international perspectives in philosophy and psychiatry

alternative perspectives on psychiatric validation

EDITED BY PETER ZACHAR, DROZDSTOJ ST. STOYANOV, MASSIMILIANO ARAGONA, AND ASSEN JABLENSKY

Alternative Perspectives on Psychiatric Validation

International Perspectives in Philosophy and Psychiatry

Series editors: Bill (K. W. M.) Fulford, Lisa Bortolotti, Matthew Broome, Katherine Morris, John Z. Sadler, and Giovanni Stanghellini

VOLUMES IN THE SERIES:

Portrait of the Psychiatrist as a Young Man
The Early Writing and Work of R. D. Laing, 1927–1960
Beveridge

Mind, Meaning, and Mental Disorder 2e
