . . then . . . " behavioral signatures (Shoda, Mischel, & Wright, 1989, 1993a, 1993b, 1994) has thus become an important area of personality research (Funder, 2006; Mischel & Shoda, 1998). Importantly, this has been directly applied within the interpersonal tradition by the assessment of interpersonal behavior contextualized within interpersonal situations, both based on the common metric of agentic and communal dimensions (Fournier, Moskowitz, & Zuroff, 2008). That is, the most important contextual factors of proximal or internal interpersonal situations are the agentic and communal characteristics of the person or persons to whom one is relating.

Contemporary models integrating persons, situations, and behaviors include the cognitive–affective personality system (Mischel & Shoda, 1995), knowledge-and-appraisal personality architecture (Cervone, 2004), the state density distributions approach (Fleeson, 2001; Fleeson & Leicht, 2006), and latent state–trait theory (Steyer, Schmitt, & Eid, 1999). At varying levels of specificity, these models all employ intrapersonal perceptual and meaning-making processes (e.g., explicit cognitive and affective subsystems are often proposed). Conceptualizing and measuring patterns of variability and stability of interpersonal behavior over time and across situations is an important development for interpersonal description of personality and psychopathology that has the potential to enhance the sophistication of our current diagnostic systems (Pincus, 2005a). Variation in psychopathology may be characterized by stable patterns of variability in interpersonal behavior or by unique interpersonal behavioral signatures integrating the individual's agentic and communal motives with the perceived agentic and communal characteristics of others (Fournier et al., 2008; Horowitz, 2004).

INTERPERSONAL FLUX, PULSE, AND SPIN

Moskowitz and Zuroff (2004a, 2004b) introduced the terms "flux," "pulse," and "spin" to describe the stable levels of intraindividual variability in interpersonal behaviors sampled from the interpersonal circumplex (see Figure 27.6). "Flux" refers to variability around an individual's mean behavioral score on agentic or communal dimensions (e.g., dominant flux, submissive flux, friendly flux, hostile flux); "spin" refers to variability of the angular coordinate around the individual's mean interpersonal style; and "pulse" refers to variability of the

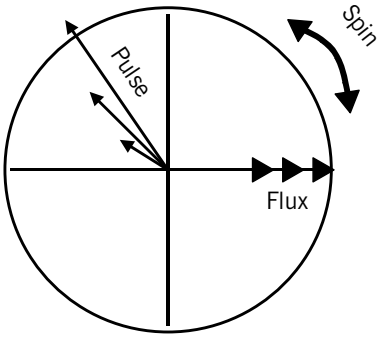


FIGURE 27.6. Interpersonal flux, pulse, and spin.

overall extremity of the emitted behavior. Low spin thus reflects a narrow repertoire of interpersonal behaviors enacted over time, and in traditional terms would be considered high interpersonal rigidity. Low pulse reflects little variability in behavioral intensity, and if it were associated with a high mean intensity generally, it would be consistent with the enactment of consistently extreme interpersonal behaviors. This dynamic lexicon has important implications for the assessment of normal and abnormal behavior. Theoretical analyses, as well as empirical results, suggest that assessments of intraindividual variability and interpersonal behavioral signatures extend conceptions of interpersonal extremity and rigidity, and offer unique and important new methods for the description of psychopathology. These advances deepen the personality context for understanding of symptomology.

IMPLICATIONS FOR PERSONALITY DISORDERS

The constructs of flux, pulse, and spin have significant implications for an interpersonal description of personality disorders. Pincus (2005a) has proposed using intraindividual variability to differentiate the phenomenological expression of personality pathology, with dependent and narcissistic personality disorders as examples (see Figure 27.7). On the basis of previous research demonstrat-

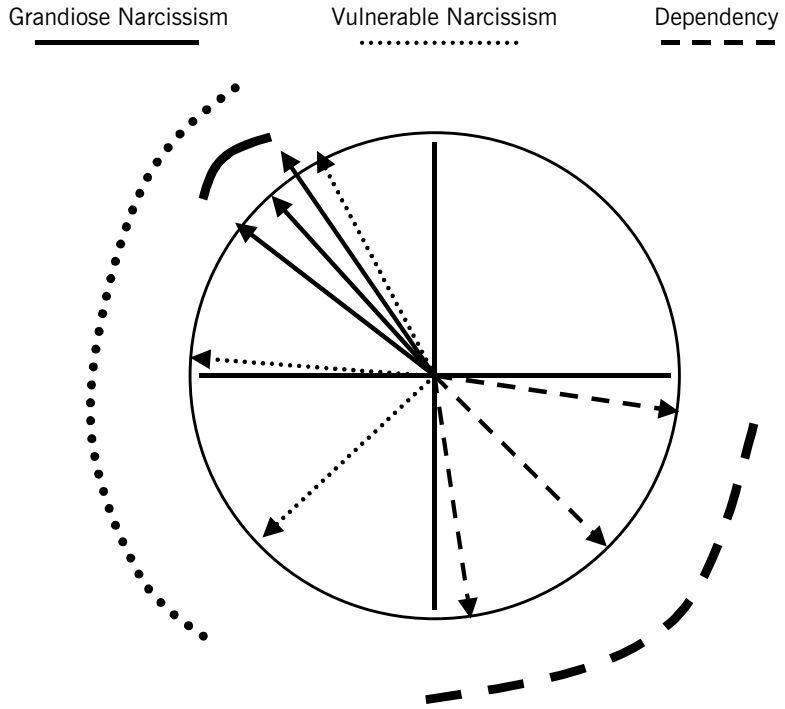


FIGURE 27.7. Interpersonal spin and pulse in grandiose narcissism, vulnerable narcissism, and dependency.

ing that dependency is associated with the entire range of friendly submissive interpersonal functioning (Pincus, 2002; Pincus & Gurtman, 1995; Pincus & Wilson, 2001), dependent personality can be represented by a limited amount of spin centered in the friendly submissive quadrant of the circumplex and low pulse around an extreme mean level. Pincus (2005a) has also used spin and pulse to theoretically differentiate "grandiose" and "vulnerable" narcissism (Cain et al., 2008). Previous research suggests that persons with grandiose narcissism tend to fall into the PA and BC octants of the IPC, characterized by domineering, vindictive, and arrogant behaviors (e.g., Dickinson & Pincus, 2003; Pincus & Wiggins, 1990). Furthermore, they tend to enact this style of behavior rigidly in relation to others, and to exhibit extreme behaviors that include narcissistic rage and chronic devaluing of others. In terms of intraindividual variability, persons with grandiose narcissism assessed across situations and across extended periods of time are likely to demonstrate a low amount of spin reflecting high agency and somewhat low communion, and low pulse around an extreme mean level suggesting limited variability in their behavior. In contrast, those with vulnerable narcissism tend to be much more labile in their interpersonal style and behaviors. Likewise, their sense of self often vacillates between grandiose and depressive psychological states and between vindictive and avoidant interpersonal behaviors (Cain et al., 2008). Thus, in contrast to persons with grandiose narcissism, Pincus (2005a) has suggested that those with vulnerable narcissism are likely to exhibit greater spin and to demonstrate variability ranging from hostile dominant behaviors during episodes of grandiosity to hostile submissive behaviors during episodes of self-depletion. In both self-states, persons with vulnerable narcissism are likely to exhibit low pulse around high mean behavioral intensity.

The emotional and behavioral lability that characterizes individuals with BPD has also been conceptualized in terms of intraindividual variability. For example, Hurt (2002) demonstrated the specific role of intraindividual variability in the diagnostic criteria for BPD in a sample of incarcerated female felons. Hopwood and Morey (2007) found that patients high in borderline features exhibited greater inconsistency in self-ratings of dominance and nurturance than patients without borderline features; they concluded that "variability, as well as mean scores, on the interpersonal dimensions may be important for conceptualizing some disorders" (p. 1097). Using the constructs of flux, pulse, and spin, Russell, Moskowitz, Zuroff, Sookman, and Paris (2007) differentiated individuals with BPD from nonclinical control participants on the basis of intraindividual variability in behavior over a 20-day period. Specifically, individuals with BPD reported a mean level of agreeable (communal) behavior similar to that of their nonclinical counterparts, but the participants with BPD displayed greater flux in their agreeable behaviors. This suggested that the control participants demonstrated consistent agreeable behavior across situations, while the individuals with BPD varied greatly in their agreeable behaviors, vacillating between high and low levels. Results also suggested elevated mean levels of submissive behaviors, in conjunction with low mean levels of dominant behavior coupled with greater flux in dominant behaviors, for the individuals with BPD relative to the control participants. However, the groups did not differ in the variability of submissive behaviors. In other words, individuals with BPD were consistently more submissive than the nonclinical controls were, but also demonstrated acute elevations and declines in their relatively low level of dominant behavior. Finally, as predicted, individuals with BPD endorsed higher mean levels of quarrelsome behavior and higher levels of flux in quarrelsome behavior than did controls. Individuals with BPD also demonstrated greater spin than their nonclinical counterparts, suggesting greater behavioral lability.

IMPLICATIONS FOR SYMPTOM SYNDROMES

Conceptualizing psychopathology in terms of intraindividual behaviors is not limited to personality disorders, although less theoretical rationale and research has been proposed for symptom syndromes. Consistent with the results suggesting greater spin in patients with BPD than in controls, Moskowitz and Zuroff (2004b) found that trait neuroticism was positively correlated with interpersonal spin. Consider Mineka, Watson, and Clark's

(1998) integrative hierarchical model of anxiety and depression. In this model, depression and anxiety share a common, higher-order factor of negative affect, and each disorder is differentiated by its own specific factor. This could suggest that high levels of negative affect, combined with individuals' agentic and communal motives and traits, could give rise to variable interpersonal behavior and unique behavioral signatures across interpersonal situations.

Consistent with the pathoplasticity model for GAD, Moskowitz and Zuroff (2004b) suggested that high levels of negative affect may lead individuals to experience interpersonal situations as threatening or dangerous and employ various interpersonal strategies to cope. For example, highly anxious individuals may try to cope with perceived interpersonal threats by arguing with others, by smiling and laughing in order to build closer connections to others, or by passively giving in to others. Moskowitz and Zuroff concluded that "trying a variety of behaviors to cope with frequent perceptions of interpersonal danger would contribute to spin, frequent switching among the interpersonal circumplex behaviors" (p. 143). One can also imagine that individuals with dysthymia may exhibit chronic passivity (i.e., low spin around intense submissiveness), leading to a failure to engage in agentic actions to change the circumstances and promote self-esteem (e.g., Horowitz & Vitkus, 1986). In contrast, individuals with bipolar disorders or impulse control disorders may exhibit a high amount of flux, pulse, and spin, contingent upon their mood states.

Interpersonal Reciprocity and Transaction

Interpersonal behavior is not emitted in an isolated vacuum; rather, it is reciprocally influential in ongoing human transaction. The notion of reciprocity in human relating has been reflected in a wide variety of psychological concepts (Pincus & Ansell, 2003). Within the interpersonal tradition, these have typically been referred to in terms of "adaptive and maladaptive transaction cycles" (Kiesler, 1991), "self-fulfilling prophecies" (Carson, 1982), and "vicious circles" (Millon, 1996). If we assume that an interpersonal situation involves two or more people relating to each other in ways that bring about social and self-related learning, this implies that something more is happening than mere random activity. Reciprocal relational patterns create an interpersonal field (Wiggins & Trobst, 1999) in which various transactional influences affect both interactants as they resolve, negotiate, or disintegrate the interpersonal situation. Within this field, an individual initiates interpersonal behaviors in order to elicit, invite, or evoke "restricted classes" of responses from the other, and this is a continual, dynamic transactional process. Thus contemporary interpersonal theory emphasizes "field-regulatory" processes in addition to "self-regulatory" or "affect-regulatory" processes (Mitchell, 1988; Pincus, 2005a; Sullivan, 1948).

The concept of interpersonal behavioral signatures places the interpersonal tradition's view of reciprocity and field regulation in a new and important light. Adaptive and maladaptive transaction cycles are the proximal and internal interpersonal situations of interest for describing personality and psychopathology. The most salient psychological features of the interpersonal situation are the agentic and communal characteristics of the interactants, and the consistent patterns described reflect behavioral signatures associated with perception of these characteristics (Fournier et al., 2008). The IPC provides conceptual anchors and a lexicon to systematically describe the patterned regularity of reciprocal interpersonal processes. The most basic of these processes is referred to as interpersonal "complementarity" (Carson, 1969; Kiesler, 1983). Interpersonal complementarity occurs when there is a match between the field-regulatory goals of each person. That is, reciprocal patterns of activity evolve in which the agentic and communal motives of both persons are fulfilled in the interpersonal situation, leading to stability and likely recurrence of the pattern (Horowitz et al., 2006). Carson (1969) first proposed that complementarity can be defined via the IPC. He proposed that complementarity is based on the social exchange of status (agency) and love (communion), as reflected in reciprocity for the vertical dimension (i.e., dominance pulls for submission, submission pulls for dominance) and correspondence for the horizontal dimension (friendliness pulls

for friendliness, hostility pulls for hostility). Kiesler (1983) extended this by adapting complementarity beyond the cardinal points of the IPC (hostile dominance pulls for hostile submission, friendly dominance pulls for friendly submission, etc.).

Although complementarity neither is the only reciprocal interpersonal pattern that can be described by the IPC nor has been proposed as a universal law of interaction, empirical studies consistently find support for its probabilistic predictions (e.g., Gurtman, 2001; Markey et al., 2003; Sadler & Woody, 2005; Tracey, 1994; Woody & Sadler, 2003). This leads to the fourth contemporary assumption of the interpersonal tradition. Complementarity is most helpful if considered a common baseline for the field-regulatory pulls and invitations of interpersonal behavior. If used in this way, chronic deviations from complementary reciprocal patterns may be indicative of pathological functioning. For example, GAD or social phobia may impair relational functioning by reducing a patient's ability to provide complementary responses in interpersonal situations. If many interpersonal situations are misinterpreted as threatening, the need to reduce the threat prioritizes motivation, rather than an accurate encoding of the interpersonal bids reflecting the agentic and communal motives of the other. Alternatively, the extreme agentic motives of persons with grandiose narcissism lead to low interpersonal spin, limiting their behavioral repertoire and thus their ability to engage in complementary relations across interpersonal situations. The high spin of patients with BPD may also impair complementary relations, as massive shifts in their concepts of self and other color their ability to mutually satisfy agentic and communal motives consistently.

The two other broad classes of reciprocal interpersonal patterns anchored by the IPC model are referred to as "acomplementary" and "anticomplementary" patterns (Kiesler, 1983, 1996). When reciprocal interpersonal patterns meet one of the two rules of complementarity, this is referred to as an acomplementary pattern. In such a case, interactants may exhibit correspondence with regard to nurturance or reciprocity with regard to dominance, but not both. When interactants exhibit neither reciprocity on dominance nor correspondence on nurturance, this is referred to as an anticomplementary pattern. The patterned regularity in human transaction directly affects the outcomes of interpersonal situations by satisfying or frustrating individuals' agentic and communal motives (Horowitz, 2004; Horowitz et al., 2006). Complementary reciprocal patterns are considered to promote relational stability; that is, such interpersonal situations are resolved, mutually reinforcing, and recurring. Acomplementary patterns are less stable and instigate negotiation toward or away from greater complementarity. Finally, anticomplementary patterns are the most unstable and may lead to avoidance, escape, and disintegration of the interpersonal situation (i.e., disturbed interpersonal relations).

Interpersonal complementarity (or any other reciprocal pattern) should not be conceived of as some sort of stimulus–response process based solely on overt actions and reactions (Pincus, 1994). A comprehensive account of the contemporaneous interpersonal situation must somehow bridge the gap between the proximal interpersonal situation and the internal interpersonal situation (e.g., Safran, 1992). Kiesler's (1986, 1988, 1991, 1996) "interpersonal transaction cycle" is the most widely applied framework to describe the relations among proximal and internal interpersonal behavior within the interpersonal tradition. He proposes that the basic components of an interpersonal transaction are (1) person *X*'s covert experience of person *Y*, (2) person *X*'s overt behavior toward person *Y*, (3) person *Y*'s covert experience in response to person *X*'s action, and (4) person *Y*'s overt behavioral response to person *X*. These four components are part of an ongoing transactional chain of events cycling toward resolution, further negotiation, or disintegration. Within this process, overt behavioral output serves the purpose of regulating the proximal interpersonal field via elicitation of complementary overt responses in the other. The IPC specifies the range of descriptive taxa, while the motivational conceptions of interpersonal theory give rise to the nature of regulation of the interpersonal field. For example, dominant interpersonal behavior (e.g., "You have to call your mother") communicates a bid for status (e.g., "I am in charge here"); this affects the other person in ways that elicit ei-

ther complementary (e.g., "You're right, I should do that now") or noncomplementary (e.g., "Quit bossing me around!") responses in an ongoing cycle of reciprocal causality, mediated by internal subjective experiences, motives, and regulatory needs.

Parataxic Distortions

Sullivan (1953b) proposed the concept of "parataxic distortion" to describe the mediation of proximal relational behavior by internal, covert factors, and suggested that these occur "when, beside the interpersonal situation as defined within the awareness of the speaker, there is a concomitant interpersonal situation quite different as to its principle integrating tendencies, of which the speaker is more or less completely unaware" (p. 92). The effects of parataxic distortions on interpersonal relations can occur in several forms, including chronic distortions of new interpersonal experiences (input); generation of rigid, extreme, and/or chronically nonnormative interpersonal behavior (output); and dominance of affect or self-regulation goals, leading to the disconnection of interpersonal input and output (such as the prior examples for GAD, social phobia, BPD, and narcissistic personality disorder).

Normality and psychopathology may be differentiated by their enduring tendencies to organize interpersonal experience in particular ways, leading to integrated or disturbed interpersonal relations (Pincus, Lukowitsky, Wright, & Eichler, 2009). Contemporary interpersonal theory proposes that healthy interpersonal relations are promoted by the capacity to organize and elaborate incoming interpersonal input in generally undistorted ways, allowing for the mutual needs of self and other to be met. That is, the proximal interpersonal situation and the internal interpersonal situation are relatively consistent (i.e., free of parataxic distortion). Maladaptive interpersonal functioning is promoted when the proximal interpersonal situation is encoded in distorted or biased ways, leading to behavior (output) that disrupts interpersonal relations due to conflicting or disconnected field-regulatory influences and the chronic frustration of agentic and communal motives of self and other. In the psychotherapy context, this can be identified by a preponderance of acomplementary and anticomplementary cycles of transaction between therapist and patient (Kiesler, 1988). Such therapeutic experiences are very common in the treatment of personality disorders (Anchin & Pincus, in press) and certainly not uncommon in the treatment of significant symptom syndromes.

Development, Motivation, and Regulation

Although such an analysis is not the emphasis of the present chapter, a comprehensive theory of personality and psychopathology goes beyond description to include a developmental analysis emphasizing historical origins and the continuing significance of past experience on current functioning (Millon, 1996). Significant contemporary developments in the interpersonal tradition include developmental learning and loving (DLL) theory (Benjamin, 1993, 1996, 2003), the revised interpersonal model (Horowitz, 2004; Horowitz & Wilson, 2005; Horowitz et al., 2006), and contemporary integrative interpersonal theory (CIIT; Pincus, 2005a, 2005b; Pincus & Ansell, 2003; Pincus & Cain, 2008; Pincus & Gurtman, 2006; Pincus & Wright, in press). We briefly distill important theoretical convergences around issues of development, motivation, and regulation.

Attachment and the Internalization of Interpersonal Experience

Contemporary interpersonal theorists agree that the first interpersonal situations are associated with infant attachment. Horowitz (2004) has proposed that the two fundamental tasks associated with the infant attachment system (staying close/connecting to caregivers, separating/exploring) are the first communal and agentic motives, respectively. According to attachment theory (Bowlby, 1969, 1973; Cassidy, 1999), repeated interactions become schematized interpersonal representations or "internal working models," which guide perception, emotion, and behavior in relationships. These processes lead to the development of secure or insecure attachment, which has significant implications for personality and psychopathology (Shorey & Snyder, 2006). Over time, these

generalize via adult attachment patterns associated with agentic and communal motives, traits, and behaviors. Horowitz (2004) has suggested that insecure attachment leads to significant self-protective motivations that can interfere with healthy agentic and communal functioning.

Similarly, according to Benjamin's (2003) DLL theory, attachment itself is the fundamental motivation that catalyzes social learning processes. Benjamin has proposed three developmental "copy processes," or ways in which early interpersonal experiences are internalized as a function of achieving attachment (be it secure or insecure). The first is "identification," which is defined as treating others as one has been treated. To the extent that an individual strongly identifies with early caretakers, there will be a tendency to act toward others in ways that copy how important others have acted toward the developing person. When the person does so, behavior in proximal interpersonal situations is associated with positive reflected appraisals of the self from the internal working model of the attachment figure. This mediates the perception of the proximal situation and may lead to repetition of such behavior, regardless of the field-regulatory pulls of the actual other (i.e., noncomplementary reciprocal patterns). The second copy process is "recapitulation," which is defined as maintaining a position complementary to an internalized other. This can be described as reacting "as if" the internalized other is still there. In this case, new interpersonal input is likely to be elaborated in a distorted way, such that the proximal other is experienced as similar to the internalized other; or the person may simply ignore new interpersonal input from the proximal other and focus field regulation on the dominant internalized other. This again may lead to noncomplementary reciprocal patterns in the proximal interpersonal situation, while complementary interpersonal patterns are played out in the internal interpersonal situation. The third copy process is "introjection," which is defined as treating the self as one has been treated, and is related to Sullivan's conceptions of "reflected appraisals" as a source of self-personification. When the self is treated in introjected ways, the internal interpersonal situation may promote security and esteem, even while generating noncomplementary behavior in the proximal interpersonal situation. In CIIT, Pincus and colleagues have extended the catalysts of internalization and social learning beyond attachment motives.

Catalysts of Internalization and Social Learning

Pincus and Ansell (2003) have proposed that "Reciprocal interpersonal patterns develop in concert with emerging motives that take developmental priority" (p. 223). These developmentally emergent motives may begin with the formation of early attachment bonds and felt security; later, however, separation–individuation, the experience of self-esteem and positive affects, development of gender identity, and resolution of Oedipal issues may become priorities. Later still, adult identity formation and its confirmation from the social world, as well as mastery of continuing unresolved conflicts, may take precedence. In addition to the achievement of emerging developmental goals, influential interpersonal patterns are associated with traumatic learning, stemming from the need to cope with impinging events (e.g., early loss of an attachment figure, childhood illness or injury, or physical or sexual abuse). The consequences of internalizing such experiences are an individual's consistently sought-after relational patterns and typical strategies for achieving them. These become the basis for the recurrent interpersonal situations that characterize a human life. If we are to understand the relational strategies individuals employ when such developmental motives or traumas are reactivated, we must learn what interpersonal behaviors and patterns were associated with achievement or frustration of particular developmental milestones or were required to cope with a trauma in the first place. Table 27.2 presents a list of probable catalysts.

Identifying the developmental and traumatic catalysts for internalization and social learning of reciprocal interpersonal patterns allows for greater understanding of current behavior. For example, in terms of achieving adult attachment relationships, some individuals have developed hostile strategies (e.g., verbally or physically fighting in order to elicit some form of interpersonal connection), while others have developed

TABLE 27.2. Some Possible Catalysts of Internalization and Social Learning

Developmental achievements	Traumatic learning
Attachment	Early loss of attachment figure
Security	Childhood illness or injury
Separation–individuation	Physical abuse
Positive affects	Sexual abuse
Gender identity	Emotional abuse
Resolution of Oedipal issues	Parental neglect
Self-esteem	
Self-confirmation	
Mastery of unresolved conflicts	
Identity formation	

submissive strategies (e.g., avoiding conflict and deferring to the wishes of the other in order to be liked and elicit gratitude). Although interpersonal theory asserts that internal interpersonal situations can mediate the perception and encoding of new input, the overt behavior of the other is influential, particularly as it activates a person's expectancies, wishes, fears, and so on that are associated with important motives or traumas. This will significantly influence the person's covert experience. Along with unfortunate traumatic experiences, the most important motives of individuals are those associated with the central achievements of personality development that have been identified across the theoretical spectrum.

Regulatory Metagoals: Generalized Social Learning

Pincus (2005a) has proposed an additional level of interpersonal learning that takes place concurrently with the association of particular patterns of interpersonal relating to the specific goals associated with emerging developmental achievements and coping with trauma. The second condition necessary for internalization of interpersonal experience is the association of the interpersonal situation with one or more of three superordinate regulatory functions or metagoals: field regulation, emotion regulation, and self-regulation. The concept of regulation has become almost ubiquitous in psychological theory, particularly in the domain of human development. Most theories of personality emphasize the importance of developing mechanisms for emotion regulation and self-regulation. Interpersonal theory is unique in its added emphasis on "field regulation" (i.e., the processes by which the behaviors of self and other transactionally influence each other). This has led to operational definitions of reciprocal interpersonal processes to describe the patterning of mutual influence of self and other within the interpersonal field. The emerging developmental motives and the coping demands of traumas listed in Table 27.2 all have significant implications for emotion regulation, self-regulation, and field regulation. This further contributes to the generalization of interpersonal learning to new interpersonal situations by providing a small number of superordinate psychological triggers to activate internal motives, schemas, expectancies, and so forth.

The importance of distinguishing these three regulatory metagoals is most directly related to understanding the shifting priorities that may be associated with interpersonal behavior, giving rise to unique patterns of intraindividual variability and interpersonal behavioral signatures. At any given time, the most prominent metagoal may be proximal field regulation. However, interpersonal behavior may also be associated with self-regulation (such as the derogation of others to promote self-esteem in narcissistic personality disorder) or emotion regulation (such as the use of sexual availability in order to feel more emotionally secure and stable in histrionic personality disorder). In such instances, interpersonal behavior may play a central role, even if the priority is not explicitly field regulation. Interpersonal behavior enacted in the service of regulating the self or emotion may reduce the contingencies associated with the behavior of the other person. This is another pathway to parataxic distortion, and also helps to account for the fluctuating symptomatology of psychopathology.

Normality and Abnormality

Contemporary interpersonal theory suggests that normality and psychopathology can be differentiated via the relative success or impairment in calibrating interpersonal relations to facilitate the mutual satisfac-

tion of agentic and communal motives and goals. The key processes involve the capacity to enter into new proximal interpersonal situations without parataxic distortion (Pincus, 2005a). In other words, the wider the range of proximal interpersonal situations in which a person exhibits anxiety-free functioning (little need for emotion regulation) and maintains self-esteem (little need for self-regulation), the more adaptive the individual is. When this is the case, there is no need to activate mediating interpersonal schemas, self-protective motives, or competing regulatory needs. The person can focus on the proximal situation, encode incoming interpersonal input without distortion, respond in adaptive ways that facilitate interpersonal relations (i.e., meet the agentic and communal needs of self and other), and establish complementary patterns of reciprocal behavior by fully participating in the relationship. The individual's current behavior will exhibit relatively strong contingency with the proximal behavior of the other and the normative contextual press of the situation. Adaptive interpersonal functioning is promoted by relatively trauma-free development in a culturally normative facilitating environment that has allowed the person to achieve most developmental milestones in normative ways, leading to full capacity to encode and elaborate incoming interpersonal input without bias from competing psychological needs.

In contrast, when the individual develops in a traumatic or non-normative environment, significant non-normative interpersonal learning around basic motives (attachment, individuation, gender identity, etc.) may be internalized and associated with difficulties in self-regulation, emotion regulation, and field regulation. In contrast to normality, psychopathology is reflected in a large range of proximal interpersonal situations that elicit anxiety (activating emotion-regulatory strategies), threaten self-esteem (activating self-regulatory strategies), and elicit dysfunctional behaviors (non-normative field-regulatory strategies). When this is the case, internal interpersonal situations are activated, and the individual is prone to exhibit various forms of parataxic distortion as his or her interpersonal learning history dictates. Thus the perception of the proximal interpersonal situation is mediated by internal experience; incoming interpersonal input is distorted; behavioral responses (output) disrupt interpersonal relations (i.e., fail to meet the agentic and communal needs of self and other); and relationships tend toward maladaptive patterns of reciprocal behavior. The individual's current behavior will exhibit relatively weak contingency with the proximal behavior of the other.

The Interpersonal Nexus of Personality and Psychopathology Revisited

We hope to have demonstrated that interpersonal functioning can play a central role in the description of psychopathology and the theory of psychopathology. The contemporary interpersonal tradition is a nomological net that can provide the architecture to coordinate these clinical tasks and advance the science and profession of personology and psychopathology (Millon, 2005). When expressions of psychopathology are described in terms of individual differences in agentic and communal constructs, pathoplastic relationships with those constructs, patterns of intraindividual variability, and interpersonal behavioral signatures, disorders are tied directly to psychological theory that has implications for etiology, maintenance, and treatment planning. Thus we agree with Krueger and colleagues (2008), Widiger and Clark (2000), and others in recommending that revisions of DSM (and ICD) provide a system to contextualize psychopathology within individual differences in personality. Given the advances in interpersonal description and interpersonal theory discussed here, we would argue that agentic and communal personality characteristics should be essential components of new diagnostic systems.

Note

1. Although it is not the focus of the current chapter, it is important to recognize that a pathoplastic relationship is bidirectional, such that an individual's characteristic expression of personality may be distorted by a psychiatric symptom syndrome, potentially resulting in more intra- and interpersonal distress.

References

aan het Rot, M., Moskowitz, D. S., & Young, S. N. (2008). Exposure to bright light is associated with positive social interaction and good mood over short time periods: A naturalistic study in mildly seasonal people. *Journal of Psychiatric Research*, *42*, 311–319.

Alden, L. E., & Capreol, M. J. (1993). Avoidant personality disorder: Interpersonal problems as predictors of treatment response. *Behavior Therapy*, *24*, 357–376.

Alden, L. E., Wiggins, J. S., & Pincus, A. L. (1990). Construction of circumplex scales for the inventory of interpersonal problems. *Journal of Personality Assessment*, *55*, 521–536.

Anchin, J. C., & Pincus, A. L. (in press). Evidence-based interpersonal psychotherapy with personality disorders: Theory, components, and strategies. In J. Magnavita (Ed.), *Evidence-based treatment of personality dysfunction: Principles, methods, and processes*. Washington, DC: American Psychological Association.

Atkinson, J. W. (1957). Motivational determinants of risk-taking behavior. *Psychological Review*, *64*, 359–372.

Bakan, D. (1966). *The duality of human existence: Isolation and communion in Western man*. Boston: Beacon Press.

Barrett, M. S., & Barber, J. P. (2007). Interpersonal profiles in major depressive disorder. *Journal of Clinical Psychology*, *63*, 247–266.

Bartholomew, K., & Horowitz, L. M. (1991). Attachment styles among young adults: A test of a four-category model. *Journal of Personality and Social Psychology*, *61*, 226–244.

Beck, A. T. (1983). Cognitive therapy of depression: New perspectives. In P. J. Clayton & J. E. Barrett (Eds.), *Treatment of depression: Old controversies and new approaches* (pp. 265–290). New York: Raven Press.

Benjamin, L. S. (1974). Structural Analysis of Social Behavior. *Psychological Review*, *81*, 392–425.

Benjamin, L. S. (1993). Every psychopathology is a gift of love. *Psychotherapy Research*, *3*, 1–24.

Benjamin, L. S. (1995). Good defenses make good neighbors. In H. Conte & R. Plutchik (Eds.), *Ego defenses: Theory and measurement* (pp. 38–78). New York: Wiley.

Benjamin, L. S. (1996). *Interpersonal diagnosis and treatment of personality disorders* (2nd ed.). New York: Guilford Press.

Benjamin, L. S. (2003). *Interpersonal reconstructive therapy: Promoting change in nonresponders*. New York: Guilford Press.

Benjamin, L. S. (2005). Addressing interpersonal and intrapsychic components of personality during psychotherapy. In S. Strack (Ed.), *Handbook of personology and psychopathology* (pp. 417–441). Hoboken, NJ: Wiley.

Benjamin, L. S., & Friedrich, F. J. (1991). Contributions of structural analysis of social behavior (SASB) to the bridge between cognitive science and a science of object relations. In M. Horowitz (Ed.), *Person schemas and maladaptive interpersonal patterns* (pp. 379–412). Chicago: University of Chicago Press.

Benjamin, L. S., & Pugh, C. (2001). Using interpersonal theory to select effective treatment interventions. In W. J. Livesley (Ed.), *Handbook of personality disorders: Theory, research, and treatment* (pp. 414–436). New York: Guilford Press.

Blatt, S. J. (1974). Levels of object representation in anaclitic and introjective depression. *Psychoanalytic Study of the Child*, *29*, 7–157.

Blatt, S. J. (2004). *Experiences of depression: Theoretical, clinical, and research perspectives*. Washington, DC: American Psychological Association.

Blatt, S. J., Auerbach, J. S., & Levy, K. N. (1997). Mental representations in personality development, psychopathology, and the therapeutic process. *Review of General Psychology*, *1*, 351–374.

Blatt, S. J., & Zuroff, D. C. (1992). Interpersonal relatedness and self-definition: Two prototypes for depression. *Clinical Psychology Review*, *12*, 527–562.

Borkovec, T. D., Newman, M. G., Pincus, A. L., & Lytle, R. (2002). A component analysis of cognitive-behavioral therapy for generalized anxiety disorder and the role of interpersonal problems. *Journal of Consulting and Clinical Psychology*, *70*, 288–298.

Bowlby, J. (1969). *Attachment and loss: Vol. 1. Attachment*. New York: Basic Books.

Bowlby, J. (1973). *Attachment and loss: Vol. 2. Separation: Anxiety and anger.* New York: Basic Books.

Cain, N. M. (2008). *Interpersonal pathoplasticity in social phobia: A clinical replication*. Unpublished doctoral dissertation, Pennsylvania State University.

Cain, N. M., Pincus, A. L., & Ansell, E. B. (2008). Narcissism at the crossroads: Phenotypic description of pathological narcissism across clinical theory, social/personality psy-

chology, and psychiatric diagnosis. *Clinical Psychology Review*, *28*, 638–656.
Carson, R. C. (1969). *Interaction concepts of personality*. Chicago: Aldine.
Carson, R. C. (1982). Self-fulfilling prophecy, maladaptive behavior, and psychotherapy. In J. C. Anchin & D. J. Kiesler (Eds.), *Handbook of interpersonal psychotherapy* (pp. 64–77). New York: Pergamon Press.
Carson, R. C. (1991). The social-interactional viewpoint. In M. Hersen, A. Kazdin, & A. Bellack (Eds.), *The clinical psychology handbook* (2nd ed., pp. 185–199). New York: Pergamon Press.
Carson, R. C. (1996). Seamlessness in personality and its derangements. *Journal of Personality Assessment*, *66*, 240–247.
Cassidy, J. (1999). The nature of the child's ties. In J. Cassidy & P. Shaver (Eds.), *Handbook of attachment: Theory, research, and clinical applications* (pp. 3–20). New York: Guilford Press.
Cervone, D. (2004). The architecture of personality. *Psychological Review*, *111*, 183–204.
Clark, L. A., Livesley, W. J., & Morey, L. (1997). Personality disorder assessment: The challenge of construct validity. *Journal of Personality Disorders*, *11*, 205–231.
Conroy, D. E., Willow, J. P., & Metzler, J. N. (2002). Multidimensional fear of failure measurement: The Performance Failure Appraisal Inventory. *Journal of Applied Sport Psychology*, *14*, 76–90.
Côté, S., & Moskowitz, D. S. (1998). On the dynamic covariation between interpersonal behavior and affect: Prediction from neuroticism, extraversion, and agreeableness. *Journal of Personality and Social Psychology*, *75*, 1032–1046.
D'Antono, B., Moskowitz, D. S., Miners, C., & Archambault, J. (2005). Gender and communal trait differences in the relations among social behaviour, affect arousal, and cardiac autonomic control. *Journal of Behavioral Medicine*, *28*, 267–279.
Dickinson, K. A., & Pincus, A. L. (2003). Interpersonal analysis of grandiose and vulnerable narcissism. *Journal of Personality Disorders*, *17*, 188–207.
Eid, M., & Diener, E. (1999). Intraindividual variability in affect: Reliability, validity, and personality correlates. *Journal of Personality and Social Psychology*, *76*, 662–676.
Eizenman, D. R., Nesselroade, J. R., Featherman, D. L., & Rowe, J. W. (1997). Intraindividual variability in perceived control in an older sample: The MacArthur Successful Aging Studies. *Psychology and Aging*, *12*, 489–502.
Fleeson, W. (2001). Toward a structure- and process-integrated view of personality: Traits as density distributions of states. *Journal of Personality and Social Psychology*, *80*, 1011–1027.
Fleeson, W., & Leicht, C. (2006). On delineating and integrating the study of variability and stability in personality psychology: Interpersonal trust as illustration. *Journal of Research in Personality*, *40*, 5–20.
Florsheim, P., Henry, W. P., & Benjamin, L. S. (1996). Integrating individual and interpersonal approaches to diagnosis: The Structural Analysis of Social Behavior and attachment theory. In F. Kaslow (Ed.), *Handbook of relational diagnosis* (pp. 81–101) New York: Wiley.
Fournier, M. A., Moskowitz, D. S., & Zuroff, D. C. (2008). Integrating dispositions, signatures, and the interpersonal domain. *Journal of Personality and Social Psychology*, *94*, 531–545.
Fournier, M. A., Zuroff, D. C., & Moskowitz, D. S. (2007). The social competition theory of depression: Gaining from an evolutionary approach to losing. *Journal of Social and Clinical Psychology*, *26*, 786–790.
Funder, D. C. (2006). Towards a resolution of the personality triad: Persons, situations, and behaviors. *Journal of Research in Personality*, *40*, 21–34.
Gallo, L. C., Smith, T. W., & Cox, C. M. (2006). Socioeconomic status, psychosocial processes, and perceived health: An interpersonal perspective. *Annals of Behavioral Medicine*, *31*, 109–119.
Gifford, R. (1991). Mapping nonverbal behavior on the interpersonal circle. *Journal of Personality and Social Psychology*, *61*, 279–288.
Grawe, K. (2004). *Psychological therapy*. Ashland, OR: Hogrefe & Huber.
Gurtman, M. B. (1994). The circumplex as a tool for studying normal and abnormal personality: A methodological primer. In S. Strack & M. Lorr (Eds.), *Differentiating normal and abnormal personality* (pp. 243–263). New York: Springer.
Gurtman, M. B. (1997). Studying personality traits: The circular way. In R. Plutchik & H. R. Conte (Eds.), *Circumplex models of personality and emotion* (pp. 81–102). Washington, DC: American Psychological Association.
Gurtman, M. B. (1999). Social competence: An

interpersonal analysis and reformulation. *European Journal of Psychological Assessment*, *15*, 233–245.

Gurtman, M. B. (2001). Interpersonal complementarity: Integrating interpersonal measurement with interpersonal models. *Journal of Counseling Psychology*, *48*, 97–110.

Gurtman, M. B., & Balakrishnan, J. D. (1998). Circular measurement redux: The analysis and interpretation of Interpersonal Circle Profiles. *Clinical Psychology: Science and Practice*, *5*, 344–360.

Gurtman, M. B., & Pincus, A. L. (2000). Interpersonal adjective scales: Confirmation of circumplex structure from multiple perspectives. *Personality and Social Psychology Bulletin*, *26*, 374–384.

Gurtman, M. B. & Pincus, A. L. (2003). The circumplex model: Methods and research applications. In I. B. Weiner (Series Ed.) & J. A. Schnika & W. F. Velicer (Eds.), *Handbook of psychology: Vol. 2. Research methods in psychology* (pp. 407–428). New York: Wiley.

Heck, S. A., & Pincus, A. L. (2001). Agency and communion in the structure of parental representations. *Journal of Personality Assessment*, *76*, 180–184.

Heller, D., Watson, D., Komar, J., Min, J., & Perunovic, W. Q. E. (2007). Contextualized personality: Traditional and new assessment procedures. *Journal of Personality*, *75*, 1229–1254.

Hopwood, C. J., Clarke, A. N., & Perez, M. (2007). Pathoplasticity of bulimic features and interpersonal problems. *International Journal of Eating Disorders*, *40*, 652–658.

Hopwood, C. J., & Morey, L. C. (2007). Psychological conflict in borderline personality as represented by inconsistent self-report item responding. *Journal of Social and Clinical Psychology*, *26*, 1096–1106.

Horowitz, L. M. (2004). *Interpersonal foundations of psychopathology*. Washington, DC: American Psychological Association.

Horowitz, L. M., Rosenberg, S. E., & Bartholomew, K. (1993). Interpersonal problems, attachment styles, and outcome in brief dynamic psychotherapy. *Journal of Consulting and Clinical Psychology*, *61*, 549–560.

Horowitz, L. M., & Vitkus, J. (1986). The interpersonal basis of psychiatric symptoms. *Clinical Psychology Review*, *6*, 443–469.

Horowitz, L. M., & Wilson, K. R. (2005). Interpersonal motives and personality disorders. In S. Strack (Ed.), *Handbook of personology and psychopathology* (pp. 495–510). Hoboken, NJ: Wiley.

Horowitz, L. M., Wilson, K. R., Turan, B., Zolotsev, P., Constantino, M. J., & Henderson, L. (2006). How interpersonal motives clarify the meaning of interpersonal behavior: A revised circumplex model. *Personality and Social Psychology Review*, *10*, 67–86.

Hoyenga, K. B., Hoyenga, K. T., Walters, K., & Schmidt, J. A. (1998). Applying the interpersonal circle and evolutional theory to gender differences in psychopathological traits. In L. Ellis & L. Ebertz (Eds.), *Males, females, and behavior: Toward biological understanding* (pp. 213–240). Westport, CT: Praeger.

Hurt, S. (2002). Personality disorders in female prisoners: Intraindividual variability in borderline and narcissistic criteria. *Dissertation Abstracts International*, *62*(7), 3379B.

Kachin, K. E., Newman, M. G., & Pincus, A. L. (2001). An interpersonal problem approach to the division of social phobia subtypes. *Behavior Therapy*, *32*, 479–501.

Kasoff, M. B. (2002). Interpersonal subtypes of generalized anxiety disorder: Derivation and differentiation in patterns of adult attachment and psychiatric comorbidity. *Dissertation Abstracts International*, *62*(12), 5967B.

Kernis, M. H. (2005). Measuring self-esteem in context: The importance of stability of self-esteem in psychological functioning. *Journal of Personality*, *73*, 1569–1605.

Kiesler, D. J. (1983). The 1982 Interpersonal Circle: A taxonomy for complementarity in human transactions. *Psychological Review*, *90*, 185–214.

Kiesler, D. J. (1986). The 1982 Interpersonal Circle: An analysis of DSM-III personality disorders. In T. Millon & G. L. Klerman (Eds.), *Contemporary directions in psychopathology: Toward the DSM-IV* (pp. 571–598). New York: Guilford Press.

Kiesler, D. J. (1988). *Therapeutic metacommunication: Therapist impact disclosure as feedback in psychotherapy*. Palo Alto, CA: Consulting Psychologists Press.

Kiesler, D. J. (1991). Interpersonal methods of assessment and diagnosis. In C. R. Snyder & D. R. Forsyth (Eds.), *Handbook of social and clinical psychology* (pp. 438–468). New York: Pergamon Press.

Kiesler, D. J. (1996). *Contemporary interpersonal theory and research: Personality, psychopathology, and psychotherapy*. New York: Wiley.

Kiesler, D. J., Goldston, C. S., & Schmidt, J. A. (1991). *Manual for the Check List of Interpersonal Transactions—Revised (CLOIT-R) and the Check List of Psychotherapy Transactions—Revised (CLOPT-R).* Richmond: Virginia Commonwealth University.

Kiesler, D. J., Schmidt, J. A., & Wagner, C. C. (1997). A circumplex inventory of impact messages: An operational bridge between emotion and interpersonal behavior. In R. Plutchik & H. Contes (Eds.), *Circumplex models of personality and emotions* (pp. 221–244). Washington, DC: American Psychological Association.

Klein, M. H., Wonderlich, S., & Shea, M. T. (1993). Models of relationships between personality and depression: Toward a framework for theory and research. In M. Klein, D. Kupfer, & M. T. Shea (Eds.), *Personality and depression: A current view* (pp. 1–54). New York: Guilford Press.

Krueger, R. F., Skodol, A. E., Livesley, W. J., Shrout, P. E., & Huang, Y. (2008). Synthesizing dimensional and categorical approaches to personality disorders: Refining the research agenda for DSM-V Axis II. In J. Helzer, H. C. Kraemer, R. F. Kruger, H. Wittchen, & P. Sirovatka (Eds.), *Dimensional approaches in diagnostic classification: Refining the research agenda for DSM-V* (pp. 85–99). Washington, DC: American Psychiatric Association.

Lackner, J. M., & Gurtman, M. B. (2004). Pain catastrophizing and interpersonal problems: A circumplex analysis of the communal coping model. *Pain*, *110*, 597–604.

Lackner, J. M., & Gurtman, M. B. (2005). Patterns of interpersonal problems in irritable bowel syndrome patients: A circumplex analysis. *Journal of Psychosomatic Research*, *58*, 523–532.

LaForge, R. (2004). The early development of the interpersonal system of personality (ISP). *Multivariate Behavioral Research*, *39*, 359–378.

LaForge, R., Freedman, M. B., & Wiggins, J. S. (1985). Interpersonal circumplex models: 1948–1983. *Journal of Personality Assessment*, *49*, 613–631.

Leary, T. (1957). *Interpersonal diagnosis of personality.* New York: Ronald Press.

Leichsenring, F., Kunst, H., & Hoyer, J. (2003). Borderline personality organization in violent offenders: Correlations of identity diffusion and primitive defense mechanisms with antisocial features, neuroticism, and interpersonal problems. *Bulletin of the Menninger Clinic*, *67*, 314–327.

Leihener, R., Wagner, A., Haaf, B., Schmidt, C., Lieb, K., Stieglitz, R., et al. (2003). Subtype differentiation of patients with borderline personality disorder using a circumplex model of interpersonal behavior. *Journal of Mental and Nervous Disease*, *191*, 248–254.

Lenzenweger, M. F., Johnson, M. D., & Willett, J. B. (2004). Individual growth curve analysis illuminates stability and change in personality disorder features: The longitudinal study of personality disorders. *Archives of General Psychiatry*, *61*, 1015–1024.

Locke, K. D. (2000). Circumplex scales of interpersonal values: Reliability, validity, and applicability to interpersonal problems and personality disorders. *Journal of Personality Assessment*, *75*, 249–267.

Locke, K. D. (2006). Interpersonal circumplex measures. In S. Strack (Ed.), *Differentiating normal and abnormal personality* (2nd ed., pp. 383–400). New York: Springer.

Locke, K. D., & Sadler, P. (2007). Self-efficacy, values, and complementarity in dyadic interactions: Integrating interpersonal and social-cognitive theory. *Personality and Social Psychology Bulletin*, *33*, 94–109.

Maling, M. S., Gurtman, M. B., & Howard, K. I. (1995). The response of interpersonal problems to varying doses of psychotherapy. *Psychotherapy Research*, *5*, 63–75.

Markey, P. M., Funder, D. C., & Ozer, D. J. (2003). Complementarity of interpersonal behaviors in dyadic interactions. *Personality and Social Psychology Bulletin*, *29*, 1082–1090.

McAdams, D. P. (1995). What do we know when we know a person? *Journal of Personality*, *63*, 365–396.

McGlashan, T. H., Grilo, C. M., Sanislow, C. A., Ralevski, E., Morey, L. C., Gunderson, J. G., et al. (2005). Two-year prevalence and stability of individual DSM-IV criteria for schizotypal, borderline, avoidant, and obsessive–compulsive personality disorders: Toward a hybrid model of Axis II disorders. *American Journal of Psychiatry*, *162*, 883–889.

McGregor, H. A., & Elliot, A. J. (2005). The shame of failure: Examining the link between fear of failure and shame. *Personality and Social Psychology Bulletin*, *31*, 218–231.

McLemore, C. W., & Benjamin, L. S. (1979). Whatever happened to interpersonal diagnosis?: A psychosocial alternative to DSM-III. *American Psychologist*, *34*, 17–34.

Miller, J. D., & Campbell, W. K. (2008). Comparing clinical and social–personality conceptualizations of narcissism. *Journal of Personality*, *76*, 449–476.

Millon, T. (2000). Reflections on the future of DSM Axis II. *Journal of Personality Disorders*, *14*, 30–41.

Millon, T. (2005). Reflections on the future of personology and psychopathology. In S. Strack (Ed.), *Handbook of personology and psychopathology* (pp. 527–546). Hoboken, NJ: Wiley.

Millon, T. (with Davis, R. D.). (1996). *Disorders of personality: DSM-IV and beyond* (2nd ed.). New York: Wiley.

Mineka, S., Watson, D., & Clark, L. A. (1998). Comorbidity of anxiety and unipolar mood disorders. *Annual Review of Psychology*, *49*, 377–412.

Mischel, W., & Shoda, Y. (1995). A cognitive–affective system theory of personality: Reconceptualizing situations, dispositions, dynamics, and invariance in personality structure. *Psychological Review*, *102*, 246–268.

Mischel, W., & Shoda, Y. (1998). Reconciling processing dynamics and personality dispositions. *Annual Review of Psychology*, *49*, 229–258.

Mitchell, S. A. (1988). The intrapsychic and the interpersonal: Different theories, different domains, or historical artifacts? *Psychoanalytic Inquiry*, *8*, 472–496.

Moskowitz, D. S. (1994). Cross-situational generality and the interpersonal circumplex. *Journal of Personality and Social Psychology*, *66*, 921–933.

Moskowitz, D. S. (2005). Unfolding interpersonal behavior. *Journal of Personality*, *73*, 1607–1632.

Moskowitz, D. S., Pinard, G., Zuroff, D. C., Annable, L., & Young, S. N. (2001). The effect of tryptophan on social interaction in everyday life: A placebo-controlled study. *Neuropsychopharmacology*, *25*, 277–289.

Moskowitz, D. S., & Zuroff, D. C. (2004a). Flux, pulse, and spin: Dynamic additions to the personality lexicon. *Journal of Personality and Social Psychology, 86*, 880–893.

Moskowitz, D. S., & Zuroff, D. C. (2004b). Robust predictors of flux, pulse, and spin. *Journal of Research in Personality*, *39*, 130–147.

Parker, G., Both, L., Olley, A., Hadzi-Pavlovic, D., Irvine, P., & Jacobs, G. (2002). Defining disordered personality functioning. *Journal of Personality Disorders*, *16*, 502–533.

Paulhus, D. L., & Martin, C. L. (1987). The structure of personality capabilities. *Journal of Personality and Social Psychology*, *52*, 354–365.

Paulhus, D. L., & Martin, C. L. (1988). Functional flexibility: A new conception of interpersonal flexibility. *Journal of Personality and Social Psychology*, *55*, 88–101.

Pincus, A. L. (1994). The interpersonal circumplex and the interpersonal theory: Perspectives on personality and its pathology. In S. Strack & M. Lorr (Eds.), *Differentiating normal and abnormal personality* (pp. 114–136). New York: Springer.

Pincus, A. L. (2002). Constellations of dependency within the five-factor model of personality. In P. T. Costa & T. A. Widiger (Eds.), *Personality disorders and the five-factor model of personality* (2nd ed., pp. 203–214). Washington, DC: American Psychological Association.

Pincus, A. L. (2005a). A contemporary integrative interpersonal theory of personality disorders. In J. Clarkin & M. Lenzenweger (Eds.), *Major theories of personality disorder* (2nd ed., pp. 282–331). New York: Guilford Press.

Pincus, A. L. (2005b). The interpersonal nexus of personality disorders. In S. Strack (Ed.), *Handbook of personology and psychopathology* (pp. 120–139). Hoboken, NJ: Wiley.

Pincus, A. L., & Ansell, E. B. (2003). Interpersonal theory of personality. In I. B. Weiner (Vol. Ed.) & T. Millon & M. Lerner (Vol. Eds.), *Handbook of psychology: Vol. 5. Personality and social psychology* (pp. 209–229). New York: Wiley.

Pincus, A. L., Ansell, E. B., Pimentel, C. A., Cain, N. M., Wright, A. G. C., & Levy, K. N. (2009). Initial construction and validation of the Pathological Narcissism Inventory. *Psychological Assessment*, *21*, 365–379.

Pincus, A. L., & Cain, N. M. (2008). Interpersonal psychotherapy. In D. C. S. Richard & S. K. Huprich (Eds.), *Clinical psychology: Assessment, treatment, and research* (pp. 213–245). San Diego, CA: Academic Press.

Pincus, A. L., & Gurtman, M. B. (1995). The three faces of interpersonal dependency: Structural analyses of self-report dependency measures. *Journal of Personality and Social Psychology*, *69*, 744–758.

Pincus, A. L., & Gurtman, M. B. (2003). Interpersonal assessment. In J. S. Wiggins, *Paradigms of personality assessment* (pp. 246–261). New York: Guilford Press.

Pincus, A. L., & Gurtman, M. B. (2006). In-

terpersonal theory and the interpersonal circumplex: Evolving perspectives on normal and abnormal personality. In S. Strack (Ed.), *Differentiating normal and abnormal personality* (2nd ed., pp. 83–111). New York: Springer.

Pincus, A. L., & Lukowitsky, M. R. (in press). Pathological narcissism and narcissistic personality disorder. *Annual Review of Clinical Psychology*.

Pincus, A. L., Lukowitsky, M. R., Wright, A. G. C., & Eichler, W. C. (2009). The interpersonal nexus of persons, situations, and psychopathology. *Journal of Research in Personality*, *43*, 264–265.

Pincus, A. L., Przeworski, A., Yamasaki, A., Kasoff, M. B., Newman, M. G., Castonguay, L. G., et al. (2005, June). *Interpersonal pathoplasticity in generalized anxiety disorder: A cluster analytic replication*. Paper presented at the meeting of the Society for Interpersonal Theory and Research, Montréal.

Pincus, A. L., & Wiggins, J. S. (1990). Interpersonal problems and conceptions of personality disorders. *Journal of Personality Disorders*, *4*, 342–352.

Pincus, A. L., & Wilson, K. R. (2001). Interpersonal variability in dependent personality. *Journal of Personality*, *69*, 223–251.

Pincus, A. L., & Wright, A. G. C. (in press). Interpersonal diagnosis of psychopathology. In L. M. Horowitz & S. Strack (Eds.), *Handbook of interpersonal psychology*. New York: Wiley.

Puschner, B., Kraft, S., & Bauer, S. (2004). Interpersonal problems and outcome in outpatient psychotherapy: Findings from a long-term longitudinal study in Germany. *Journal of Personality Assessment*, *83*, 223–234.

Regier, D. A., Kaelber, C. T., Rae, D. S., Farmer, M. E., Knauper, B., Kessler, R. C., et al. (1998). Limitations of diagnostic criteria and assessment instruments for mental disorders: Implications for research and policy. *Archives of General Psychiatry*, *55*, 109–115.

Russell, J. J., Moskowitz, D. S., Zuroff, D. C., Sookman, D., & Paris, J. (2007). Stability and variability of affective experience and interpersonal behavior in borderline personality disorder. *Journal of Abnormal Psychology*, *116*, 578–588.

Ryan, K., & Shean, G. (2007). Patterns of interpersonal behaviors and borderline personality characteristics. *Personality and Individual Differences*, *42*, 193–200.

Sadler, P., & Woody, E. (2003). Is who you are who you're talking to?: Interpersonal style and complementarity in mixed-sex interactions. *Journal of Personality and Social Psychology*, *84*, 80–95.

Safran, J. D. (1990a). Towards a refinement of cognitive therapy in light of interpersonal theory: I. Theory. *Clinical Psychology Review*, *10*, 87–105.

Safran, J. D. (1990b). Towards a refinement of cognitive therapy in light of interpersonal theory: II. Practice. *Clinical Psychology Review*, *10*, 107–121.

Safran, J. D. (1992). Extending the pantheoretical applications of interpersonal inventories. *Journal of Psychotherapy Integration*, *2*, 101–105.

Salzer, S., Pincus, A. L., Hoyer, J., Kreische, R., Leichsenring, F., & Leibling, E. (2008). Interpersonal subtypes within generalized anxiety disorder. *Journal of Personality Assessment*, *90*, 292–299.

Sansone, R. A., & Sansone, L. A. (2008). A longitudinal perspective on personality disorder symptomatology. *Psychiatry*, *5*, 53–57.

Schaefer, E. S. (1961). Converging conceptual models for maternal behavior and child behavior. In J. C. Glidwell (Ed.), *Parental attitudes and child behavior* (pp. 124–146). Springfield, IL: Thomas.

Shechtman, N., & Horowitz, L. (2006). Interpersonal and noninterpersonal interactions, interpersonal motives, and the effect of frustrated motives. *Personality and Social Psychology Bulletin*, *32*, 1126–1139.

Shoda, Y., Mischel, W., & Wright, J. C. (1989). Intuitive interactionism in person perception: Effects of situation–behavior relations on dispositional judgments. *Journal of Personality and Social Psychology*, *56*, 41–53.

Shoda, Y., Mischel, W., & Wright, J. C. (1993a). Links between personality judgments and contextualized behavior patterns: Situation–behavior profiles of personality prototypes. *Social Cognition*, *11*, 399–429.

Shoda, Y., Mischel, W., & Wright, J. C. (1993b). The role of situational demands and cognitive competencies in behavior organization and personality coherence. *Journal of Personality and Social Psychology*, *65*, 1023–1035.

Shoda, Y., Mischel, W., & Wright, J. C. (1994). Intraindividual stability in the organization and patterning of behavior: Incorporating psychological situations into the idiographic analysis of personality. *Journal of Personality and Social Psychology*, *67*, 674–687.

Shorey, H. S., & Snyder, C. R. (2006). The role

of adult attachment styles in psychopathology and psychotherapy outcomes. *Review of General Psychology*, *10*, 1–20.

Sibrava, N. J., Pincus, A. L., Przeworski, A., Yamasaki, A. S., Newman, M. G., & Borkovec, T. D. (2007, November). *Heterogeneity in the presentation of GAD*. Paper presented at the 41st Annual Meeting of the Association for Behavioral and Cognitive Therapies, Philadelphia.

Slaney, R. B., Pincus, A. L., Uliaszek, A. A., & Wang, K. T. (2006). Conceptions of perfectionism and interpersonal problems: Evaluating groups using the structural summary method for circumplex data. *Assessment*, *13*, 138–153.

Steyer, R., Schmitt, M., & Eid, M. (1999). Latent state–trait theory and research in personality and individual differences. *European Journal of Personality*, *13*, 389–408.

Sullivan, H. S. (1948). The meaning of anxiety in psychiatry and in life. *Psychiatry: Journal for the Study of Interpersonal Processes*, *11*, 1–13.

Sullivan, H. S. (1953a). *Conceptions of modern psychiatry*. New York: Norton.

Sullivan, H. S. (1953b). *The interpersonal theory of psychiatry*. New York: Norton.

Sullivan, H. S. (1954). *The psychiatric interview*. New York: Norton.

Sullivan, H. S. (1956). *Clinical studies in psychiatry*. New York: Norton.

Sullivan, H. S. (1962). *Schizophrenia as a human process*. New York: Norton.

Sullivan, H. S. (1964). *The fusion of psychiatry and social science*. New York: Norton.

Tracey, T. J. (1994). An examination of the complementarity of interpersonal behavior. *Journal of Personality and Social Psychology*, *67*, 864–878.

Trobst, K. K. (2000). An interpersonal conceptualization and quantification of social support transactions. *Personality and Social Psychology Bulletin*, *26*, 971–986.

Widiger, T. A. (1991). Definition, diagnosis, and differentiation. *Journal of Personality Disorders*, *5*, 42–51.

Widiger, T. A., & Clark, L. A. (2000). Toward DSM-V and the classification of psychopathology. *Psychological Bulletin*, *126*, 946–963.

Widiger, T. A., & Trull, T. J. (1991). Diagnosis and clinical assessment. *Annual Review of Psychology*, *42*, 109–133.

Widiger, T. A., Verheul, R., & van den Brink, W. (1999). Personality and psychopathology. In L. A. Pervin & O. P. John (Eds.), *Handbook of personality: Theory and research* (2nd ed., pp. 347–366). New York: Guilford Press.

Wiggins, J. S. (1979). A psychological taxonomy of trait-descriptive terms: The interpersonal domain. *Journal of Personality and Social Psychology*, *37*, 395–412.

Wiggins, J. S. (1982). Circumplex models of interpersonal behavior in clinical psychology. In P. C. Kendall & J. N. Butchner (Eds.), *Handbook of research methods in clinical psychology* (pp. 183–221). New York: Wiley.

Wiggins, J. S. (1991). Agency and communion as conceptual coordinates for the understanding and measurement of interpersonal behavior. In D. Cicchetti & W. M. Grove (Eds.), *Thinking clearly about psychology: Essays in honor of Paul E. Meehl. Vol. 2. Personality and psychopathology* (pp. 89–113). Minneapolis: University of Minnesota Press.

Wiggins, J. S. (1996). An informal history of the interpersonal circumplex tradition. *Journal of Personality Assessment*, *66*, 217–233.

Wiggins, J. S. (1997a). Circumnavigating Dodge Morgan's interpersonal style. *Journal of Personality*, *65*, 1069–1086.

Wiggins, J. S. (1997b). In defense of traits. In R. Hogan, J. A. Johnson, & S. R. Briggs (Eds.), *Handbook of personality psychology* (pp. 95–141). San Diego, CA: Academic Press.

Wiggins, J. S. (2003). *Paradigms of personality assessment*. New York: Guilford Press.

Wiggins, J. S., & Broughton, R. (1985). The interpersonal circle: A structural model for the integration of personality research. In R. Hogan & W. H. Jones (Eds.), *Perspectives in personality* (Vol. 1, pp. 1–47). Greenwich, CT: JAI Press.

Wiggins, J. S., Phillips, N., & Trapnell, P. (1989). Circular reasoning about interpersonal behavior: Evidence concerning some untested assumptions underlying diagnostic classification. *Journal of Personality and Social Psychology*, *56*, 296–305.

Wiggins, J. S., & Pincus, A. L. (1989). Conceptions of personality disorders and dimensions of personality. *Psychological Assessment*, *1*, 305–316.

Wiggins, J. S., Trapnell, P., & Phillips, N. (1988). Psychometric and geometric characteristics of the Revised Interpersonal Adjective Scales (IAS-R). *Multivariate Behavioral Research*, *23*, 517–530.

Wiggins, J. S., & Trobst, K. K. (1997). When is a circumplex an "interpersonal circumplex"?:

The case of supportive actions. In R. Plutchik & H. R. Conte (Eds.), *Circumplex models of personality and emotions* (pp. 57–80). Washington, DC: American Psychological Association.

Wiggins, J. S., & Trobst, K. K. (1999). The fields of interpersonal behavior. In L. A. Pervin & O. P. John (Eds.), *Handbook of personality: Theory and research* (2nd ed., pp. 653–670). New York: Guilford Press.

Woody, E., & Sadler, P. (2005). Structural equation models for interchangeable dyads: Being the same makes a difference. *Psychological Methods*, *10*, 139–158.

Wright, A. G. C., Pincus, A. L., Conroy, D. E., & Elliot, A. J. (2009). The pathoplastic relationship between interpersonal problems and fear of failure. *Journal of Personality*, *77*, 997–1024.

Wright, A. G. C., Pincus, A. L., Conroy, D. E., & Hilsenroth, M. J. (2009). Integrating methods to optimize circumplex description and comparison of groups. *Journal of Personality Assessment*, *91*, 311–322.

Zeigler-Hill, V., Clark, C. B., & Pickard, J. D. (2008). Narcissistic subtypes and contingent self-esteem: Do all narcissists base their self-esteem on the same domains? *Journal of Personality*, *76*, 753–774.

Zuroff, D. C., Moskowitz, D. S., & Côté, S. (1999). Dependency, self-criticism, interpersonal behaviour and affect: Evolutionary perspectives. *British Journal of Clinical Psychology*, *38*, 231–250.

CHAPTER 28

Reconceptualizing Autism Spectrum Disorders as Autism-Specific Learning Disabilities and Styles

BRYNA SIEGEL

In this chapter, I examine contemporary trends in the conceptualization of autism spectrum disorders. The term "autism spectrum disorders" is itself part of the natural history of the evolving taxonomy of this disorder. A modern awareness of autism as a distinctive behavioral disorder started in the middle of the 20th century. Since then, as its conceptualization has been scrutinized, there has been an inexorable broadening of its boundaries. Once seen as a rare condition, it is now characterized as very common. A review of changes in the phenomenology of autism, and in our understanding of its etiology, forms a narrative of how our concepts have changed to reflect the results of using increasingly precise tools to view autism's phenomenology. Where is this process going? Where does it need to go? How are changes in conceptualizing autism being driven by etiological studies? How are changes in conceptualization being driven by observations about the efficacy of new treatments, treatment responses, and prognosis? How should the nosology of developmental neuropathology in general, and autism in particular, be taxonomized to meet all these needs?

Natural History of the Contemporary Autism Spectrum

Leo Kanner and Hans Asperger

The modern understanding of the autism spectrum started with Leo Kanner, an American child psychiatrist working at John Hopkins University (Kanner, 1943), and with Hans Asperger, a Austrian pediatrician working in Vienna (Asperger, 1944). Using the state-of-the-art clinical methodology of the 1940s, each investigator detailed his small, accumulated series of cases that shared deficits in social awareness, social understanding, and social interaction. Rereading these cases today reveals that each investigator included a rather broad range of socially impaired individuals, some of whom today might be noted to have features of intellectual disability, severe language impairment, obsessive–compulsive disorder, conduct problems, perseverative behavior, and/or attentional deficits. It would be fair to conclude that the heterogeneity of what is now considered the "autism spectrum" in fact began with Kanner and Asperger.

The United States and Germany (which at this time controlled Austria) were at war as these investigators published their earliest studies. For over 30 years, there was no point-by-point analysis of what each had detailed. It was not until 1981 that Lorna Wing, a prominent British child psychiatrist and autism researcher, began explicating Asperger's work for the English-language literature. Wing (1981) pointed out the similarities and differences between the original cases described by Kanner and Asperger, launching further comparative taxonomic work focused on whether Kanner and Asperger were talking about distinctive, adjacent, or overlapping conditions. Szatmari (1991; Szatmari et al., 1995) produced an authoritative review of such work, ultimately supporting the inclusion of Asperger's disorder along with autistic disorder among the "pervasive developmental disorders" (PDDs) in DSM-IV (American Psychiatric Association, 1994).

At the time when Wing introduced Asperger's work to the English-language audience, DSM-III (American Psychiatric Association, 1980) was the current U.S. diagnostic manual (and ICD-9 [World Health Organization, 1977] contained the international classification of psychiatric disorders). With DSM-III, Kanner's original description had already been broadened to include an early concept of PDD, as well as the notion of "residual-state autism," which acknowledged clinical studies of cases with mild or partial subsets of the characteristics of social dysfunction originally described by Kanner. By the time DSM-III was developed, it was recognized that autistic signs could occur across individuals in different combinations and in different numbers, could vary in severity, and could sometimes be ameliorated with age (and maybe treatment).

What were the separate and specific contributions of Kanner and Asperger to this understanding? How do their bodies of work contribute, separately and together, to where we are today in viewing these conditions jointly and collectively as "autism spectrum disorders"?

Kanner's Contribution

Kanner characterized the similarities among his original cases as constituting "early infantile autism." The word "autism" was derived from the Greek word *autos* for "self," since Kanner described autism as characterized by a focus on the self, much like that seen in the infantile stage preceding the development of a capacity for specific and reciprocal social attachments and relations. Over the years, it became common clinically to refer back to this original case series as illustrating "Kanner's autism," "Kannerian autism," or "classical autism." However, when Kanner's original 13 cases are examined, the differences are nearly as prominent as their similarities. Some had verbal proficiency, though with odd use of expressive language tied to social deficits; others were nonverbal. Some had unusual and repetitive motor movements; others did not. Some have what is now recognized as intellectual disability; others seem to have been of much more normal intellectual capacity, but with notable excellence in nonverbal capacities. The cases did share social deficits of "autistic aloneness." In some cases there were striking similarities in sensory or motor disturbances or in language use. Kanner was not able to formulate a cohesive hypothesis as to why this latter set of traits might cluster with the ubiquitous social deficits. Initially, he believed that aloof parenting might play a role in the development of pathologically aloof children (this idea is discussed further below). Over time, however, he became increasingly persuaded that these traits were "inborn" defects, although he never did offer a coherent theory for their comorbidity.

Asperger's Contribution

The story is not much different for Asperger. Several years after Wing's (1981) original paper on Asperger, Uta Frith (1991) one of Wing's students, produced an in-depth volume exploring the sui generis nature of Asperger's disorder; this book included the first English-language translation of Asperger's original paper and its cases. Like Kanner's cases, Asperger's series contained individuals with and without atypical features of language development, and with and without atypical motor disturbances. As a whole, however, Asperger's cases less often seemed impaired by comorbid and significant intellectual disability. They shared a

propensity for odd and eccentric ways of relating to others, but some eschewed interaction less than others. As a group, all suffered from lack of success in forming meaningful social relationships; however, some seemed closer to qualifying for a contemporary diagnosis of oppositional defiant disorder than others, or than any of Kanner's cases. As in Kanner's series, there were some striking similarities in regard to social disability, but there was also heterogeneity with respect to these other traits.

Interestingly, though the heterogeneity of the autism spectrum existed from its initial framing, the prevalence rates took years to catch up. Why? Does a high reported rate now encourage diagnosticians to see it everywhere, while in the past, did a very low reported rate encourage diagnosticians to see it seldom?

The Diagnosis of Autism Prior to DSM-III

Another way to understand better where we are today in thinking of autism as a "spectrum disorder" is to understand what went on in the almost 40 years between the publication of Kanner's original paper in 1943 and the inclusion of "infantile autism" as a diagnostic category in DSM-III in 1980. Kanner, immersed in the contemporary psychiatric theory of the 1940s, initially tried to explain the symptoms he observed in terms of psychoanalytic theory.

Following in the psychoanalytic formulations of infant emotional development studied by such workers as Margaret Mahler (Mahler & Gosliner, 1955), Kanner descriptively characterized autism as resulting from a disturbance in affective contact. Relying upon psychoanalytic theory, he speculated that autistic aloneness could arise from an early rejection of an infant by an emotionally cold mother who failed to provide the secure, predictable, and contingent responses the infant sought to construct his or her earliest schema of a social world. Most of Kanner's original cases were the children of highly successful, highly educated, well-off parents who had come to Johns Hopkins University to seek out the expertise that an academic child psychiatrist might impart regarding strategies for reaching their apparently socially unreachable offspring. Kanner looked at these parents as a group—in particular, noting the manner in which they could provide objective, detailed, almost clinical characterizations of their children—and initially endorsed the psychoanalytic explanation. However, he was also convinced that a biological vulnerability might somehow have interacted with such parenting. This way of thinking about autism spectrum disorders as the result of an interaction of factors has subsequently been transformed, but has persisted to the present; we now regard genetic influences as vulnerabilities and carry out research on possible environmental triggers (for a review, see Altevogt, Hanson, & Leschner, 2008).

After Kanner and prior to DSM-III, a central touchpoint in thinking about the classification of autism was comparing and contrasting autism with childhood schizophrenia. What was being called "childhood schizophrenia" was also poorly understood, but it was clinically less often associated with disturbances of basic cognitive functions, and was considered a possible *form fruste* of (severe) schizophrenia in adults. Autism was postulated to be congruent with childhood schizophrenia, even in mute children (Bender, 1947).

Writers following directly upon Kanner and the early child psychoanalysts, such as Bruno Bettelheim (1967), described the "refrigerator mother" as the primary causal factor in autism. Bettelheim founded the Orthogenic School at the University of Chicago as a residential treatment milieu for such children, whom he felt would benefit from a "parent-ectomy." This marked the apex of autism viewed as a disorder of parenting. Many children at the Orthogenic School had indeed experienced poor parenting, but did not specifically manifest the full range of signs associated with Kannerian autism, and were very largely without language or other cognitive impairments (Bettelheim & Rosenfeld, 1993). Bettelheim's work remains most notable for being among the earliest efforts to provide a specific treatment method for the diagnosis of autism. This is a notable milestone since over time the prevalence of autism spectrum diagnoses happens to have grown in tandem with the recognition of specific autism treatments' being available.

The Shift to Understanding Autism as a Neurobiological Disorder

In 1964, a psychologist who was himself the parent of a boy with autism, Bernard Rimland, published a book titled *Infantile Autism*. This volume offered the first comparative review of existing evidence for a psychodynamic versus a biological etiology for autism, and Rimland argued convincingly that there was more support for biological hypotheses. Although he offered only a "broad-brushstrokes" picture of how the brain might operate differently in children with autism, it resonated with other parents and treaters of autistic children, who by now had realized that many children with autism had loving, supportive families and typically developing siblings. The first strong empirical support for a biological yet familial link came in the work of Folstein and Rutter (1977), who showed exceedingly high concordance rates in twins; they also noted that concordance was much higher in identical than in fraternal twins. Later, as they continued their studies of familiality, they noted a high rate of language difficulties in nonautistic siblings—who sometimes seemed also to suffer mild social difficulties (Folstein & Rutter, 1988). This work was another milestone as it was the first biological support for a spectrum of autism.

In the early 1970s, as biological views of autism gained support, Rimland and other parents—including Ruth Christ-Sullivan (whose son became a model for Dustin Hoffman's character in the movie *Rain Man*) and Clara Claiborne Parks (who wrote *The Siege* [1967], the first parental account of raising a child with autism)—founded the National Society for Autistic Children, which is now the Autism Society of America. This group then developed its own diagnostic criteria for autism, authored by Edward Ritvo and B. J. Freeman at the University of California, Los Angeles. These criteria operationalized all the features of Kanner's early infantile autism, but eschewed any reference to parenting as a determinant of the disorder. These events epitomize the unique way in which layworkers and scientific workers have partnered from early on in deciding what is autism.

Increasingly, the literature contained less speculation about psychodynamics and instead reflected the view that biological differences must govern etiology. In 1971, the *Journal of Autism and Childhood Schizophrenia* was founded by Eric Schopler at the University of North Carolina. This was the first scientific journal devoted to autism. The journal's mission reflected a further move away from psychodynamic classification of autistic symptoms, as the treatment arm of the North Carolina group focused on a remedial educative approach to treating autism (Project TEACCH). While Bettelheim and some other individual clinicians trained in the 1940s, 1950s, and early 1960s persisted in viewing and treating autism and related clinical conditions with psychodynamic psychotherapy, the shift to treatment and classification of autism as a neurodevelopmental disorder was fairly complete 25–30 years after the publication of Kanner's 1943 paper.

Nosological Implications of Autism as a Heterogeneous Biological Disorder

Comorbid Impairment in Intellectual Functioning

When the field came to view autism as the result of a biological abnormality, acceptance of the frequently observed impairment in intellectual functioning as a comorbidity became more common. This acceptance has brought to the fore a few important nosological taxonomic issues.

First, is the intellectual impairment often observed in conjunction with autism the product of more global brain impairment? What are the implications of studying autism with or without accompanying mental retardation? Some investigators have focused on brain research (e.g., Minshew, 1992, 1996) or neuropsychological studies (e.g., studies of "theory of mind" or executive functioning; Baron-Cohen, 1995; Happé, 1994), but only in those meeting criteria for autism but not intellectual impairment, in an attempt to better understand any unitary contribution of autism-specific biological impairment. Even when studying individuals without intellectual disability but with autism, investigators had no way to ascertain whether core clinical phenomena resulted from geneti-

cally controlled maldevelopment or from acquired structural damage such as a perinatal insult. This raised a question: How useful would a clinically based classification system be in studies of the etiology of autism if some cases might represent maldevelopment (genetic abnormality) and other cases might represent structural damage (of possibly the same systems)? Such a clinically based classification system might be adequate for understanding responses to behavioral treatments, but might be much less useful in a genome search, for example.

Second, could the intellectual impairment often seen in autism accrue in part, or even wholely, from social deficits that block learning (e.g., via impaired imitative capacity, or impairment in the drive to learn in order to please others; see Siegel, 2003)? If so, could the brain structures apart from those most specific to social facility be intact but inaccessible in such cases? If this could be the case, what would the implications be for a clinically based classification system if some cases occur because of an atypical pattern of learning and other cases occur because of structural damage? If such cases were grouped together because the observed behavior used for diagnostic classification did not differentiate them, would such a system of classification be of any use to those studying etiology? Probably not.

Finally, what about educators or laypeople who have offered the idea that whatever causes autism (or even some cases of intellectual disability without autism) only selectively impairs certain expressive capacities, and who have hypothesized that all capacities are intact but "locked in"? This is essentially the hypothesis offered by promulgators of such treatments as "facilitated communication" (e.g., Biklen, Morton, Gold, Berrigan, & Swaminathan, 1992) or the unpublished "rapid prompting" method. There is very little empirical evidence to support the validity of these methods, but if some cases do respond to these treatments, how would or could these treatment responders be identified by a taxonomic system based on clinical phenomenology? The point is that as the "tent" under which all cases reside becomes larger, etiological factors almost certainly grow more numerous. The result is that a broadly defined diagnosis is likely less informative than a narrow one, which returns us to the question of "why classify" at all if the result is to be a broader classification.

Lack of Specificity of Autism's Signs

The problem of disentangling "what is autism" from "what is intellectual disability" is paralleled by the larger conundrum presented by the lack of specificity associated with each individual criterion used in diagnosing autism. This lack of specificity has been found in DSM-III-R (American Psychiatric Association, 1987) criteria (Spitzer & Siegel, 1990), as well as in DSM-IV criteria (Volkmar et al., 1994). Differential diagnosis of an autism spectrum disorder (or a PDD, in DSM-IV terminology) is complicated by inclusion of clinical phenomena that present very similarly in other neurodevelopmental disorders, such as language disorders, anxiety, compulsiveness, disruptive behavior disorders, and inattention. As are signs of intellectual disability, these clinical phenomena, when associated with other neurodevelopmental/neuropsychiatric disorders, are likely to be of heterogeneous etiology (e.g., genetic or acquired).

Presently, "gold-standard" quantitative clinical assessments for autism spectrum disorders—such as the Autism Diagnostic Interview—Revised (ADI-R; Lord, Rutter, & Le Couteur, 1994; Rutter, Le Couteur, & Lord, 2003) and the Autism Diagnostic Observation Schedule (ADOS; Lord et al., 2000; Lord, Rutter, DiLavore, & Risi, 2001)—do not provide differential diagnostic algorithms for disorders with overlapping symptoms such as those just described. This logically increases the probability that an autism spectrum disorder may be diagnosed instead of another disorder if an individual has marked impairments in overlapping domains, although primary symptomatology may lie elsewhere. (This is discussed further below as possible "diagnostic substitution.")

At this time, development of a taxonomic system for autism that can classify signs and symptoms genetically or neuroanatomically is still premature. The same would be true for any attempt to causally link a putative agent of structural brain damage to a specific localization corresponding to a sign or symptom of autism. Presently, about 1.5% of cases meeting criteria for an autism spec-

trum disorder can be classified as having a known genetic etiology (Constantino, 2008). However, without such a taxonomy, there remains the "chicken-and-egg" conundrum of how clinical features can most specifically be related back to genetic or neural defects as they are identified by etiological studies, and vice versa. Presently, we need a classification system that can link clinical diagnostic/endophenotypic phenomena to specific genetic and neuroanatomical findings. This new kind of classification would be most useful if it could also link targeted educational, behavioral, or psychopharmacological treatments to specific clinical phenomena—the topic that I now consider.

Nosological Implications of Autism as a Heterogeneously Treatment-Responsive Disorder

The same dilemma of linking clinical phenomena to etiology arises when we consider how to link clinical phenomena to treatment responses. The concept of clinical subtypes of autism is only a slightly less bad idea than the current conception of the PDDs in DSM-IV and DSM-IV-TR (American Psychiatric Association, 2000), since subtypes, as long as they are multidimensional, will have limited specificity. Relatively little progress has been made on identifying specific clinical phenomena with good predictive validity for treatment outcomes, aside from one obvious finding (i.e., subjects with more mental retardation demonstrate less benefit from behavioral interventions than those with less mental retardation do; e.g., Smith, Groen, & Wynn, 2000). Beyond study of intellectual impairment as a responder characteristic, little work has been done to demonstrate the responses of specific clinical subgroups to one class of psychoactive drugs, one type of behavioral treatment, or one type of curriculum.

Possible Directions in Clinical Classification

There are several requirements for designing a taxonomy for a heterogeneous, developmental, neurally based, genetically influenced collection of clinical signs like the autism spectrum. First, it would be most helpful to characterize clinical signs with a degree of resolution that would be useful in correlational studies of neural or genetic differences. Second, it would be helpful to characterize clinical signs with a degree of resolution that would facilitate analysis of specific clinical traits most responsive to a specific intervention. The field of behavioral genetics now refers to "endophenotypes"—specific observable phenomena that can be regarded as innate, primary disabilities. With respect to the autism spectrum, candidates might include lack of affiliative drive, defects in auditory processing speed, retention or recall deficits, defects in habituation to novelty, perseverative drive, lack of motor imitative capacity, and the like (Siegel, 2003; Siegel, Ficcaglia, Hayer, & Tanguay, 2007).

Prevalence Rates, Nosology, and the Politics of Autism

It seems clear that during the time when diagnostic criteria for autism spectrum disorders/PDDs have been clinically defined by DSM-IV and DSM-IV-TR, more and more individuals have been found to have these disorders. Some of this increase is probably due to changes in how the diagnostic criteria themselves are understood. Yeargin-Allsop (2008) has shown that rates of identified cases have changed with each successive taxonomic system, starting with Kanner and moving through each iteration of DSM to DSM-IV(-TR). In addition, autistic traits have come to be recognized in very mild forms that border on personality traits. These include being a "loner" (personality trait) versus "absence of peer relationships" (psychopathology); being "detail-oriented" or "rigid" (personality trait) versus "insisting on routines and nonfunctional rituals" (psychopathology); and having "intense hobbies" (personality trait) versus having "narrow interests" (psychopathology). There has been no clear delineation of where a personality trait becomes part of psychopathology, as in adult psychiatry with the state-versus-trait taxonomy (e.g., anxious state vs. anxiety disorder, depressed mood vs. depression). Some of these traits studied singly or in profile have come to be recognized as constituting a "broader phenotype" for autism, such as what has been

dubbed "the geek syndrome" (Silberman, 2001). These may be associated with little or no significant adaptive impairment, or may actually confer benefits with respect to attention, memory, or mathematical skill (e.g., Treffert, 1989).

This lack of clear boundaries between a personality trait and a more pathological manifestation appears to have worked synergistically with the absence of clear differential diagnostic criteria (such as in the "gold-standard" ADI-R and ADOS) to identify an autism spectrum disorder. When there is no comparable algorithm validated to affirm a differential diagnosis, the probability of an autism spectrum disorder can be expected to increase. In the absence of a higher quantitative probability of a condition's being something other than autism, quantitatively borderline cases can accurately be described as "autism-like," even though a diagnosis such as intellectual disability, severe language impairment, an anxiety disorder, or a disruptive behavior disorder might emerge if equal and simultaneous efforts were to be put into its ascertainment. Indeed, Yeargin-Allsop's (2008) data support massive increases in diagnoses of autism spectrum disorders since widespread adoption of the ADI-R and ADOS. At the same time, this lack of boundary between what is "trait-like" and what is "state-like" has corresponded with a few other areas of research and practice: The first is the emergence of the earlier-described work recognizing the existence of a broader autism phenotype. Second is the increased use of nonspecific autism screening measures such as the Childhood Autism Rating Scale (Mesibov, Schopler, Schaffer, & Michal, 1989) and the Gilliam Autism Rating Scale (South et al., 2002), which are mistaken for, misunderstood to be, or accepted as diagnostic measures by parents and nonautism professionals. Third, the fact that educational authorities (e.g., U.S. state departments of education) each have their own separate, less restrictive diagnostic criteria for autism as a condition qualifying a child for an individualized education program. It can be argued that one result of these three factors has been rampant diagnostic substitution, in which individuals previously diagnosed with something else (e.g., a language disorder) are now rather uniformly reclassified as on the autism spectrum (Bishop, Whitehouse, Watt, & Line, 2008; Ihle, Cerros, Sendowski, & Siegel, 2008).

Functional, Clinical Reconceptualization of Autism

The ideas presented so far support the need for a diagnostic system for the autism spectrum disorders that will help researchers studying the etiology of these disorders, as well as clinicians describing specific impairments to be targeted by specific treatments (whether they are pharmacological, behavioral, or educational). A descriptive taxonomy such as the present DSM-IV-TR's, resulting in a diagnosis of either autistic disorder or some other PDD, largely obfuscates what may be a way of specifically linking deficits and treatment responses as well as prognoses.

Defining Autistic Learning Disabilities

The content of this chapter so far lays the foundation for a new kind of taxonomy for autism spectrum disorders, in which individual signs associated with the autism spectrum are operationalized as "autistic learning disabilities" (ALDs) or "autistic learning styles" (ALSs). The purpose of this new kind of taxonomy is to reconceptualize simply descriptive criteria such as those in DSM with operational definitions that refer to functional defects in perception, processing, storage, retrieval, or output of sensory, affective, or cognitive inputs. Each ALD, then, is defined as a characterization of a clinically distinctive aspect of the autism spectrum in terms of one or more of these functional defects. This approach produces a much more specific, fine-grained clinical characterization than exists in DSM. This type of operationalized, more microscopic definition is intended to be closer to an endophenotype. Such operationalized traits can be hypothesized to have a stronger probability of correlating with specific genetic, neural, or neurochemical markers. Equally important, the use of such operationalized traits can be hypothesized to have stronger predictive validity in treatment outcome research than the use of gross autism categories (e.g., autistic disorder vs. PDD not otherwise specified, or

mute vs. speaking) to examine differential treatment response.

Defining Autistic Learning Styles

Conversely, ALSs can be defined as characteristically stronger, intact functions (sometimes exceptional and seen as "splinter," "peak," or "savant" capacities) that individuals with autism spectrum disorders automatically deploy in compensation for their ALDs. These include clinically distinctive, unusual, and sometimes extreme patterns of reliance on visual memory, routines, auditory memory without semantic processing (echolalia), repetitive motor patterns, and the like. These ALSs arise as ways of compensating for disabled functions. ALSs point to dimensions of perception, processing, storage, retrieval, or expression of sensory, affective, and cognitive inputs that are not "hit." This means that an inventory of ALSs (along with ALDs) can be instrumental in selecting or designing interventions for a particular presentation of defects leading to the diagnosis of an autism spectrum disorder. Like the use of ALDs, the use of operationalized ALSs can be hypothesized to have stronger predictive validity in treatment outcome research than the use of gross autism categories to examine differential treatment response. The more operationalized and fine-grained definitions of autism-specific traits in both ALDs and ALSs allow for apportioning variance due to the great individual differences seen among individuals with autism spectrum disorders. More detailed formulations of ALDs and ALSs have been provided elsewhere (Siegel, 2003; Siegel et al., 2007).

A New Taxonomic Framework

Table 28.1 presents a taxonomic framework for reconceptualizing autistic signs and symptoms as ALDs or ALSs. The first column enumerates the social, communicative, and "activities and interests" characteristics constituting the 12 DSM-IV-TR criteria for autistic disorder. In the second column, each of these is reframed in terms of a social type of ALD, a communicative one, or one specific to the individual's activities and interests. At this level, the descriptors can be hypothesized to be direct clinical reference points to endophenotypic aspects of the autism spectrum. In the third column, each ALD from the second column is then translated operationally into a readily measured behavior that can be specifically targeted for intervention. In other work, these behaviorally specified aspects of ASDs are mapped onto specific evidence-based treatment strategies. This allows the taxonomy of ALDs and ALSs to serve as a road map to interventional planning, rather than leading the clinicians to treat "agnostically" according to autism spectrum disorder diagnosis alone with no linkage between deficit and the specificity of the intervention for that deficit. From the perspective of intervention, indeed, this model is critical. It is clear that not all children benefit equally from the same treatment, and it is not clear which children with autism spectrum disorders benefit most from which treatment. The ALD/ALS model provides a method for cross-matching learning strengths and weaknesses with intervention methods that are specific to some area of deficit or play to some area of strength (Siegel, 2003, 2008).

What Should Criteria for DSM-V Autism Spectrum Disorders Look Like?

Do we need DSM-V autism spectrum disorders/PDDs that are conceptually equivalent to those in DSM-IV(-TR)? The ALDs and ALSs formulated here offer a rather different approach: one more specific; one that gives clinical significance to endophenotypic traits; one that holds promise for studies of treatment responder characteristics would be a collection of traits with high total predictive value for the diagnosis of autism spectrum disorders, but none would be completely pathognomonic. Like each specific DSM-IV(-TR) criterion for autistic disorder, each ALD or ALS is conceptualized as highly sensitive though not unique to the autism spectrum. However, adoption of the ALD/ALS approach would support an important conceptual shift: These ALDs and ALSs, though often associated with autism spectrum disorders, may be expected to occur in conjunction with other forms of developmental neuropathology. In the study of polygenic etiologies for heterogeneous disor-

TABLE 28.1. Reconceptualization of Autistic Signs as Autistic Learning Disabilities (ALDs) and Autistic Learning Styles (ALSs)

DSM-IV-TR criteria for autistic disorder[a]	ALDs	"Translational"/operational qualities[b]
"Social" criteria	Social-specific disabilities	
Impairment in the use of nonverbal behaviors, such as eye-to-eye gaze, facial expression, body posture, and gestures used to regulate social interaction	• Problems in comprehension of natural gesture and emotional expression	• Misses some or all information delivered or modified by nonverbal means; message received is absent or attenuated
Failure to develop peer relationships	• Lack of affiliative drive	• Not motivated to "do like" or "be like" peers, which is key to motivating learning in educational settings • Not self-reinforced via altruistic acts
Lack of seeking to share enjoyment, interests, achievements with others (e.g., by bringing, pointing to, or showing off things of interest)	• Lack of joint attention/social referencing • Lack of modeling or imitation • Lack of response to social reward	• In infant, no pointing indicates lack of realization that adult is on "same page" (and can "download" information on caregiver's topic of interest) • No modeling means no acquisition of new schemas via social desire to be more like others • Doesn't "practice" to get attention/praise
Lack of social–emotional reciprocity	• Lacks intersubjective perception/theory of mind	• Others are not understood to have parallel feeling states, so no motive to "do unto others"
"Communication" criteria	Communication-specific disabilities	
Delay in spoken language, not compensated for by other means of communication	• Problems in use of natural gestures and facial expressions	• Misses information imbedded in facial expressions and body language • Nonverbal communication needs to be associated with visuals and with direct teaching of consequences to convey meaning
Marked inability to initiate or sustain conversation	• Lack of affiliative drive • No theory of mind	• No motive to talk when it's chit-chat • Lacks perspective to identify topicality
Stereotyped (e.g., echolalic) repetitive or idiosyncratic use of language	• Gestalt perception and processing	• Language is memorized/reproduced whole; some meaning missed, but may indicate good memory[b]
Lack of varied, spontaneous make-believe play or of social imitation	• Lack of modeling of imitation • Preference for visual over auditory modalities[b]	• Lacks drive for "re-presentation" of experience; attenuated opportunities to understand experience

(cont.)

TABLE 28.1. *(cont.)*

DSM-IV-TR criteria for autistic disorder[a]	ALDs	"Translational"/operational qualities[b]
"Activities and interests" criteria	Activity- and interest-specific disabilities	
Encompassing preoccupation with stereotyped and restrictive patterns of interests, abnormal in intensity or focus	• Stimulus overselectivity/ perseveration on parts of objects	• Low novelty-seeking/high habituation threshold leads to fewer learning experiences • Creates opportunity to motivate with preferred topic as reinforcer/motivator[b]
Compulsive adherence to nonfunctional routines or rituals	• Engagement in routines/ ritual over novelty attenuates learning opportunities	• Creates opportunity to motivate via engagement in highly predictable routines[b]
Stereotyped and repetitive motor movements	• Engagement in repetition attenuates attention to novelty, modulates incoming stimuli	• Can be "read" as need to modify inputs for better "throughput" without overstimulation[b]
Persistent preoccupation with parts (e.g., sensory aspects) of objects	• Sensory modulation difficulties	• Preferred modalities can be used as contextual reinforcers for teaching/therapy[b]

[a]From DSM-IV-TR (American Psychiatric Association, 2000). Copyright 2000 by the American Psychiatric Association. Adapted by permission.
[b]An ALS (i.e., a positive adaptation developed to compensate for an ALD).

ders like the autism spectrum disorders, the ALD/ALS approach encourages a regrouping of individuals by traits, not diagnoses. Moving away from categorical diagnoses for autistic spectrum disorders (as well as other neurodevelopmental disorders with overlapping traits), there is an opportunity to liberate ourselves from conceptually unhelpful groupings such as "autistic disorder," "PDD not otherwise specified," or "Asperger's disorder" and move to a format that encourages etiological research and supports specific treatment of individual traits.

Conclusions

The classification of autism spectrum disorders is one exemplar of the issues faced by contemporary psychiatric nosology. Psychiatry itself is part of another, broader taxonomic scheme. Progress in autism classification, and more broadly in development of a scientific understanding of behavioral atypicality, is dependent on being mindful of the need not to reify any scheme when new kinds of data suggest that the scheme is increasingly obsolete. Recent genetic, neurochemical, and neuroimaging research on the autism spectrum provides convincing support for abandoning a multisymptom taxonomy as irrelevant to the search for etiologies or treatment outcomes.

In a recent commencement address to graduating psychiatry residents at the University of California, San Francisco, the director of the National Institute of Mental Health (himself a graduate of the same psychiatric residency) noted that much of what he had left his training "knowing" in the early 1970s has been disproved or supplanted (Insell, commencement address, June 20, 2008), and that today's operational understanding of brain and behavior was largely unknown only 30-plus years ago. This observation underscores the imperative to move toward a new autism classification that will be sensitive to endophenotypic traits, and toward a multidimensional clinical taxonomy with predictive validity for treatment responses.

References

Altevogt, B. M., Hanson, S. L., & Leschner, A. I. (2008). Autism and the environment: Challenges and opportunities for research, *Pediatrics*, *121*(6), 1225–1229.

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., rev.). Washington, DC: Author.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.

Asperger, H. (1944). Die "autistischen Psychopathen" im Kindersalter. *Archive fur Psychiatrie und Nerrenkrankneiten, 177*, 76–136.

Baron-Cohen, S. (1995). *Mindblindness: An essay on autism and theory of mind*. Cambridge, MA: MIT Press.

Bender, L. (1947). Psychopathic behavior disorders in children. In R. M. Linder (Ed.), *Handbook of correctional psychology* (pp. 360–377). New York: New York Philosophical Library.

Bettelheim, B. (1967). *The empty fortress*. New York: Free Press.

Bettelheim, B., & Rosenfeld, A. (1993). *The art of the obvious: Developing insight for psychotherapy and everyday life*. New York: Knopf.

Biklen, D., Morton, M., Gold, D., Berrigan, C., & Swaminathan, S. (1992). Facilitated communication: Implications for individuals with autism. *Topics in Language Disorders*, *12*(4), 1–28.

Bishop, D. V., Whitehouse, A. J., Watt, H. J., & Line, E. A. (2008). Autism and diagnostic substitution: Evidence from a study of adults with a history of developmental language disorder. *Developmental Medicine and Child Neurology*, *50*(5), 341–345.

Constantino, J. (2008). *The broader autism phenotype and the new genetics of familial and non-familial autism*. Paper presented at the International Meetings for Autism Research, London.

Folstein, S., & Rutter, M. (1977). Infantile autism: A genetic study of 21 twin pairs. *Journal of Child Psychology and Psychiatry*, *18*, 297–321.

Folstein, S., & Rutter, M. (1988). Autism: Familial aggregation and genetic implications. *Journal of Autism and Developmental Disorders*, *18*(1), 3–30.

Frith, U. (1991). *Autism and Asperger syndrome*. Cambridge, UK: Cambridge University Press.

Happé, F (1994). *Autism: An introduction to psychological theory*. London: University College London Press.

Ihle, E., Cerros, C., Sendowski, T., & Siegel, B. (2008). *Closer scrutiny of the autism epidemic*. Paper presented at the International Meetings for Autism Research, London.

Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, *2*, 217–250.

Lord, C., Risi, S., Lambrecht, L., Cook, E. H., Leventhal, B. L., DiLavore, P. C., et al. (2000). The Autism Diagnostic Observation Schedule—Generic: A standard measure of social and communication deficits associated with the spectrum of autism. *Journal of Autism and Developmental Disorders*, *30*(3), 205–223.

Lord, C., Rutter, M., DiLavore, P., & Risi, S. (2001). *Autism Diagnostic Observation Schedule (ADOS) manual*. Los Angeles: Western Psychological Services.

Lord, C., Rutter, M., & Le Couteur, A. (1994). Autism Diagnostic Interview—Revised: A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, *24*(5), 659–685.

Mahler, M. S., & Gosliner, B. J. (1955). On symbiotic child psychosis: Genetic, dynamic, and restitutive aspects. *Psychoanalytic Study of the Child*, *10*, 195–212.

Mesibov, G., Schopler, E., Schaffer, B., & Michal, N. (1989). Use of the Childhood Autism Rating Scale with autistic adolescents and adults. *Journal of the American Academy of Child and Adolescent Psychiatry*, *28*(4), 538–541.

Minshew, N. J. (1992). Neurological localization in autism. In E. Schopler & G. B. Mesibov (Eds.), *High functioning individuals with autism* (pp. 65–90). New York: Plenum Press.

Minshew, N. J. (1996). Brief report: Brain mechanisms in autism: Functional and structural

abnormalities. *Journal of Autism and Developmental Disorders*, *26*, 205–209.

Parks, C. C. (1967). *The siege*. New York: Harcourt, Brace & World.

Rimland, B. (1964). *Infantile autism*. Englewood Cliffs, NJ: Prentice-Hall.

Rutter, M., Le Couteur, A., & Lord, C. (2003). *ADI-R: Autism Diagnostic Interview—Revised*. Los Angeles: Western Psychological Services.

Siegel, B. (2003). *Helping children with autism learn: Treatment approaches for parents and professionals*. New York: Oxford University Press.

Siegel B (2008). *Autistic learning disabilities and autistic learning styles: A framework for developing individualized treatment plans for autistic spectrum disorders*. Washington, DC: American Psychiatric Association.

Siegel, B., Ficcaglia, M., Hayer, C., & Tanguay, P. (2007). Treatment of pervasive developmental disorders. In G. Gabbard (Ed.), *Gabbard's treatment of psychiatric disorders* (4th ed., pp. 79–92). Washington, DC: American Psychiatric Association Press.

Silberman, S. (2001, December). The geek syndrome. *Wired Magazine*, *9*(12). Available at http://www.wired.com/wired/archive/9.12/aspergers.html

Smith, T., Groen, A., & Wynn, J. (2000). Randomized trial of intensive early intervention for children with pervasive developmental disorder. *American Journal on Mental Retardation*, *105*(4), 269–285.

South, M., Williams, B. J., McMahon, W. M., Owley, T., Filipek, P. A., Shernoff, E., et al. (2002). The utility of the Gilliam Autism Rating Scale in research and clinical populations. *Journal of Autism and Developmental Disorders*, *32*(6), 593–599.

Spitzer, R. L., & Siegel, B. (1990). The DSM-III-R field trial of pervasive developmental disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, *26*(6), 855–862.

Szatmari, P. (1991). Asperger's syndrome: Diagnosis, treatment, and outcome. *Psychiatric Clinics of North America*, *14*, 81–93.

Szatmari, P., Archer, L., Fisman, S., Streiner, D. L., & Wilson, F. (1995). Asperger's syndrome and autism: Differences in behavior, cognition, and adaptive functioning. *Journal of the American Academy of Child and Adolescent Psychiatry*, *34*, 1662–1671.

Treffert, D. (1989). *Extraordinary people: Understanding savant syndrome*. New York: Random House.

Volkmar, F., Klin, A., Siegel, B., Szatmari, P., Lord, C., Campbell, M., et al. (1994). Field trial for autistic disorder in DSM-IV. *American Journal of Psychiatry*, *151*(9), 1361–1367.

Wing, L. (1981). Asperger's syndrome: A clinical account. *Psychological Medicine*, *11*, 115–129.

World Health Organization. (1977). *International classification of diseases* (9th rev.). Geneva: Author.

World Health Organization. (1992). *International classification of diseases* (10th rev.). Geneva: Author.

Yeargin-Allsop, M. (2008). The prevalence and characteristics of autism spectrum disorders in the ALSPAC cohort. *Developmental Medicine and Child Neurology*, *50*(9), 646.

CHAPTER 29

Describing Relationship Patterns in DSM-V

A Preliminary Proposal

MARIANNE Z. WAMBOLDT
STEVEN R. H. BEACH
NADINE J. KASLOW
RICHARD E. HEYMAN
MICHAEL B. FIRST
DAVID REISS

Recognition of the interconnections between relationship context and outcomes of clinical interest has led to routine treatment of relationship difficulties in inpatient (Heru, 2006) and outpatient mental health settings. The clinical use of relational interventions is widespread and empirically supported (Wamboldt & Wamboldt, 2000). Myriad treatments for marital/couple conflict have been shown to be effective in controlled clinical trials (e.g., O'Leary & Vega, 2005). Likewise, there is evidence for the effectiveness of parenting interventions (Kazdin, 1998), family interventions in schizophrenia and bipolar disorders (Butzlaff & Hooley, 1998; Rea et al., 2003), and partner involvement in the treatment of substance abuse (O'Farrell & Fals-Stewart, 2003). In some cases, family therapy is even mandated by third-party payers (e.g., as an integral component of inpatient psychiatric hospitalization for children). Indeed, the effects of relationships and relationship events are so central to every aspect of psychopathology and mental health practice that it is hard to imagine how any diagnostic system could deal adequately with issues of impairment (see Whisman, 2007)—let alone etiology (Wamboldt & Reiss, 2006) and treatment (Davies et al., 2006)—without substantial attention to the relationships that provide the primary context for the development, maintenance, and remediation of the disorders of interest. Ironically, the main taxonomic text for psychiatric disorders currently does not describe relationship variables at the same level of detail that it describes other symptom complexes. At the time that DSM-IV was implemented, the editors concluded that research on relationship disorders had not yet reached a level that would allow better inclusion, although reviews of the state of the science and recommendations for diagnostic criteria were commissioned by the DSM-IV editors (see American Psychiatric Association, 1997).

Over the past several years, the Relational Processes Work Group (sponsored by the Fetzer Institute and the National Institute of Mental Health [NIMH]) has reviewed the research background for including relational problems in DSM-V (see Beach et al., 2007). We now translate those research findings into specific nosological recom-

mendations. We advocate sustained and in-depth review of these initial recommendations from three perspectives: their coverage of evidence-based assessments that are essential for adequate mental health care; the practicality of their assessment by mental health professionals without specific training in relationship assessments; and their fit within the evolving DSM system. Four types of relational processes are identified, with each type clearly distinguished in terms of its pattern of association with psychopathology. In view of the importance of the connections between relational processes and mental health, we argue that reliable and standardized assessments of relational processes are needed, and we suggest five possible approaches for providing better coverage of relational processes and relational problems in DSM-V.

How Are Relationships Covered in DSM-IV(-TR)?

The fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) and its text revision (DSM-IV-TR), adopted by the American Psychiatric Association (1994, 2000) to guide reliable diagnosis, made an explicit effort to use the best available science to inform decisions about nomenclature. This approach continued the advances in psychiatric assessment begun with DSM-III. Since 1980, reliability has been improved by the creation of empirically informed criterion sets and the application of structured clinical interviews. The results have been gains in the ease of communication regarding specific problems and in developing science-based recommendations to guide clinical decision making.

DSM-IV(-TR) acknowledges the importance of relationships in several ways, including relational problems referenced in the "V Codes" (e.g., partner relational problem, sibling relational problem, parent–child relational problem); categories of psychosocial problems listed on Axis IV (e.g., problems with primary support group, problems related to social environment); and the Global Assessment of Relational Functioning (GARF) scale in the appendix of criteria sets and axes needing further study (Appendix B). In addition, some relational problems have been addressed in supplemental materials, such as the discussion of abuse and neglect and other relational problems in Volume 3 of the *DSM-IV Sourcebook* (American Psychiatric Association, 1997). Nonetheless, the descriptions of relationship patterns provided by the V Codes are vague, making it difficult to utilize DSM for research or clinical comparisons.

Why Weren't Relationship Disorders Included Before?

Prior groups had advocated the inclusion of relationship disorders in DSM-III and DSM-IV (Anonymous, 1995; Kaslow & Patterson, 2006). This strategy was not implemented, primarily due to the scientific and conceptual question of whether a disorder can reside in more than one person. If we consider one definition of a mental disorder, Wakefield's (1992a, 1992b) "harmful-dysfunction analysis" (see also Wakefield, Chapter 14, this volume), a mental disorder is "a psychological or behavioral condition that satisfies two requirements: (1) it is negative or harmful according to cultural values; and (2) it is caused by a dysfunction (i.e., by a failure of some psychological mechanism to perform a natural function for which it was evolutionarily designed)" (1992a, p. 385). By this argument, validly distinguishing disorder from nondisorder depends on an evolutionary–functional analysis, as well as an additional criterion requiring significant distress or social impairment (Spitzer & Wakefield, 1999). No one argues that some relationship problems can cause harm that is of sufficient severity to be a matter of clinical interest, thus satisfying the first criterion. It is also agreed that some relationship problems are caused by a failure of some psychological or biological mechanism (e.g., the social avoidance seen in patients with autism). At question is whether a relationship problem can be caused by a psychological or biological dysfunction that does not reside solely in one individual—that is, by a failure of psychological mechanisms involving at least two individuals, who are connected in a poor fit for promoting an evolutionarily important relational process (e.g., successful rearing of offspring or maintenance of a caregiving relationship).

Dysfunctions in one individual may not lead to a failure of the relational goal unless there is also a dysfunction in the other individual that accentuates this dysfunction, or at least does not ameliorate it to the extent that most individuals in that culture could ameliorate the dysfunction. This idea is similar to the hypothesis that a disorder in an individual may not arise if the individual has inherited a defective recessive gene from one parent, as long as he or she has inherited a different gene from the other parent that can buffer the effects of the first defective gene. If the person is unfortunate enough to have inherited defective recessive genes from each parent, so that, working together, the genes cannot make a viable protein, the person will express the disorder. We do not label the first defective gene a "disorder," but a "risk factor." Yet, in conjunction with the loss of the gene from another parent that could buffer this risk factor, the individual develops a disorder. That is, the inability of the two genes to work together is what constitutes the basis of the disorder. Similarly, some relational patterns evolve in a predictable harmful direction due to a mismatch of input or output from both individuals, and may thus result in dysfunction despite the lack of a "disorder" in either individual. As a consequence, the pair bond or parenting relationship that has been selected by evolution is unable to succeed in its biological task. As the scientific base for relationship problems continues to evolve, more decision makers may be comfortable with the notion of relationship disorders.

At this time, however, despite very compelling research findings, the broader mental health field may not readily accept the concept of a disorder residing in the relationship of two or more people. This discussion will have to occur over the next 5–10 years, as the field examines fully the implications of current and continuing clinical research on relationships. However, it is not necessary to define relationship problems as mental disorders in order to make important revisions of the DSM system. Thus whether or not these problems are considered disorders should be based on scientific evidence, clear reasoning, and consensus building, as has led to progress in defining other mental disorders. If they are not considered disorders, they may still be described and better utilized within the diagnostic system.

What Are the Implications of Research Since DSM-IV?

As possible future directions for the development of DSM-V are considered, a substantial empirical foundation connecting relational processes to mental disorders and a substantial literature on the nature of these relational processes now exist. The current research literature (much of it summarized in Beach, Wamboldt, Kaslow, Heyman, & Reiss, 2006) illustrates that evidence-based assessments of relationships are essential for adequate mental health assessment and care, and that relational assessments can be effectively accomplished by mental health professionals without specific training in assessment of relationships. This suggests that conclusions regarding the adequacy of the research base to guide inclusion of relational processes in DSM-V may be substantially different from those reached for DSM-IV.

Two types of family relationships appear to be of great importance. Intimate adult relationships and parenting relationships are quite powerful in their effects on psychopathology. This suggests that relationships in the family and events that occur in a family context may be particularly relevant in understanding the development and maintenance of psychopathology. The salience of family relationships and family events (Insel & Young, 2001; Young & Francis, 2008) may arise because humans are "hard-wired" to respond to certain types of relationship events, suggesting that they require special attention in the revision process for DSM. Practical considerations also dictate special attention to family relationships, since family members are often the primary sources of support and influence that are amenable to clinical intervention. In addition, mental health problems in one family member have a direct impact on the emotional well-being of other individuals in the family system. This is not to suggest that other relationships can be ignored, but it does indicate the importance of special and careful attention to family processes in DSM-V.

As the field of psychiatry begins to explore the role of genes within specific environments (Caspi & Moffitt, 2006) as core pathogenic pathways to the development of disorders, it becomes more critical for the official taxonomy to include a mechanism whereby these

critical environmental risk factors can be evaluated and noted. The effects of "high-risk" genes for psychiatric disorders cannot be clearly elucidated if these effects are only salient in certain relationship environments (e.g., one in which parenting is harsh or one in which parenting is neglectful), particularly during key developmental phases. The work of Caspi and colleagues has presented a preliminary look at how relationship variables may interact with risk alleles for genes coding for serotonin receptors (Caspi et al., 2003) or monoamine oxidase inhibitor variants (Caspi et al., 2002) to yield higher rates for depression or antisocial personality disorder; this research is a good example of how this work should proceed over the next decade. Work with nonhuman primates similarly demonstrates that young primates with genetic risk factors will be more likely to exhibit substance abuse behaviors only if they have been raised with peers (Barr & Goldman, 2006)—presumably a more neglectful manner of rearing than being raised by a mother. It would help the field move forward if there were standard and reliable methods of assessing and noting relationship risk factors.

Preliminary Distinctions

Disordered Relational Patterns versus Relational Risk Factors

For now, we have elected to use the term "relational syndromes" to distinguish those relationship patterns that produce clinically recognizable sets of symptoms, are associated with serious distress and malignant disruption of personal functioning, and have evidence of underlying psychological and/or biological processes that are and that should be considered as the focus of clinical attention in the absence of any other currently recognized mental disorder. An example of a relational syndrome is marital/relationship discord syndrome. As with other clinically relevant syndromes (e.g., neuroleptic malignant syndrome), the complex of related signs and symptoms can be reliably identified and treatments can be empirically tested, without need to define the constellation as a disorder per se. These relational problems may never receive proper attention in clinical training programs, in emergency evaluations, or in considerations for pharmacotherapy or psychotherapy unless they are specifically identified as serious problems that can be reliably assessed.

However, not all relationship patterns that should be identified and included in DSM-V will meet the stringent criteria for a relational syndrome. Other relationship problems are of central clinical importance because they can be risk factors for negative outcomes of an established psychiatric disorder or can increase risk for relapse of that disorder, even though they are not problematic in themselves, do not typically interfere with personal functioning in themselves, and would not typically be the focus of clinical intervention in the absence of any other disorder (e.g., expressed emotion, or EE). This second type of relational process is not currently highlighted in DSM. Both relational syndromes and general relational risk factors require reliable assessment to provide optimal guidance for clinical diagnosis and clinical intervention.

General versus Specific Effects of Relational Processes

It is also important to distinguish between relationship processes with robust consequences for maintenance or progression of a number of disorders (e.g., EE or attachment styles) and those relationship problems that have been shown to have an effect in the context of only a single set of disorders (e.g., partner confirmation processes in depression) or that play a salient role in only a single disorder (e.g., intrusive feeding behavior in the context of feeding disorder of infancy or early childhood). Because of their different practical implications, general versus specific relational processes may require different treatment in DSM. General relational processes could be described without reference to other specific mental or physical disorders, and so could be described in their own section of the manual. This would allow general treatment guidelines and case identification methods to be explicated in an efficient manner, would be conducive to scientific communication, and would be clinically useful. Conversely, specific relationship

processes that may be of great importance in one disorder, but of limited relevance for other disorders, could be described in the context of that disorder. At a minimum, the variability in types of relational processes suggests that a comprehensive diagnostic system may need to use more than one approach to describe key relational processes and disorders that may have an impact on the etiology and treatment of mental illness.

Four basic categories of relational process potentially requiring different treatment emerge from the distinctions above: (1) general relational processes that meet criteria as clinical syndromes, requiring treatment of their own accord, such as physical child abuse or marital/relationship discord; (2) general relational processes that are not problematic in and of themselves, but can moderate outcomes for a number of other mental disorders, such as high EE, which moderates outcomes of schizophrenia, depression, and anorexia, among others; (3) specific disordered relational processes that are integral to the development and/or maintenance of specific disorders, such as intrusive parental feeding in feeding disorder of infancy or early childhood, or harsh and inconsistent parenting in conduct disorder; and (4) specific relational processes that are not problematic in themselves but can affect outcomes of particular disorders, such as low parental monitoring in teen drug use. It may be possible for relational processes to move from "specific" to "general" as research regarding the range of effects accumulates. Each of these categories may require different characterization in DSM, and in some cases it may be appropriate to use more than one approach to capture key relational processes.

Is There a Way to Sort Relational Processes into Four Categories?

In the proposed scheme, all relational processes of interest could be parsed into one of four categories formed by the crossing of "general versus specific" with "disordered versus nondisordered." Relational processes would be considered "relational syndromes" if (1) they are negative or harmful according to cultural values; (2) they are caused by a mismatch of psychological mechanisms from each member of the relationship, which makes that relationship unable to succeed in its biological task; and (3) they are not caused by a failure of some psychological mechanism in one individual alone. Accordingly, an empirical criterion would separate syndromal from nonsyndromal behavior and so protect against stigmatizing family members or suggesting premature relationship-oriented intervention. Likewise, relational processes would be considered specific until clearly demonstrated to have implications for multiple forms of psychopathology. As new relational processes are considered for inclusion in DSM, there would be a clear set of both defaults and empirical hurdles for inclusion in each category.

General and Syndromal Relational Processes

Relational problems are already identified as V Codes in DSM-IV(-TR). Of central relevance for DSM-V is that the indicator set for each of these problems needs to be more clearly specified. Given the accumulation of data, specification of reliable and valid indicators are possible for most of the current V Codes, and some (not all) may merit criteria of a relational syndrome. Circumstances under which these relationship syndromes should be a focus of clinical attention also may be an appropriate target for clarification in DSM-V. It may be possible to provide guidance about the threshold for considering a relational syndrome a relevant target for urgent clinical attention, rather than merely a background variable for treatment planning. These problems should be listed on Axis I under a category entitled "relational syndromes." Enhanced descriptions in this section of DSM-V could underscore the value of continued research on these problems, and at the same time provide guidance for clinical intervention. More detailed criteria sets for relational syndromes have been proposed previously, with good convergence across prior efforts (First et al., 2002). Indeed, much of the work required to develop criteria appropriate for clinical assessment has already been accomplished, and some have already been subjected to clinical trials (e.g., Heyman & Slep, 2006, 2009).

General but Nondisordered Relational Processes

The importance of nonsyndromal and general relational processes (e.g., EE, attachment style, patterns of peer affiliation) could be highlighted by describing them as "clinically important relational processes." If they were described in an overall introduction, they might then be included as specifiers in all diagnoses where clinical evidence suggests they play a central role in clinical course. Important contextual processes could be better specified, epidemiologists could examine the prevalence and incidence of key aspects of relational context in the general population, and clinical decision making could be better informed. For example, a child with a diagnosis of major depression who has a secure attachment may have a very different response to antidepressant medications than a child with a diagnosis of depression who has an insecure or disorganized attachment. Clarifying the key relational context of various disorders may elucidate differential responses to certain therapies. Much of the work required to develop specific criteria for clinical assessment has already been accomplished (see, e.g., Hooley & Parker, 2006, for EE).

Specific and Disordered Relational Processes

A third proposal for enhancing the description of relational processes would incorporate a reference to the presence or absence of disorder-specific relational processes by using relational specifiers or by including the relational process in the diagnostic criteria. Specifiers are most often used to describe the course of a disorder or to highlight prominent symptoms. However, specifiers can also be used to indicate associated behavioral patterns of clinical interest. For example, it may be useful to add a specifier to the diagnosis of feeding disorder of infancy or early childhood, to subtype the disorder appropriately as to whether or not intrusive feeding practices are involved. An instance where relational criteria may best be embedded in the diagnostic criteria may be conduct disorder, where research clearly shows the necessity of coercive and inconsistent parenting in the evolution and maintenance of the disorder. Indeed, effective treatment may be impossible without recognizing and delineating the relational aspects of the disorder. Elaborating the embedded relational criteria may be critical for enhancing our understanding of treatment response and relapse. Many other disorders may be amenable to similar analyses and might benefit from addition of specifiers or elaboration of the embedded or implicit relational problems characteristic of the disorder. In some cases the specifier could be described more fully in the "relational problems" section of DSM-V.

Specific but Nondisordered Relational Processes

Some relational processes are of importance for one disorder, but are not yet known to be of general importance—or may have negative consequences only in the context of a specific disorder (e.g., partner verification processes or reassurance seeking in depression, or parental monitoring and supervision in adolescent substance abuse). In such cases, it might be most efficient to discuss the relevance of the process in the text that describes the disorder. As the text is revised to reflect new findings related to particular disorders, the empirical literature linking particular relational processes (whether disordered or nondisordered) to etiology, maintenance, relapse, or burden of the disorder could be included. Alternatively, many of these relational processes could be introduced briefly in the "relational problems" section and then referenced in other sections of DSM.

Even with changes that highlight specific relational disorders and relationship dimensions with particular clinical relevance, incorporating relational context into treatment planning will be challenging. Relational processes are often embedded in larger systems, requiring attention to a multisystemic perspective for the prevention or treatment of many disorders (e.g., Kotchick, Shaffer, Forehand, & Miller, 2001; Liddle, Rowe, Dakof, Ungaro, & Henderson, 2004). It may be useful therefore to provide a guide to thinking about disorders in a relationship context as an appendix of DSM-V. This would follow the same general format as the "cultural formulation" in Appendix I

of DSM-IV(-TR). Although formulations of this kind may unfortunately never become part of universal and routine clinical practice, they can be invaluable for conscientious clinical teaching.

Is Reliable and Valid Assessment Possible?

Current DSM definitions of relationship problems do not include many processes of interest; even for those relationship problems that are mentioned in DSM, the definitions provided do not permit communication of results with the ease customary for other areas dealt with in the manual. This slows both research and practice. Data-based criteria for relational disorders, context, specifiers, and embedded diagnostic criteria, and greater attention to the available data linking particular relational processes and forms of psychopathology, should help correct this problem. Nonetheless, some additional research is necessary (1) to demonstrate the reliability and validity of particular criterion sets; and (2) to determine whether criterion sets are better treated as assessing continuous or categorical constructs, and, if categorical, at what point the transition occurs to problematic intensity.

As an example of how criteria can be developed for relationship syndromes or risk factors, Heyman and Slep (2006) collaborated with the U.S. Air Force to develop and test criteria that would allow a committee to make reliable decisions as to whether an episode of violence within a family would merit the diagnosis of "partner maltreatment" or "child abuse." The issue is seen as clinically important, as only with a reliable manner of assessment can cases be identified as needing services, and only with reliable assessments can varying interventions be tested. These efforts have already led to the development of a set of highly reliable criteria (Heyman & Slep, 2006) for partner and child maltreatment, available from one of us (Richard E. Heyman). Table 29.1 shows an example of these criteria—the criteria set for "partner physical abuse syndrome." As can be seen, the criteria are explicit enough to generate reliable assessment, and they require actions or emotions from both partners involved in the episode.

Overall Recommendations

Two conferences were sponsored by the Fetzer Institute and NIMH[1] to bring together a variety of researchers in the field of relationships, during which a consensus was reached that there are several areas where the inclusion of relational variables would enhance the DSM-V. In addition to adding more specific clarifiers of the relationship problems coded as V Codes (e.g., partner maltreatment), the group agreed that there were other disorders that needed to include some relationship focus. These included depression, substance use disorders, disruptive behavior disorders, eating disorders, sexual dysfunctions, and personality disorders. Evidence was strongest for depression, substance use disorders, and disruptive behavior disorders. However, there was good evidence that family therapy is efficacious for eating disorders (Lock, Couturier, & Agras, 2006); that ratings of attachment style are helpful in understanding and treating patients with personality disorders (Aaronson, Bender, Skodol, & Gunderson, 2006); and that personality disorders predict marital outcomes (Whisman, Tolejko, & Chatav, 2007). The data indicate that these areas require more research into mechanisms of how relationships are involved with the exacerbation or amelioration of these disorders.

The group recommended that a specifier be tested for depressive disorders, such that each could be coded as occurring with or without relationship discord (further coded into partner discord or caretaker–child discord). Each could also be subcoded as occurring with or without a history of childhood abuse (see Table 29.2). Evidence supporting this recommendation included two epidemiological studies demonstrating that couple discord predicts the occurrence of major depression and substance abuse (Whisman, 2007; Whisman, Uebelacker, & Bruce, 2006), and that it predicts a worse course, recurrence, and relapse in adults with depression. Furthermore, treatment research indicates that improving couple discord improves depression outcomes, and that individual therapy for persons in discordant relationships does not yield as good outcomes (Whisman, 2001). For these reasons, coding relational variables will indicate differential treatment planning, meeting the criterion of

TABLE 29.1. Example of Description of a Relational Syndrome: Partner Physical Abuse Syndrome

A. Nonaccidental use of physical force. Physical force includes, but is not limited to, pushing; shoving; slapping; grabbing; throwing; poking; hair pulling; scratching; pinching; restraining; shaking; throwing object at; biting; kicking; hitting with fist; hitting with a stick, strap, or other object; scalding; burning; poisoning; stabbing; applying force to throat; cutting off air supply; holding under water; using a weapon.

B. Significant impact on the victim, as evidenced by any of the following:
   (1) any physical injury (including, but not limited to, pain that lasts at least 4 hours, bruises, cuts, sprains, broken bones, loss of consciousness)
   (2) more than inconsequential fear reaction (see subcriteria below)
   (3) reasonable potential for more than inconsequential physical injury (see subcriteria below), given the inherent dangerousness of the act, the degree of force used, and the physical environment in which the acts occurred

C. The acts of physical force were not committed for any of the following reasons:
   (1) to protect self from imminent physical harm because the partner was in the act of physical force (see subcriteria below)
   (2) to protect self from imminent harm because of partner's threat (see subcriteria below) and a history of more than inconsequential physical injury (see subcriteria below)
   (3) to play with the partner
   (4) to protect partner or another person from imminent physical harm (including, but not limited to, pushing partner out of the way of a car, taking weapon away from suicidal partner, stopping partner from inflicting injury on child). *Note*: Subsequent actions that were not directly protective (e.g., smacking partner for making suicidal gesture) would *not* meet this criterion.

Subcriteria for "More than inconsequential physical injury"

An injury involving any of the following:
   A. Any injury to the face or head.
   B. Any injury to a child under 2 years of age.
   C. More than superficial bruise(s) (i.e., bruise that is other than very light red in color—for example, violet, blue, black—*or* bruises with total area exceeding that of the victim's hand *or* bruises that are tender to light touch).
   D. More than superficial cut(s)/scratch(es) (i.e., would require pressure to stop bleeding).
   E. Bleeding internally or from mouth or ears.
   F. Welt (bump or ridge raised on the skin).
   G. Burns.
   H. Loss of consciousness.
   I. Loss of functioning (including, but not limited to, sprains, broken bones, detached retina, loose or chipped teeth).
   J. Heat exhaustion or heat stroke.
   K. Damage to internal organs.
   L. Disfigurement (including, but not limited to, scarring).
   M. Swelling lasting at least 24 hours.
   N. Pain felt (1) in the course of normal activities and (2) at least 24 hours after the physical injury was suffered.

Subcriteria for "More than inconsequential fear reaction"

Victim's significant fear reaction, as evidenced by both of the following:
   A. Fear (verbalized or displayed) of bodily injury to self or others.
   B. At least one of the following signs of fear or anxiety lasting at least 48 hours:
      (1) persistent intrusive recollections of the incident
      (2) marked negative reactions to cues related to incident, as evidenced by any of the following:
         (a) avoidance of cues
         (b) subjective or overt distress to cues (*Note*: partner can be a cue)
         (c) physiological hyperarousal to cues (*Note*: partner can be a cue)
      (3) acting or feeling as if incident is recurring

(cont.)

TABLE 29.1. *(cont.)*

(4) persistent symptoms of increased arousal, as evidenced by any of the following:
  (a) difficulty falling or staying asleep
  (b) irritability or outbursts of anger
  (c) difficulty concentrating
  (d) hypervigilance (i.e., acting overly sensitive to sounds and sights in the environment; scanning the environment expecting danger; feeling keyed up and on edge)
  (e) exaggerated startle response

Subcriteria for "Protection of self from imminent physical harm because partner was in the act of physical force"

Acts of physical force were committed to protect self from imminent physical harm because the partner was in the act of physical force, as evidenced by all three of the following:

A. Act(s) occurred while other was in the act of using physical force. "In the act" begins with the initiation of motoric behavior that typically would result in an act of physical force (for example, charging to hit him or her) and ends when the use of force is no longer imminent.
B. Sole function of act(s) was to stop other's use of physical force.
C. Act(s) used minimally sufficient force to stop other's use of physical force.

clinical utility (see First et al., 2004) for inclusion in DSM.

A third recommendation was that the descriptive text defining some of the disruptive behavior disorders—in particular, oppositional defiant disorder and conduct disorder—should include some mention of the parent–child relational patterns that have been implicated in the etiology and maintenance of these disorders. In fact, since these parenting patterns are so essential to the development of conduct disorder, they may additionally be coded into the criteria for that disorder (Reid, Patterson, & Snyder, 2002).

TABLE 29.2. Example of Description of a Relational Specifier

Major depressive disorder, recurrent	
296.36	In full remission
296.36a	In full remission, with current relationship discord
296.35	In partial remission
296.35a	In partial remission, with current relationship discord
296.31	Mild
296.31a	Mild, with current relationship discord
296.32	Moderate
296.32a	Moderate, with current relationship discord
296.33	Severe without psychotic features
296.33a	Severe without psychotic features, with current relationship discord
296.34	Severe with psychotic features
296.34a	Severe with psychotic features, with current relationship discord
296.30	Unspecified
296.30a	Unspecified, with current relationship discord

Conclusions

A substantial body of basic research shows that the relational context of disorder is consequential for making decisions about etiology and treatment. The need to make better provisions for the description of relational context is most obvious when the focus is on disorders of childhood. However, the effect of relational context on a wide range of outcomes throughout the lifespan underscores the need to provide criteria for relational syndromes and general relational processes, and to identify relevant relational processes for adult–adult relationships (and possibly for child–child relationships as well). At a minimum, it will be important to better specify the relational problems currently listed as V Codes in DSM-IV(-TR), identify empirically supported relational risk factors, and provide relational specifiers for some forms of psychopathology and relational characteristics of some disorders that may lead to greater precision in their characterization.

The four relational processes suggest four ways in which DSM-V could be improved over DSM-IV(-TR): (1) devising clear criteria for the relational syndromes currently depicted in V Codes—perhaps creating a separate "relational problems" category on Axis I that would provide descriptions of both

relational syndromes and general relational risk factors, with clear criteria for assessing empirically supported contextual processes that might be the targets of clinical intervention, including both family and multisystem contexts; (2) using relational specifier codes to indicate important disorder-specific relational processes; (3) elaborating relationship criteria for existing disorders where appropriate; and (4) describing relevant relational processes in the text associated with the disorders. In addition to these four approaches, it might be useful to provide guidelines for relational formulations in an appendix or a companion volume, to guide clinical use of the new information regarding relational processes. For each option, enhancing the current system could stimulate new research, guide the integration of applied and basic research findings, and inform services. Each relational process could have several well-supported candidates before the DSM-V revision process is concluded. However, it is of greatest importance that attention be directed to testing criterion sets for relational syndromes and general relational processes. Field trials are needed to demonstrate that the proposed nosology (or components of the proposed nosology) could be implemented by residents, social workers, or psychology interns and could yield clinically useful information that could be readily incorporated into psychiatric decision making.

Note

1. The Relational Task Force Work Group held two conferences, with support from the Fetzer Institute and NIMH. The first, a systematic review of the empirical literature on the role of relational processes in psychopathology, was held in Bethesda, Maryland, in March 2005. The second, "Relational Processes and DSM-V: Revising Current Nosology and Improving Assessment," was held in La Jolla, California, in May 2005. Among others, attendees included Lorna Benjamin, Jane Costello, Judith Crowell, Joanne Davila, Thomas Dishion, Michael B. First, Richard Heyman, Kristin Holmes, Nadine J. Kaslow, Danny Pine, David Reiss, Doug Snyder, Marianne Z. Wamboldt, Myrna Weissman, Mark Whisman, Charles Zeanah, representatives from the National Institute on Alcohol Abuse and Alcoholism, and representatives of the DSM-V revision process.

References

Aaronson, C. J., Bender, D. S., Skodol, A. E., & Gunderson, J. G. (2006). Comparison of attachment styles in borderline personality disorder and obsessive–compulsive personality disorder. *Psychiatric Quarterly*, *77*(1), 69–80.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

American Psychiatric Association. (1997). *DSM-IV sourcebook* (Vol. 3). Washington, DC: Author.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.

Anonymous. (1995). A model for the classification and diagnosis of relational disorders: Committee on the Family, Group for the Advancement of Psychiatry. *Psychiatric Services*, *46*(9), 926–931.

Barr, C. S., & Goldman, D. (2006). Non-human primate models of inheritance vulnerability to alcohol use disorders. *Addiction Biology*, *11*(3–4), 374–385.

Beach, S., Wamboldt, M., Kaslow, N., Heyman, R., First, M., Underwood, L., et al. (2007). *Relational processes and DSM-V: Neuroscience, assessment, prevention and intervention*. Washington, DC: American Psychiatric Association.

Beach, S. R., Wamboldt, M. Z., Kaslow, N. J., Heyman, R. E., & Reiss, D. (2006). Describing relationship problems in DSM-V: Toward better guidance for research and clinical practice. *Journal of Family Psychology*, *20*(3), 359–368.

Butzlaff, R. L., & Hooley, J. M. (1998). Expressed emotion and psychiatric relapse: A meta-analysis. *Archivs of General Psychiatry*, *55*(6), 547–552.

Caspi, A., & Moffitt, T. E. (2006). Gene–environment interactions in psychiatry: Joining forces with neuroscience. *Nature Reviews Neuroscience*, *7*(7), 583–590.

Caspi, A., McClay, J., Moffitt, T. E., Mill, J., Martin, J., Craig, I. W., et al. (2002). Role of genotype in the cycle of violence in maltreated children. *Science*, *297*, 851–854.

Caspi, A., Sugden, K., Moffitt, T. E., Taylor, A.,

Craig, I. W., Harrington, H., et al. (2003). Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. *Science*, *301*, 386–389.

Davies, W. H., Satter, E., Berlin, K. S., Sato, A. F., Silverman, A. H., Fischer, E. A., et al. (2006). Reconceptualizing feeding and feeding disorders in interpersonal context: The case for a relational disorder. *Journal of Family Psychology*, *20*(3), 409–417.

First, M. B., Bell, C., Cuthburt, B., Krystal, J., Malison, R., Offord, D., et al. (2002). Personality disorders and relational disorders: A research agenda for addressing crucial gaps in DSM. In D. Kupfer, M. B. First, & D. Regier (Eds.), *A research agenda for DSM-V* (pp. 123–199). Washington, DC: American Psychiatric Association.

First, M. B., Pincus, H. A., Levine, J. B., Williams, J. B., Ustun, B., & Peele, R. (2004). Clinical utility as a criterion for revising psychiatric diagnoses. *American Journal of Psychiatry*, *161*(6), 946–954.

Heru, A. M. (2006). Family psychiatry: From research to practice. *American Journal of Psychiatry*, *163*(6), 962–968.

Heyman, R. E., & Slep, A. M. S. (2006). Creating and field-testing diagnostic criteria for partner and child maltreatment. *Journal of Family Psychology*, *20*, 397–408.

Heyman, R. E., & Slep, A. M. S. (2009). Reliability of family maltreatment diagnostic criteria: 41 site dissemination field trial. *Journal of Family Psychology, 23*(6), 905–910.

Hooley, J. M., & Parker, H. A. (2006). Measuring expressed emotion: An evaluation of the shortcuts. *Journal of Family Psychology*, *20*(3), 386–396.

Insel, T. R., & Young, L. J. (2001). The neurobiology of attachment. *Nature Reviews Neuroscience*, *2*(2), 129–136.

Kaslow, F., & Patterson, T. (2006). Relational diagnosis—a brief historical overview: Comment on the special section. *Journal of Family Psychology*, *20*(3), 428–431.

Kazdin, A. E. (1998). Psychosocial treatments for conduct disorder in children. In P. E. Nathan & J. M. Gorman (Eds.), *A guide to treatments that work* (pp. 65–89). London: Oxford University Press.

Kotchick, B. A., Shaffer, A., Forehand, R., & Miller, K. S. (2001). Adolescent sexual risk behavior: A multi-system perspective. *Clinical Psychology Review*, *21*(4), 493–519.

Liddle, H. A., Rowe, C. L., Dakof, G. A., Ungaro, R. A., & Henderson, C. E. (2004). Early intervention for adolescent substance abuse: Pretreatment to posttreatment outcomes of a randomized clinical trial comparing multidimensional family therapy and peer group treatment. *Journal of Psychoactive Drugs*, *36*(1), 49–63.

Lock, J., Couturier, J., & Agras, W. S. (2006). Comparison of long-term outcomes in adolescents with anorexia nervosa treated with family therapy. *Journal of the American Academy of Child and Adolescent Psychiatry*, *45*(6), 666–672.

O'Farrell, T. J., & Fals-Stewart, W. (2003). Alcohol abuse. *Journal of Marital and Family Therapy*, *29*(1), 121–146.

O'Leary, K. D., & Vega, E. M. (2005). Can partner aggression be stopped with psychosocial interventions? In W. Pinsof & J. Lebow (Eds.), *Family psychology: The art of the science* (pp. 243–266). Oxford, UK: Oxford University Press.

Rea, M. M., Tompson, M. C., Miklowitz, D. J., Goldstein, M. J., Hwang, S., & Mintz, J. (2003). Family-focused treatment versus individual treatment for bipolar disorder: Results of a randomized clinical trial. *Journal of Consulting and Clinical Psychology*, *71*, 482–492.

Reid, J., Patterson, G., & Snyder, J. (2002). *Antisocial behavior in children and adolescents: A developmental analysis and model for intervention*. Washington, DC: American Psychological Association.

Spitzer, R. L., & Wakefield, J. C. (1999). DSM-IV diagnostic criterion for clinical significance: Does it help solve the false positives problem? *American Journal of Psychiatry*, *156*(12), 1856–1864.

Wakefield, J. C. (1992a). The concept of mental disorder: On the boundary between biological facts and social values. *American Psychologist*, *47*(3), 373–388.

Wakefield, J. C. (1992b). Disorder as harmful dysfunction: A conceptual critique of DSM-III-R's definition of mental disorder. *Psychological Review*, *99*(2), 232–247.

Wamboldt, M. Z., & Reiss, D. (2006). Genetic strategies for clarifying a nosology of relational distress. *Journal of Family Psychology*, *20*(3), 378–385.

Wamboldt, M. Z., & Wamboldt, F. S. (2000). Role of the family in the onset and outcome of childhood disorders: Selected research findings. *Journal of the American Academy*

of Child and Adolescent Psychiatry, *39*(10), 1212–1219.

Whisman, M. A. (2001). Marital adjustment and outcome following treatments for depression. *Journal of Consulting and Clinical Psychology*, *69*, 125–129.

Whisman, M. A. (2007). Marital distress and DSM-IV psychiatric disorders in a population-based national survey. *Journal of Abnormal Psychology*, *116*(3), 638–643.

Whisman, M. A., Tolejko, N., & Chatav, Y. (2007). Social consequences of personality disorders: probability and timing of marriage and probability of marital disruption. *Journal of Personality Disorders*, *21*(6), 690–695.

Whisman, M. A., Uebelacker, L. A., & Bruce, M. L. (2006). Longitudinal association between marital dissatisfaction and alcohol use disorders in a community sample. *Journal of Family Psychology*, *20*(1), 164–167.

Young, L. J., & Francis, D. D. (2008). The biochemistry of family commitment and youth competence: Lessons from animal models. In K. Kovner-Kline (Ed.), *Authoritative communities: The scientific case for nurturing the whole child* (pp. 71–86). New York: Springer.

CHAPTER 30

On the Diversity of the Borderline Syndromes

MICHAEL H. STONE

A Historical Note

In earlier times, clinicians used the term *heterogeneity* when referring to the wide varieties and striking dissimilarities they encountered when discussing patients to whose personality disorders the label *borderline* was affixed. The phrase *diversity of the borderline syndromes* answers to an even greater variety of clinical forms. The idea of different syndromes arose out of the growing awareness in the late 19th century that the initial use of borderline referred to a wide, and vaguely demarcated, territory on the psychopathological map situated between the (equally wide and vaguely defined) regions of psychosis and neurosis. Apart from a few authors like Zeller (1844), who championed the notion that psychosis was unitary (he spoke of an *Einheitspsychose*), most psychiatrists in the late 19th century saw the field of psychosis as divided into two broad regions: one relating to thinking (*démence précoce* of Morel; *dementia praecox* of Kraepelin; *schizophrenia* of Bleuler); another, relating to mood (*folie à double forme* of Baillarger; *folie circulaire* of Falret; *manisch–depressives Irresein* of Kraepelin).

Some of the early descriptions of borderline conditions depicted a primarily mood-centered disorder; others, a mostly thought-centered disorder. But this "diversity of two" tended in the United States in the period between the two World Wars to collapse back into a unitary disorder. This was largely because those who were now providing psychotherapy for these not-too-well but not-too-ill "borderline" patients were psychoanalysts. In the mid-20th century, psychoanalysis had grown more prestigious and popular, especially in the United States. *Borderline* still signified something in Kraepelin's "borderland" (*Zwischengebeit*) between psychosis and neurosis, but the term also began to take on the meaning of "borderline with respect to schizophrenia" (little attention was being paid to the possibility of some cases being near-cousins of manic–depression). There was a reason for this, too. Bleuler's (1911/1950) definition of schizophrenia became a successful competitor in the nosological marketplace because he defined the condition in a more hopeful way than Kraepelin (1896) had defined dementia praecox, and he also broadened the definition (which did not become clear till many years later) to include all what was meant by the Munich

school's dementia praecox *plus* a great deal of what we now subsume under the heading of bipolar mania. Thus Bleuler considered the composer Robert Schumann "schizophrenic," though now we understand his condition as distinctly bipolar. Adolf Meyer (1951), who was influential in American psychiatry at midcentury, went so far as to view schizophrenia as a "reaction," in opposition to the then gloomier notion of a genetic underpinning to the condition.

Psychoanalyst Adolf Stern (1938), whose classic paper created a way of thinking about "borderline" that overshadowed the earlier definitions, saw the disorder as borderline to Bleuler's expanded domain of schizophrenia; that is, he viewed the patients he so labeled as *borderline schizophrenics*. Helene Deutsch (1942), in her widely read paper on the "as-if" patient, was unsure whether her patients were at the border of schizophrenia, though she did see them as too well adjusted to be called psychotic. Currently we would regard her "as-if" patients as fragile, clingingly dependent persons, *borderline* perhaps in their *level of function* (in the language of Otto Kernberg)—a concept unrelated to the concept of schizophrenia. In the late 1940s, Hoch and Polatin (1949) embodied the same mindset when they proposed the diagnosis of *pseudoneurotic schizophrenia*. Only in the 1950s and later do we see allusions to a connection of "borderline" to the affective domain, as in the writings of Edith Jacobson (1953).

A little later, we encounter psychoanalysts like Robert Knight (1953) in the 1950s and, more importantly, Otto Kernberg (1967) took the position that borderline ought to be decoupled from the concept of schizophrenia, and established as a clinical entity in its own right—reserved for patients who psychoanalysts thought might be good candidates for the psychoanalytic method, but who, on better acquaintance, turned out to be too fragile for amenability to the classic technique. There were already intimations of this decoupling in the views of such analytic writers as Melitta Schmideberg (1947), whose "stably unstable" patients showed a variety of personality patterns: schizoid, narcissistic, and even antisocial. Similarly, the psychotic character of John Frosch (1960) described a disorder with a mix of antisocial and chaotically impulsive features—too ill for classic analysis, but not in the penumbra of schizophrenia either. The "in-between" patients of Sandor Rado (1962), in contrast, apparently were in this shadow realm; he spoke of such conditions as *compensated schizoadaptation*, which mirrors our contemporary schizotypal personality disorder.

We can already see the beginnings of heterogeneity in the borderline domain: In modern language, various borderline patients of these prominent psychoanalytic clinicians would meet our criteria for narcissistic, schizoid, schizotypal, depressive, antisocial, or dependent personalities. We would need to add "histrionic" as well, once Easser and Lesser (1965) began to publish on the *hysteroid* patient whom they characterized as a caricature of the (better-functioning) Freudian "hysteric": someone, that is, who was irresponsible, sexually chaotic, erratic in work, and turbulent in intimate relationships. The emerging picture was one of proximity to psychosis (though no longer confined to schizophrenia—one could be in the borderland of manic–depression also); fragility (whether in close relationships or in the workplace) in handling the stresses of everyday life; and a mixed personality picture that might contain elements of the disorders just mentioned—or of almost any well-recognized personality configuration.

When the current concept of borderline personality disorder (BPD) was enshrined in DSM-III (American Psychiatric Association, 1980), it had been cobbled together from the prevailing usages of the decade before—namely, those of Otto Kernberg (1967) and John Gunderson (Gunderson & Singer, 1975). Though both are psychoanalysts, the definition promulgated by Kernberg remained nearer to traditional analytic concepts, and emphasized a *weakness in one's sense of identity*, side by side with an *adequate capacity for testing reality*. The first distinguished borderline from neurotic; the second, from psychotic. Here we are speaking of levels of mental function and organization, borderline representing the middle or intermediate level. As such, "borderline personality organization" (BPO) is not a personality disorder at all, since it is not defined as a constellation of personality traits. That said, there are certain traits commonly encountered in patients with BPO: Impulsivity, for example,

is the red thread running not only through Kernberg's definition, but through all the competing definitions over the last 40-plus years. Furthermore, in his seminal paper of 1967, Kernberg delineated subtypes of BPO: the depressive–masochistic, the hypomanic, the paranoid, the infantile (akin to contemporary "histrionic"), and the antisocial, among others. The utility of this differentiation—heterogeneity, if you will—lay in the differing prognostic estimates attached to these subtypes. Borderline-level patients with chiefly depressive–masochistic traits, for example, tended to respond well to the modified form of psychoanalytic treatment Kernberg had been developing (called "expressive" at first; more recently, "transference-focused"). Those with prominent antisocial features responded, as one might expect, much more poorly to Kernberg's or to any other form of psychoanalytically oriented psychotherapy. Indeed, many such patients could be categorized as existing at the borderline of *treatability* altogether, rendering them "borderline" twice over. Kernberg also added to his nonspecific criteria, besides the already mentioned impulsivity, a poor capacity to handle ordinary life stresses (called *low anxiety tolerance*) and a meagerness of hobbies and interests that could maintain a person's equilibrium during stretches of being alone (called poor *sublimatory channeling*). Another important quality of the borderline-level patient is the primitivity of defense mechanisms: reliance, for example, on denial, projective identification, devaluation, or the like, rather than on rationalization, isolation of affect, or repression, let alone on the higher-level mechanisms of resignation and humor.

Grinker, Werble, and Drye (1968) at the Chicago Psychoanalytic Institute in the late 1960s based their definition of borderline on a combination of symptoms and personality traits, such as fearfulness, rejection-sensitivity, and depression, along with such traits as suspiciousness and readiness to anger. They posited four layers or levels within the borderline domain, creating a kind of spectrum with a nearness to psychosis at one end and nearness to neurosis (in the guise of an overly clingy or "anaclitic" depression) at the healthier end. They did not refer to schizophrenia as the psychosis at the opposite end.

During the 1970s, there was some dissatisfaction with the concept of BPO. It had the virtue of separateness from schizophrenia (which was by then undergoing a conceptual overhaul, reverting in part to the more rigorous and objectifiable definitions of Kraepelin), but there was the drawback of its not being understood so well outside the psychoanalytic community—that is, within the larger community of psychiatrists who dealt with borderline patients (by whatever name they were now being called), but who did not have a psychoanalytic background. It was into this wider arena that Gunderson and his colleagues entered, animated by the hope of redefining "borderline" in ways that were more objective and more easily tested via questionnaires and related instruments—such that the concept could be placed on a firmer foundation and one more accessible to the large audience of conventionally trained psychiatrists and psychologists, whether of a primarily clinical, or of a primarily research, orientation.

Gunderson's schema, similarly divorced from schizophrenia, as was Kernberg's and Grinker's, also consisted of a mixture of symptoms and traits, emphasizing diminished work capacity, manipulative suicide threats, and brief psychotic episodes when under stress (these might have a paranoid or depressive quality); the traits included impulsivity and a readiness to anger that was out of proportion to the momentary life events.

In the process of determining what criteria DSM-III should incorporate for "borderline," Robert Spitzer and his group thought at first to divide the clinical data on which they relied into two main entities: one proximate to the concept of schizophrenia; the other, to the concept of the affective disorders. Rather than name these disorders "borderline schizophrenia" and "borderline manic–depression" (or some such), the former *schizotypal personality disorder*; the latter, simply *borderline personality disorder* (our BPD). There was at first a suggestion to call this *emotionally unstable* personality disorder—a pretty accurate descriptor, actually—but the phrase was dropped, partly because of its cumbersomeness and partly because of the already long tradition of using the term borderline, even though the disorder was no longer considered as borderline to any psychosis in particular. This led to the ironic

situation in which the term borderline has no immediately recognizable meaning to those outside the mental health profession, whereas all the other DSM diagnoses point clearly to their underlying meanings, either in straightforward English (e.g., depression, conduct disorder, avoidant personality disorder) or in words whose Latin and Greek roots are easily grasped (e.g., anorexia nervosa, dementia, obsessive–compulsive disorder).

Syndromal Diversity

Strictly speaking, a personality *disorder* consists of a constellation of personality *traits*. As Livesley and others have underlined, each disorder is characterized by a prototypical trait: mistrustfulness in the case of paranoid PD (I use "PD" henceforth as shorthand for personality disorder), self-centeredness in narcissistic PD, aloofness in schizoid PD, and so on. Millon (1999) and his associates make the compelling point that a prototypical approach to personality assessment confers advantages over both the traditional categorical approach (the foundation on which Axis II of DSM rests), which "sacrifices quantitative variation in favor of discrete, binary judgments," and the dimensional approach, which "sacrifices qualitative distinctions in favor of quantitative scores" (p. 99). As these authors assert, the prototype construct represents a synthesis of both the aforementioned models.

Livesley (2001) draws attention to the long-standing preference by psychiatrists for the categorical approach, since this answers to the medical model that teaches us there is a clear distinction between the normal and the sick. While that model may have some purchase in relation to the psychoses, personality disorders tend to shade imperceptibly into the "normal" population; worse yet, some persons appear to behave in radically abnormal fashion in certain interpersonal spheres, yet retaining an equally apparent normality in most other spheres. One has only to think of Hannah Arendt's (1992) work on the "banality of evil," based on the life and crimes of Adolf Eichmann—thoughtful neighbor, family man, and oil company agent turned agent of genocide—to realize that the label "narcissistic personality" was not coextensive with his *entire* personality. Similarly, as Slavenka Drakulić (2004) reminds us, men like Dr. Radovan Karadzić and General Mladić—key perpetrators of the Bosnian genocide in the early 1990s—were not "monsters" through and through—men of ambition, to be sure, but men who lived blameless lives up till the war. I mention these notorious examples by way of underlining the pitfalls of cleaving slavishly to the categorical approach in the domain of personality because this approach blurs one's vision to the multitude of variants, subtypes, shadings, and differences in social function discoverable within the whole spectrum of personalities all pasted with the same categorical label. This has profound implications in forensic work, where—in contentious custody disagreements—some mothers, having been diagnosed with BPD, have quite unfairly lost their children—no allowance having been made for the fact that many women carrying that diagnosis are devoted and consistent in their parental functioning. Only a small minority fail egregiously in their relationship with their children.

Among the DSM personality disorders, at all events, BPD does not enjoy a handy trait prototypicality such as has been assigned to the other disorders. Impulsivity is common to all definitions and to most BPD patients, but it is not the defining feature. A story of manipulative suicide gestures will tend more readily to trigger the suspicion that one is dealing with BPD, yet not all BPD patients show that symptom. *Emotional dysregulation* is perhaps the best candidate for a prototypical feature; yet the phrase does not figure among the former eight (as in DSM-III) or current nine (DSM-IV-TR; American Psychiatric Association, 2000) criteria. Taken in the aggregate, however, affective instability (often with a depressive overlay, such as dysphoria), inordinate anger, frantic efforts to avoid abandonment, and unstable interpersonal relationships all point to this underlying attribute of emotional dysregulation. By the same token, these attributes are commonly noted in patients with depression or with bipolar conditions (especially bipolar II disorder), such that there is some overlap between BPD and certain affective disorders after all. The overlap is not so close as to warrant referring to BPD as a variant of manic–depression, since other causative fac-

tors, unrelated to primary mood disorder, may conduce to the same clinical picture—for example, having endured chronic parental brutality or incest.

The main difficulty standing in the way of assigning a prototypical trait to BPD is, as I emphasize again and again in this chapter, the very diversity in the combinations of attributes all gathered under this one rubric. Paying homage to this diversity, Millon (1999, p. 645) and his colleagues submitted a diagram sketching eight of what they called *prototypical borderline domains*. Realizing that one solitary prototype could not suffice for all that is "BPD," they divided the domain into such territories as the labile, the capricious, the spasmodic, the paradoxical, and so forth—each covering a certain subgroup of patients within the putative disorder. Westen and his coworkers, in their effort to carve out a prototype from the tangle of clinical signs and symptoms associated with BPD, gathered data from a large sample of experienced clinicians who worked with borderline patients. Using their *Q*-sort method (Shedler & Westen, 1998), they demonstrated that the qualities of "more distress" and "emotional dysregulation" stood out more prominently in the impressions of the surveyed clinicians than what were embedded in the DSM description (Conklin & Westen, 2005). Their observation bolstered their conclusion, expressed a year later, concerning prototypes—namely, that prototype-based diagnosis has the advantages of *ease in use, minimization of artifactual comorbidity, and ready translation into both categorical and dimensional diagnosis* (Westen, Shedler, & Bradley, 2006, p. 846). In the array of descriptors in defining BPD, emotional dysregulation would thus stand out as the tallest peak.

It is noteworthy that—from the standpoint of dimensionality—Livesley (2001, p. 23) has advanced a model based on 15 factors and 18 scales derived from those scales. From these he was able to abstract four higher-order factors, answering to all important segments of the personality domain: emotional dysregulation, dissocial behavior, inhibitedness, and compulsivity. The traits included in this schema under the heading of *emotional dysregulation* are anxiousness, affective lability, submissiveness, identity problems, social avoidance, insecure attachment, and cognitive dysregulation. Thus BPD fits within this broader factor, along with the avoidant and dependent personality disorders of DSM.

Use of the term *syndrome* usually implies a combination of symptoms that, taken together, define a well-recognized condition. In psychiatric parlance, a syndrome can also include a number of personality traits that usually accompany the symptoms and contribute to the establishment of the syndrome. Asperger's syndrome (or Asperger's disorder, as DSM calls it) is an example: Combined with the impairments in making eye contact with others and in manifesting appropriate gestures in the interpersonal field (symptoms) are the schizoid-like aloofness and avoidance of others (personality traits).

In relation to *borderline*, diversity of syndromes refers to the differences among the most widely accepted definitions of the term. Within each definition one confronts a wide variety in clinical expression, since borderline patients (by whatever definition) almost invariably show one or more symptom disorders as described in Axis I of DSM, along with the traits of one or several of the other personality disorders—usually in sufficient quantity as to warrant a subsidiary personality diagnosis. Oldham and colleagues (1992) noted that most borderline patients (here focusing on BPD) can be understood as having one, two, or even more additional personality disorders, if one is using a category-based rather than a dimensional approach to diagnosis. Here I am speaking of the *heterogeneity* clinicians encounter when dealing with any large population of borderline patients, irrespective of one's preferred syndromal definition. To add to the complexity and nonuniformity within the borderline domain, it must be acknowledged that matters of social class and culture also affect the clinical expression of borderline states. I expand on this point further on.

The respective definitions of borderline personality by Kernberg and Gunderson already constitute two varieties of the borderline syndrome. Once elements from these two were woven together into the DSM definition of BPD, a third syndrome was created, albeit one with more elements in common with the Gunderson than with the Kernberg definition. Heinz Kohut (1971) had earlier elaborated yet another definition—one that

embodied psychoanalytic principles, with the focus on narcissistic psychopathology. Kohut tended to regard certain narcissistic patients as "borderline" if, after several months in analytic treatment, they failed to show significant gains by his approach. Such a criterion does not honor the traditional principle that a diagnosis should reflect a clinician's impression after the initial consultation with the patient, rather than emerging as a label applied retrospectively. Kohut's patients were generally ambulatory, and to that extent, better-functioning than many patients manifesting the borderline syndrome as defined by Kernberg, by Gunderson, or by DSM.

Figure 30.1 is a Venn diagram depicting these distinctions. Kernberg's BPO occupies a considerably larger space within the domain of psychiatric disorders than do the Gunderson or DSM spaces. The latter two have a large area of overlap; their prevalence is only about a fourth that of BPO. This reflects epidemiological data suggesting that about 2.5% of the population meets DSM criteria for BPD, whereas Kernberg has estimated that 10–11% of the population show the attributes of BPO (though there are no comparable epidemiological data in support of this estimate). Not all patients considered borderline by Kohut criteria meet the more widely accepted criteria, so a fair portion of the Kohut circle lies outside the regions for BPO and BPD.

Perusal of Figure 30.1 helps to make more understandable one of the more puzzling aspects of any dialogue about borderline syndromes and their diversity—namely, that psychotherapists, depending on their training, experience, and interest, tend to specialize in working with patients either at the healthier end or at the more severe end of the functional spectrum. BPD, because it includes in its criteria *recurrent suicidal behavior* and *impulsivity in several areas*, besides being a smaller region within the borderline domain than BPO, is the region in which the sicker patients are concentrated. Some of the patients whom the followers of Kohut might call borderline show so few of the characteristics of the "borderline" as described in the more widely used criteria as to fall outside the (currently standard) domain of borderline altogether. This has led to the somewhat paradoxical situation in which much of the recent literature on borderline personality is devoted to patients who self-mutilate or who make suicide gestures (or the more serious *life-threatening attempts*). This means that the focus in these contributions is on a symp-

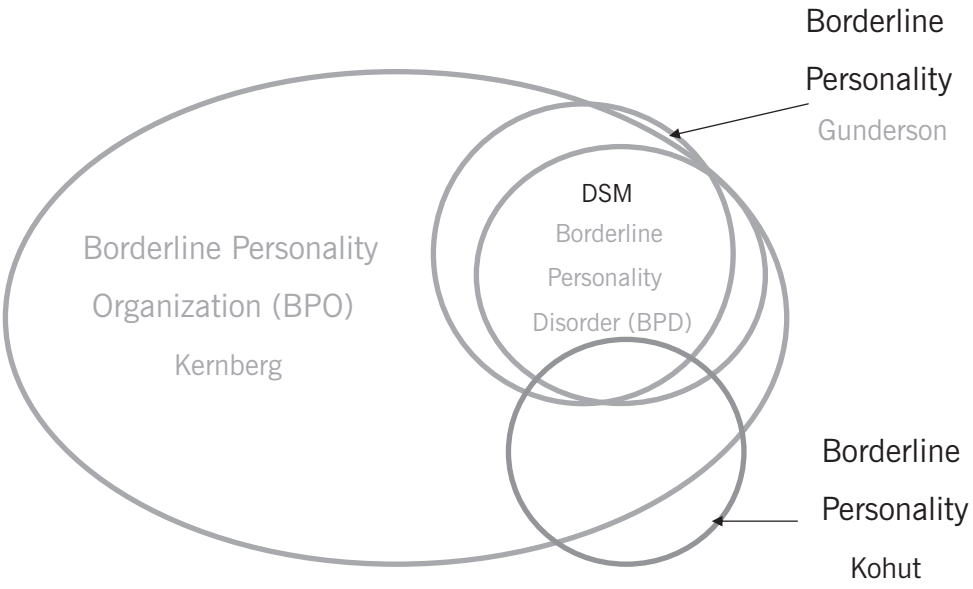


FIGURE 30.1. The main borderline syndromes.

tom, and not on a trait proper of the personality disorder in question. The high rate of completed suicide in borderline patients, as documented in the long-term follow-up studies of the past generation (varying from 3% to 9%) is, as would be expected, concentrated in the BPD moiety. The rate would be correspondingly lower if one's attention were limited to the BPO-as-a-whole group, or to the (still less suicide-prone) BPO-minus-BPD group or Kohut group.

Awareness of these differences leads us to several interesting sidelights. With respect to prognosis, it turns out that the currently most widely accepted forms of psychotherapy for borderline patients have shown good success, and approximately equal success, in reducing the proclivity toward suicidal behaviors *within the first year of treatment*. Such patients would fall preponderantly, if not exclusively, under the heading of BPD—whether by DSM or by Gunderson's criteria. Given that symptoms within the borderline domain tend to be more quickly amenable to treatment than are personality traits (which by definition have a more perduring, inflexible quality), it is not surprising that the literature about symptom reduction in borderline patients is richer than the literature about success over the life course in work, hobbies, friendships, and intimate relations (Freud's *Liebe und Arbeit*), about which data are harder to unearth and slower to be published. Still another facet, which has implications for the diversity of borderline syndromes, concerns the heightened severity of illness in BPD, especially the suicidality-prone component of BPD. For it is in this subgroup (which we could have depicted via a still smaller circle within the BPD circle of Figure 30.1) where we confront the patients who have been incest victims, or who have been outrageously brutalized or neglected in their earlier years. A proportion of such patients will have gone on to develop posttraumatic stress disorder (PTSD) or else a dissociative disorder. Granted that we are now more cautious about making a diagnosis of multiple personality disorder (MPD; now called dissociative identity disorder [DID]) than was the case during its heyday in the late 1980s, investigators who specialized in these areas were wont to declare that such patients might not be true "borderlines" in the first place, but rather examples of special syndromes readily "confused" with BPD, which might better be relabeled as PTSD or MPD/DID. Probably such discussions are best understood as casuistical, to be resolved by noting whether a particular discussant is, diagnostically, a "lumper" or a "splitter." I advocate the *lumper* position here, in the sense that it seems more meaningful to conflate BPD and PTSD when they coexist as "BPD with PTSD features" rather than as a case of PTSD alone—since optimal treatment may depend on recognition of the entire gamut of symptoms and traits that suggested the dual diagnosis originally.

A third facet relevant to syndrome diversity concerns the diversity of symptoms in borderline patients (by any definition) that center on *craving*. One of the BPD criteria in DSM-IV-TR—*frantic efforts to avoid abandonment*—points in this direction. Franticness implies an intense underlying anxiety or restlessness, which a person enveloped in this uncomfortable state can alleviate only through some behavior pattern viewed according to conventional standards as inordinate or excessive. Different borderline patients will engage in different patterns of behavior, answering to the particular cravings to which their nature and nurture have inclined them. Clinicians will rarely encounter a borderline patient who does not exhibit at least one such disorder. Taken together, these disorders, each demarcating a symptom disorder in Axis I, are such regular and *defining* features of "borderline personality" that it would be more accurate to say, "We tend to label persons with craving disorders, once we notice these, as 'borderline,' " rather than to say, "Borderline patients show craving disorders" (as if they were borderline for some other reason). Many of the craving disorders relate to an intense effort to retain, or to recapture, an attachment to some other person, such as a parent or a love object. This is very much in line with the point made by McGuire and Troisi (1998) in their treatise on evolutionary psychiatry: To wit, the patients we diagnose as borderline manifest intense longing for attachment, to achieve which they will go to excessive and inordinate lengths. Table 30.1 shows the main menu of craving disorders and the symptoms generated to satisfy the cravings.

To the extent that these symptoms are often ego-dystonic (behaviors or uncomfort-

TABLE 30.1. Disorders of Craving

Method of reducing anxiety	Associated symptom disorder
Compulsive eating	Bulimia nervosa
Self-starving	Anorexia nervosa (whether to achieve thinness/attractiveness, or to retain a "safe," prepubertal, childlike state)
Sex	Sex addiction, promiscuity, compulsive sexuality (including compulsive masturbation)
Acquisition of objects	Shoplifting, hoarding (as in some cases of obsessive–compulsive disorder)
Self-soothing through a substance	Use of alcohol, heroin
Thrill seeking through a substance	Use of cocaine, methamphetamine
Thrill seeking by other means	Gambling
Keeping a lover	Stalking, jealousy; erotomania (keeping a fantasied lover)
Physical pain (to distract from psychic pain)	Self-mutilation (as in cases of "delicate self-cutting")

able affects from which a person suffers), patients will usually bring these to the attention of a consultant or therapist at the outset. Personality traits generally represent a person's habitual patterns of adaptation to stress and conflict; they are usually ego-syntonic and are not brought so readily to the attention of a therapist (who may have to learn about them through interaction with the person over time). The assumption that a craving disorder equates with "borderline" will not always be correct, despite its usefulness as a first guess; the therapist must then look for confirmatory evidence concerning the more subtle, personality-centered attributes of a borderline disorder. Not every alcoholic (a craving disorder) is also borderline. But a craving disorder such as *stalking* does suggest that several of the BPD descriptors will also be present. Chances are that someone who stalks a current or previous lover will have had a stormy relationship with the lover, will have engaged in frantic efforts (other than just the stalking) to avoid abandonment, will have shown inordinate anger at the lover during any threatened rejection, and may at some point have made a manipulative suicidal gesture by way of coercing the lover to remain or return. These are all items of the BPD definition, helping to "clinch" the diagnosis, once the therapist becomes aware of these traits. Similarly, while not all bulimics are borderline (some allay anxiety through overeating and become obese, but don't show enough of the BPD items to warrant the diagnosis), many of the BPD items are readily discernible in those who induce vomiting so as to maintain normal weight. One such patient known to me used to save her vomitus in plastic garbage bags, which she then stored in her room. Bizarre behavior of this sort is a strong index that the other aspects of a borderline syndrome are also present.

Heterogeneity within the Diversity

The broader the definition of "borderline personality," the larger the number of clinical subtypes that will be found within it. Heterogeneity is the term we customarily use in alluding to this phenomenon. Even if we restrict our attention to BPD as defined in DSM-IV-TR, however, the number of clinically important subtypes is staggering. Approximately half of BPD patients exhibit one or another variety of craving; substance abuse and eating disorders are among the most common. Many of those patients, and most of the remaining group, also suffer from

a mood disorder, which may take the form of major depressive disorder or, less commonly, bipolar II disorder. The more common application of heterogeneity is to the variety of accompanying personality traits other than those related directly to BPD. With respect to the DSM personality categories, many BPD patients show sufficient additional traits to endorse one or more additional disorders, along with a few traits (insufficient to meet full criteria) for still other disorders. Many of these additional traits will belong to the other three disorders of the "dramatic" cluster (Cluster B): histrionic, narcissistic, or antisocial. Also common are some of the disorders not formally included in DSM, such as depressive, depressive–masochistic, hypomanic, and passive–aggressive.

Cultural differences affect the nature of this heterogeneity. In cultures where the outward expression of anger and other strong emotions is discouraged, for example, the other Cluster B disorders are less frequent accompaniments of BPD than are the more "intropunitive" configurations, such as the depressive and depressive–masochistic. This appears to be the case in Japan and in the Scandinavian countries, although this is an impression based on my personal experience in those countries, since pertinent epidemiological data are not available. In Australia, where the culture more closely resembles that of the United States, BPD patients are quite similar to those seen in America.

Social class differences, in contrast, appear not to affect the diversity in either classification or symptomatology, but instead affect amenability to psychotherapy and overall prognosis. I hope to make this clear via some clinical illustrations further on.

As for general prognosis and amenability to therapy, it would be convenient if we could rank-order the other personality disorders with their respective traits in such a way as to indicate to clinicians which combinations within the BPD domain augur the best for treatment, and which combinations the poorest. Any attempt we might make along these lines is nevertheless fraught with so many exceptions as to render any such hierarchy open to challenge. We might suppose that BPD patients whose other prominent traits lie within the Anxious cluster (Cluster C) categories—dependent, avoidant, and obsessive–compulsive—might enjoy a relatively good prognosis, since anxiety is an uncomfortable state that might spur those burdened with it to seek and to remain in treatment until their anxiety is alleviated. The same can be said for BPD patients who have marked depressive and masochistic traits, since these too are associated with discomfort and with voluntary seeking of treatment. Motivation to persevere in treatment would usually be high in these patient groups. BPD patients with significant paranoid, narcissistic, or antisocial traits tend to be "dismissive" in their attachment style; patients with these configurations might be more likely than their anxious counterparts to quit treatment prematurely, meantime forming weaker "therapeutic alliances" with their therapists.

From the standpoint of suicidality, however, *completed* suicides among BPD patients are more numerous among those with marked depressive symptoms and traits (traits such as pessimism, self-hatred, convictions of unworthiness, etc.). Suicide is less common in those with paranoid, narcissistic, and antisocial traits. As for hysterical character, the earlier generation of psychoanalysts defined the condition by paradoxical qualities, such as seductiveness side by side with sexual inhibition; they considered hysteric patients as optimally amenable to psychoanalytic therapy. The histrionic personality disorder of DSM, in contrast, represents a more serious disorder. Here the main impediments to treatment consist in the strong tendency of histrionic patients, especially BPD patients with accompanying histrionic traits, to live in a chaotic fashion, to be highly action-oriented, to be irregular about keeping appointments, and to break off treatment prematurely. One of their defining features is shallowness of affect—a consequence of which is to shift rapidly from enthusiasm to boredom, in therapy as in other activities of their life.

Long-term follow-up of BPD reveals another paradoxical situation: Some patients who responded poorly to treatment at the outset, or even quit therapy against advice, turn out to have done quite well many years later (Stone, 1990). Prognosis cannot therefore be linked too closely to initial treatment response. There is only a modest correlation between initial response to therapy with BPD patients and their life

trajectory, as viewed 10 or more years later (McGlashan, 1986). There is a strong correlation in one area at least: Patients exhibiting BPD × ASPD (antisocial PD) usually do poorly, both at first while in treatment, and also when evaluated many years later. This gloomy outlook relates to those meeting full criteria for ASPD, not to borderline patients whose only foray into antisocial behaviors consists of occasional shoplifting or a rare arrest for driving while intoxicated. Because of the nonuniformity of treatment response and prognosis within any particular trait combination in the BPD domain, we need to be aware that a borderline patient with a few of the less malignant antisocial traits, or one who is paranoid in only a small sector of everyday life, may ultimately do better than the depressive–masochistic patient (whose personality configuration is associated with a generally better prognosis) whose suicidal behaviors are extreme. There will be many tortoise-and-hare situations—that is, where a seemingly well-advantaged BPD patient may have a particular drawback so severe as to render the long-term outcome worse than is the case with another BPD patient, belonging to a supposedly less favorable category, who nevertheless has in strong measure certain assets (charm, intelligence, ambition, etc.) that conduce in the long run to a superior outcome.

The Diversity of Subgroups within the BPD Definition

We can make somewhat better estimates concerning long-term prognosis by paying attention to the particular array of DSM items displayed by any given BPD patient. Now that there are nine such items (in DSM-III there were only eight), of which five or more are needed to establish the diagnosis, there are altogether 256 combinations of five, six, seven, eight, or all nine items that could yield the BPD diagnosis. It would be a fair guess that a BPD patient who does *not* meet the criteria of *impulsivity, recurrent suicidal behavior, inordinate anger*, and *transient stress-related paranoid ideation or dissociative symptoms*, would be easier to work with and have a better outcome than a BPD patient showing all those four (plus one additional item). Some of the combinations are of course much more common than others, and can be divided into several important subgroups. There are, for example, BPD patients who are primarily labile in affect, others who are primarily impulsive and self-destructive, and others in whom the chief difficulty lies in a weak sense of identity. In still others, a tendency toward "cognitive slippage" is their most noticeable feature. Corresponding to these common subtypes are several different medications used in the more severe cases, especially at the beginning of treatment—each answering to a different key subtype and its associated symptom. For instance, an antidepressant for BPD patients with a prominent depressive counterpart; a mood stabilizer for BPD with marked lability of affect or with a tendency to outbursts of anger; and neuroleptics in modest doses for BPD with cognitive difficulties (including mild paranoid tendencies) (Soloff, George, et al., 1986).

Recently, Lenzenweger, Clarkin, Yeomans, Kernberg, and Levy (2008) studied a group of 90 BPD patients by means of a finite-mixture modeling technique—a statistical approach whose aim is to identify coherent and well-defined subgroups within a larger population. They characterized the mixture of subgroups within the larger sample into three main types: one with low levels of antisocial, paranoid, and aggressive traits; a second with elevated paranoid features; and a third with elevated antisocial and aggressive traits. One would expect a more favorable long-term outcome (and better amenability to therapy) in the first group, with the fewest antisocial, paranoid, or aggressive features. Depending on the BPD population being studied, somewhat different subgroup mixtures emerge. Burnette, South, and Reppucci (2007), for example, studied 121 young girls (age range 13–19 years) incarcerated for various offenses. They too found a three-factor solution (via factor analysis), and named their factors *vulnerable* (those with anxiety, withdrawal, somatic concerns), *erratic* (mainly just social anxiety and withdrawal), and *dramatic* (characterized by relational aggression). Since all their subjects had antisocial features, their classification is not isomorphic with that of Lenzenweger and colleagues, though their "dramatic" subjects were probably similar to the third group of the latter researchers (the patients with

antisocial and aggressive traits). Still other investigators favor a different classification of subtypes. A British group focused on associated disorders and stressful life events in BPD patients (rather than simply on the nine items themselves in DSM-IV-TR). They favored a four-class solution, ranging from those with minimal endorsement of BPD symptoms to those with all or nearly all the items, along with a clinical picture of more stressful life events (Shevlin, Dorahy, Adamson, & Murphy, 2007). This is reminiscent of the much earlier four-class solution put forward by Grinker and colleagues (1968), though the four classes were not the same. The Chicago group spoke of a healthier "anaclitically (i.e., dependent) depressed" type, and, at the other extreme, a "border with psychosis" subtype. Their main emphasis, that is, was on clinical function, not on trait prototypicality.

This great diversity within the BPD domain (greater still within the wider realm of BPO) constitutes in itself an argument for flexibility in one's approach to the treatment of borderline patients. They are much less similar to one another than are, say, patients with acute mania—who will, as a group, tend to respond favorably to initial neuroleptics while a simultaneously administered mood stabilizer is more slowly taking effect.

These considerations highlight the difference between commonality and prototypicality of a trait within a particular personality disorder. Identity disorder, for example, appears to be the red thread running through instances of BPO; yet it is less discriminating in defining the class, especially if one shifts attention to the narrower concept of BPD. Jørgensen (2006) regards identity disorder as common in BPD as well, and sees it as linked in part to contemporary culture. Yet identity disorder is, diagnostically, not the *sine qua non* of a borderline patient, as was once thought. As mentioned above, *emotional dysregulation* serves better to discriminate between BPD and non-BPD. The same can be said for interpersonal dysfunction, especially as manifested by "frantic efforts to avoid abandonment" (Clifton & Pilkonis, 2007). A counterpart of the argument concerning emotional dysregulation relates to "effortful control"—a concept allied to the notion of emotional regulation (or lack of it) over one's impulses. In a sample of BPD patients, cluster and profile analysis vis-à-vis emotional control led to differentiation into three groups: those with good control (and the fewest problems in symptoms and in interpersonal relations); those with intermediate levels of control; and finally those with poorest control, who, not surprisingly, had the worst functioning and severest symptoms (Hoermann, Clarkin, Hull, & Levy, 2005).

Genomic Diversity in the Borderline Syndromes

The completion in recent years of human genome mapping has helped to redraw boundaries within the borderline domain. In more instances than was previously possible, these boundaries answer to *genetic*—as opposed to purely clinical/psychological—differences. Kernberg (1967) had earlier advanced the hypothesis that borderline patients often manifested a surplus of "innate aggression"—a concept that had overtones of hereditary predisposition. It was not clear at the time what the particularities of such a predisposition might involve. Because of the phenomenological overlap between an important subset of borderline patients (especially those who were to meet DSM criteria for BPD) and bipolar manic–depression, Akiskal (1981) and I (Stone, 1980) argued that their innate aggression might be related to risk genes for bipolar illness. This seemed plausible, given that manic patients (even in the milder forms) generally show heightened irritability and aggressivity, as well as overactivity, strong sexual drive, rapid speech, and all the other manifestations of having—in metaphorical language—a thermostat set too high.

There is another important symptom component within the borderline domain that relates to excessive anxiety, rather than to excessive aggressivity; the result is inordinate fearfulness in situations that ordinary persons manage with equanimity. This surplus of anxiety fosters the unstable emotionality—otherwise stated, the emotional dysregulation—that defines another main subtype of BPD (Livesley 2008). In this regard, the studies of serotonin gene polymorphisms by Zetzsche and colleagues

(2008) in Munich are informative. Their group studied amygdala volume in 25 female BPD in-patients and a control group. They noted an increased amygdala volume in the BPD patients who had been experiencing their first major depressive episode. Since the amygdala is responsive to strong emotional stimuli, fear in particular, the volume change might then correlate with the hypersensitivity to fear-inspiring stimuli in the subgroup of BPD patients in whom anxiety figured prominently as a symptom. As to the polymorphism in the 5-hydroxytryptamine$_{1A}$ receptor gene, persons homozygous for the C-1019 allele showed the larger amygdala volume; those with a C-G or homozygous G configuration showed a smaller volume. Carriers of the C-1019 allele had more neurotic traits and a worse response to antidepressants than did their G-allele counterparts. The patients with the G allele *and* no major depressive episode had the lowest amygdala volume. Noticing that the high amygdala volume was found only in the BPD patients, Zetzsche and colleagues suggested that "additional disease related factors were required for the effects of genotype on brain structure" (p. 311).

Given that BPD patients report stronger histories of early trauma than do most other personality-disorder patients (Zanarini et al., 1997), Zetzsche and colleagues' (2008) findings are in line with the increasingly important concept of *gene–environment interaction* as the key to understanding the ontogenesis of BPD. It is not yet clear whether the stress-related environmental factors, the genetic factors, or both determine amygdalar changes of the sort reported by the Munich group. This means in effect that it is as yet uncertain whether some persons are born with enlarged amygdalar volume and are, to that extent, more reactive to certain stresses (enough to provoke borderline psychopathology of the hyperanxious type)—or whether the unusual magnitude of stress endured by some children promotes the amygdalar changes, and then only in the group homozygous for the C allele.

Arguing along similar lines, we know from the work of Caspi and colleagues (2002) that adolescent boys with the "short" monoamine oxidase A (MAOA) allele were more prone to conduct disorder if they had also been subjected to maltreatment as children, whereas those with the short allele who had *not* been maltreated were less prone to develop conduct disorder (they might be said to have less "innate aggression"). Those homozygous for the "long" version, in contrast, were less prone to conduct disorder even if they had suffered abuse in childhood. It may be that certain BPD patients of the particularly aggressive/antisocial variety carry the disadvantageous MAOA gene—which would point to yet another genetic source for exaggerated aggressivity, besides the risk genes for bipolar illness.

Thus far, the kinds of chromosome analyses underlying the allelic variations implicated in some of the BPD variants are not widely available. Clinicians, unless they are employed at centers where these tests are easily obtainable, are still left with a classification system for the borderline syndromes that is based on clinical phenomenology (symptoms and traits), with perhaps some additional shrewd guesses about genetic background, when dealing with patients whose family pedigrees contain several close relatives with affective or other related conditions. As Livesley (2008, p. 64) suggests, BPD appears to arise from a multiplicity of interacting factors, each having a small effect, and with no single factor (whether genetic or psychosocial) being necessary or sufficient to produce the disorder. We would have to include brain damage in key regions, resulting from intrauterine and perinatal stresses (e.g., maternal alcoholism or cocaine abuse, deficient oxygenation during delivery, etc.) or childhood head injury. In forensic populations, for example, one encounters aggressive patients who are labeled borderline (with antisocial features) because of accompanying traits and symptoms, and who have suffered brain damage from injury or disease affecting the orbitofrontal areas that subserve decision making and self- control.

Summary

In summary, we have seen that diversity in the domain of the borderline syndromes encompasses the areas of (1) diagnostic taxonomy (with different investigators championing different constellations of diagnostic attributes), (2) symptomatology, (3) trait

constellations, and (4) genomic variants. No one classificatory system is universally applicable, owing to the differing needs and different foci of researchers, forensic psychiatrists, and psychopharmacologists, as well as of the mental health professionals who undertake to treat borderline patients with various forms of psychotherapy. As a further complication, the borderline domain—and the syndromes within it—is so diverse as to make generalizations hazardous. The situation is analogous to that of a term such as *pneumonia*, which once seemed unitary until varieties could be distinguished (pneumococcal, tubercular, viral, pneumocystic, Klebsiella, etc.)—each necessitating a different treatment approach. Some diagnostic terms in psychiatry are closer to the unitary; we do not distinguish hundreds of varieties of bulimia or mania, for example. In those diagnostic domains, there are many "duplicate" cases (amenable to quite similar modes of treatment). In the borderline domain, however, we encounter complexity, with correspondingly fewer "duplicate" cases. In such a situation, there is an urgent need for further study of genetic and other factors of etiological significance, with the goal of creating a more rational taxonomy—one based on more meaningful distinctions that, in turn, inform more effective forms of pharmaco- and psychotherapy, tailored to the specific subtype shown by each erstwhile "borderline" patient. These efforts should result in a more streamlined taxonomy—one in which some cases currently included under the BPD rubric will be pared away, leaving a more homogeneous assemblage of cases identified by a prototypical term (not as yet identified) that does have the connotation of emotional dysregulation.

Case Illustrations

The great diversity of syndromes viewed diagnostically within the borderline domain, even if limited to those patients meeting DSM criteria, is associated not surprisingly with great diversity when viewed from the perspective of outcome. To do justice to this diversity would require many dozens of case histories. Here I provide a small number of clinical vignettes showing some of the diversity in accompanying Axis I conditions and other personality disorders. In any large sample of BPD patients, when followed for long periods (more than 10 years), outcomes cluster around a figure on the Global Assessment of Functioning (GAF) scale (Axis V of DSM) in the mid-60s ("some mild symptoms; some difficulty in social or occupational functioning . . . but generally functioning pretty well, has some meaningful interpersonal relationships") (American Psychiatric Association, 2000, p. 34). But some BPD patients will be found at every level of the scale, from 0 (suicide) to the mid-90s (excellent in all spheres of life). This broad range is reflected in the vignettes below. Names and certain other details have been modified to preserve confidentiality.

Audrey

Hospitalized for the first time when she was 21, Audrey was the only child in a wealthy family, the parents having divorced when she was 4. Her mother, an embittered and imperious woman, prevented all efforts on the part of her father to contact her, with the result that she grew up thinking her father had rejected her totally. She became depressed and suicidal during her first year in college; she made suicidal gestures with overdoses of hypnotics, or else with wrist-cutting. While in the hospital, she was noted to be aloof, haughty, and grandiose, fancying herself highly talented in both art and choreography. Behind this facade, she felt desperately alone and envious of others who had friends and spouses. On her weekend passes she would "go slumming," picking up men in bars for quick sex—though the men sometimes ended up bruising her. She had a dismissive attachment style, characteristic of narcissistic PD (which was her next most prominent personality configuration). When the treatment team arranged for her father to visit, she was openly hostile toward him. Their reconciliation occurred only 10 years later.

Audrey continued in treatment only briefly after leaving the hospital, and took a job in the decorating field. Toward the end of her 30s, she married (she and her husband have no children) and met again with her father; this time she was genuinely happy to see him, and they remained in touch afterward. When contacted 40 years after discharge from the hospital, she had become prominent as a decorator, with shops in several cities; she was very successful

and was living contentedly with her husband. The narcissistic traits that predominated in her earlier years were no longer in evidence. She was no longer "borderline."

Beatrice

Beatrice was the only child of immigrant parents, both of whom were physicians. Her parents argued constantly and were also critical toward her to the point of verbal abuse, particularly if her academic performance fell short of their expectations. Her father was aloof, taciturn, and dour, which added to her sense of having no one that was emotionally available to her. She had few boyfriends during her college years, felt intensely alone much of the time, and began cutting her arms and wrists as a way of relieving tension. She went afterward to medical school, but had a hard time keeping up with the work because of nearly continuous depression, relieved only slightly by antidepressants and psychotherapy. The secret self-cutting persisted. She made several suicide gestures with overdoses, all of which occurred the week before her menstrual period.

While still in school, Beatrice made a disastrous marriage to a controlling and cruel man who hoped to sponge off her, once she entered a practice. He was a gardener who was unemployed most of the time. She clung to him, even though she grew to dislike him, because to be alone was intolerable. Her general mournfulness, coupled with her picking men (her husband being the most extreme example) who humiliated her, gave a depressive–masochistic cast to her personality. When her mother died shortly after Beatrice graduated from medical school, she became seriously suicidal and had to be hospitalized. Now in her late 30s, she works in a group medical practice, was able to leave her husband, and lives alone—still moderately depressed, but able to find some consolation in her many pets. She and her father have become closer to one another. Apart from that, she has few friends, but has built her life around her medical work. Her GAF score, at 10 years after she first sought psychiatric help, is between 55 and 60.

Carol

Carol grew up in a large family, with many half-siblings and stepsiblings, but was the only child of her mother's brief affair with a Native American man. The mother switched lovers frequently, by some of whom she had additional children, but then abandoned the family when Carol was in her early teens. The family was poor, supported mainly by the maternal grandmother, who raised Carol after her mother left when she was 10. It was at that age when her mother was threatened by a jealous lover who pointed a gun at her (the gun failed to go off); the mother then fired her own gun at the man, but missed—whereupon he drove off in a hurry and she left. Carol never saw her mother again. She saw her father only once; he was cordial, but had a great many children by various women, and had no interest in adding her to the pack. Carol became sexually promiscuous and abused alcohol and cocaine. She was content to use men for their money, since she trusted neither men nor women, given the extreme parental neglect she had experienced. Twice during her adolescence, she was raped by a stranger. There was one man she thought she could trust, but when that relationship went sour in her mid-20s, she took a near-fatal dose of barbiturates and was hospitalized for a month. She met another man through the Internet and started a relationship with him. While on a vacation a few months later, they went to a nightclub, where she drank to excess and danced with other men—infuriating her partner. She couldn't understand his jealousy, since "all men are cheats"; therefore, she had a "right" to cheat on him before he could cheat on her. He persuaded her to begin therapy (at his expense), which she did sporadically for a while—talking breezily and dressing seductively, but not taking her problems in relationships at all seriously. She quit therapy after a few months, convinced that the notion of a stable and harmonious relationship with a man (indeed, with anyone) was utterly foreign to her, something she could never hope for. She refused to enroll in a drug/alcohol treatment program, instead continuing to abuse substances as before. She had had only a year of college, but hoped one day to reapply. Her work history was spotty and her earnings meager. Diagnostically, she showed the traits of BPD, along with histrionic and antisocial PDs.

Dorothy

Dorothy grew up in a working-class family and had an older brother. From the age of 5, she had been molested sexually on a weekly

basis by an uncle, and less frequently by her father. The molestation by the father ceased for a time, when the mother discovered the incest, divorced, and permitted visitation by the father only under supervised conditions. Dorothy's sexual contact with her father resumed during her late adolescence, though with the uncle it never ceased until her early 20s. The uncle had threatened to kill her if she ever revealed the secret, so that even in therapy years later, she refused to divulge the full details. She did not date until she was in college, and then only men twice her age whom she would meet in bars. Sex for her became an antidote for her intense loneliness. Even while dating one man for a time, Dorothy would have clandestine "one-night stands" with other men she met in bars or through the Internet. With the man she was dating, she would be very provocative, using the ensuing argument as "proof" that the man was "mean" or no good. On a few occasions when there was a breakup with a boyfriend, she would either cut her wrists or take a mixture of alcohol and methamphetamine.

Despite her chaotic way of living, Dorothy managed to graduate from college and earn a master's degree in finance. Briefly in psychotherapy with a man in her hometown, she seduced him and then stopped "treatment." She was a little more at ease with therapy via Skype over the Internet with a man in a different community. This seemed safer to her, yet she was still reluctant to reveal more than the skimpiest details of her life. She has no interest in men her age, preferring much older men with whom she can recapture the forbidden pleasures of the incestuous relationships. The prospect of marriage and children stir up great conflict: She desires both "because you're supposed to," but fears marriage lest the husband cheat on her as her father did (with her) on her mother, and fears motherhood lest a daughter would be molested just as Dorothy had been. Her major accompanying traits are histrionic, narcissistic, depressive–masochistic, and antisocial (in that order).

Ellen

Ellen had been adopted into a well-to-do family, who later adopted another girl. Little was known of her birth parents, other than that the mother had had depression and an eating disorder. Ellen was raised in a household with strict rules and a punitive atmosphere. If she failed to perform certain menial chores to perfection, she would forfeit the coins in her piggy bank. If she fell off her bike and scraped her knee, she would be mocked for her clumsiness. Though she was academically brilliant, other punishments awaited her on the fortunately rare occasions when she brought home a grade less than an A. In her teens, she frequently ran away from home—usually to neighbors who were kinder to her than were her parents. She became anorexic, and began to abuse cocaine, marijuana, and alcohol.

Ellen considered herself a lesbian, though she had sex with both men and women. She had been raped by a stranger when she was 13. In high school, she was awarded prizes for poetry and was considered an Olympic-level gymnast. Given college scholarships in both areas, she was compelled by her parents to pursue a different course, since they saw no future in either writing or athletics. After moving to a different city to attend college, she became involved with a jealous, possessive man; made a near-lethal suicide attempt with drugs; and was hospitalized with the diagnosis of BPD, major depression, and substance abuse. Thereafter she met and moved in with another man. She spurned his offer of marriage, but remained with him because he was very protective. For a time she did well in her courses, but then became agoraphobic, making it difficult to keep up with her classes or to attend her twice-weekly therapy. When her family withdrew support for her therapy and insisted she return home, she committed suicide.

Francene

Francene was one of three children in an upper-middle-class family; her father was an engineer. During her early teen years she was sexually molested by her father and grandfather, who were both alcoholics. Her schoolwork deteriorated, and she made a suicide gesture necessitating hospitalization. Remaining mute much of the time, she was thought to be "catatonic schizophrenic," and was given numerous electroshock treatments. When these did not improve her condition, she was transferred to a different hospital. There she indulged in various forms of self-mutilation and rarely spoke above a whisper.

Francene remained suicidal and functionally impaired for a year, until she was assigned a new therapist with whom she was better able to communicate. She improved clinically, but still became self-destructive and suicidal dur-

ing her therapist's absences. After 2 years, she was able to leave the hospital; she continued to work with the same therapist for another 8 years. During that time she enrolled in college, graduating with honors, and was accepted at a graduate school in philosophy. She married, had several children, and became a college professor. The molestation, which she was unable to talk about earlier, she was now able to acknowledge. The incorrect diagnosis of schizophrenia was changed to BPD at the second hospital, though the signs of that disorder have long since passed. Currently (40 years since her initial hospitalization), her GAF score is above 90, placing her at the extreme end of *good* functioning for a former patient with BPD.

These vignettes represent only a small sampling of the diversity of clinical presentation within the borderline domain, even when restricted to cases meeting BPD criteria in DSM. Since BPD does not occur alone, but is accompanied by traits of at least one other personality disorder (often enough to endorse a second Axis II PD) and almost always by at least one Axis I symptom condition, this generates approximately 200 combinations of [other PD] × [Axis I condition]. There is the potential for still more combinations, considering that in many BPD patients two or more additional categories of PD are present, along with several (rather than just one) Axis I conditions. From a purely taxonomic standpoint, the multiplicity of combinations may be of interest mostly to diagnosticians and epidemiologists. But the implications for therapists are profound. The treatment for a patient with *BPD × depressive–masochistic PD × dysthymia*, for example, will be very different for a patient with *BPD × histrionic and antisocial PDs × bipolar II disorder and cocaine addiction*, or for yet another with *BPD × narcissistic PD × attention-deficit/hyperactivity disorder and intermittent explosive disorders*. Early background factors related to socioeconomic status, intellectual level, early loss, parental neglect, and abuse of various kinds have their own impact on treatment and outcome. It is complexities of this kind that make comparisons of treatment and outcome so difficult, since they will have been conducted on samples that are not often comparable as to diagnostic subtype and background factors, and usually with *N*'s too small to permit statistical analysis of the many syndromes and subtypes that are nowadays subsumed under the wide umbrella of BPD. Taking this into account, it may prove useful in the future for investigators to describe their BPD patients in greater detail, paying attention to the many combinations alluded to here, allowing for meta-analyses in the literature to focus on more truly comparable subsets of borderline patients. Another alternative may be to do some further nosological surgery on the domain of BPD, dividing it into segments of greater diagnostic similarity.

References

Akiskal, H. S. (1981). Subaffective disorders: Dysthymic, cyclothymic and bipolar II disorders in the "borderline" realm. *Psychiatric Clinics of North America*, *4*, 25–46.

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

Arendt, H. (1992). *Eichmann in Jerusalem: A report on the banality of evil* (rev. ed.). New York: Penguin Books.

Bleuler, E. (1950). *Dementia praecox or the group of schizophrenias* (J. Zinkin, Trans.). New York: International Universities Press. (Original work published 1911)

Burnette, M. L., South, S. C., & Reppucci, N. D. (2007). Cluster B personality pathology in incarcerated girls: Structure, comorbidity, and aggression. *Journal of Personality Disorders*, *21*, 262–272.

Caspi, A., McClay, T., Moffitt, T., Mill, J., Martin, J., Craig, I. W., et al. (2002). Role of genotype in the cycle of violence in maltreated children. *Science*, *297*, 851–854.

Clifton, A., & Pilkonis, P. A. (2007). Evidence for a single latent class of DSM borderline personality disorder. *Comprehensive Psychiatry*, *48*, 70–78.

Conklin, C. Z., & Westen, D. (2005). Borderline personality disorder in clinical practice. *American Journal of Psychiatry*, *162*, 867–875.

Deutsch, H. (1942). Some forms of emotional disturbance and their relationships to schizophrenia. *Psychoanalytic Quarterly*, *11*, 301–321.

Drakulić, S. (2004). *They would never hurt a fly: War criminals on trial in the Hague*. New York: Penguin Books.

Easser, R. R., & Lesser, S. (1965). Hysterical personality: A reevaluation. *Psychoanalytic Quarterly*, *34*, 390–402.

Frosch, J. (1960). Psychotic character. *Journal of the American Psychoanalytic Association*, *8*, 544–551.

Grinker, R. R., Sr., Werble, B., & Drye, R. C. (1968). *The borderline syndrome*. New York: Basic Books.

Gunderson, J. G., & Singer, M. T. (1975). Defining borderline patients: An overview. *American Journal of Psychiatry*, *132*, 1–10.

Hoch, P. H., & Polatin, P. (1949). Pseudoneurotic forms of schizophrenia. *Psychiatric Quarterly*, *23*, 248–276.

Hoermann, S., Clarkin, J. F., Hull, J. W., & Levy, K. N. (2005). The construct of effortful control: An approach to borderline personality disorder heterogeneity. *Psychopathology*, *38*, 82–86.

Jacobson, E. (1953). Contribution to the metapsychology of cyclothymic depression. In P. Greenacre (Ed.), *Affective disorders* (pp. 49–83). New York: International Universities Press.

Jørgensen, C. R. (2006). Disturbed sense of identity in borderline personality disorder. *Journal of Personality Disorders*, *20*, 618–644.

Kernberg, O. F. (1967). Borderline personality organization. *Journal of American Psychoanalytic Association*, *15*, 641–685.

Knight, R. P. (1953). Borderline states. *Bulletin of the Menninger Clinic*, *17*, 1–12.

Kohut, H. (1971). *The analysis of the self*. New York: International Universities Press.

Kraepelin, E. (1896). *Psychiatrie: Ein Lehrbuch für Studierende und Ärzte* (5th ed.). Leipzig: Barth.

Lenzenweger, M. F., Clarkin, J. F., Yeomans, F. E., Kernberg, O. F., & Levy, K. N. (2008). Refining the borderline personality disorder phenotype through finite mixture modeling: Implications for classification. *Journal of Personality Disorders*, *22*, 313–331.

Livesley, W. J. (2001). Conceptual and taxonomic issues. In W. J. Livesley (Ed.), *Handbook of personality disorders: Theory, research, and treatment* (pp. 3–38). New York: Guilford Press.

Livesley, W. J. (2008). Toward a genetically-informed model of borderline personality disorder. *Journal of Personality Disorders*, *22*, 42–71.

McGlashan, T. H. (1986). The Chestnut Lodge follow-up study: III. Long term outcome of borderline patients. *Archives of General Psychiatry*, *43*, 20–30.

McGuire, M., & Troisi, A. (1998). *Darwinian psychiatry*. New York: Oxford University Press.

Meyer, A. (1951). *Collected papers* (E. E. Winters, Ed., 4 vols.). Baltimore: Johns Hopkins University Press.

Millon, T. (with Associates). (1999). *Personality-guided therapy*. New York: Wiley.

Oldham, J. M., Skodol, A. E., Kellman, H. D., Hyler, S. E., Rosnick, L., & Davies, M. (1992). Diagnosis of DSM-III personality disorders by two structured interviews: Patterns of comorbidity. *American Journal of Psychiatry*, *149*, 213–220.

Rado, S. (1962). Theory and therapy: The theory of schizotypal organization and its application to the treatment of decompensated schizotypal behavior. In S. Rado, *Collected papers: Vol. 1. Psychoanalysis of behavior* (pp. 127–140). New York: Grune & Stratton.

Schmideberg, M. (1947). The treatment of psychopaths and borderline patients. *American Journal of Psychotherapy*, *1*, 45–55.

Shedler, J., & Westen, D. (1998). Refining the measurement of Axis II: A Q-sort procedure for assessing personality pathology. *Assessment*, *5*, 333–353.

Shevlin, M., Dorahy, M., Adamson, G., & Murphy, J. (2007). Subtypes of borderline personality disorder, associated clinical disorders and stressful life-events: A latent class analysis based on the British Psychiatric Morbidity Survey. *British Journal of Clinical Psychology*, *46*, 273–281.

Soloff, P. H., George, A., Nathan, R. S., Schulz, P. M., Ulrich, R. F., & Perel, J. (1986). Progress in the pharmacotherapy of borderline disorders. *Archives of General Psychiatry*, *43*, 691–697.

Stern, A. (1938). Psychoanalytic investigation and therapy in the borderline group of neuroses. *Psychoanalytic Quarterly*, *7*, 467–489.

Stone, M. H. (1980). *The borderline syndromes*. New York: McGraw-Hill.

Stone, M. H. (1990). *The fate of borderline patients*. New York: Guilford Press.

Westen, D., Shedler, J., & Bradley, R. (2006). A prototype approach to personality disorder di-

agnosis. *American Journal of Psychiatry*, *163*, 846–856.

Zanarini, M. C., Williams, A. A., Lewis, R. E., Reich, R. B., Vera, S. C., Marino, M. F., et al. (1997). Reported pathological childhood experiences associated with the development of borderline personality disorder. *American Journal of Psychiatry*, *154*, 1101–1106.

Zeller, E. (1844). Die Einheitspsychose. *Allgemeine Zeitschrift für Psychiatrie*, *1*, 1–79.

Zetzsche, T., Preuss, U. W., Bondy, B., Frodi, T., Zill, P., Schmitt, G., et al. (2008). S-HT_{1A} receptor gene C-1019 G polymorphism and amygdala volume in borderline personality disorder. *Genes, Brain and Behavior*, *7*, 306–313.

Author Index

Subject Index

Page numbers followed by *f* indicate figure; *n*, note; and *t*, table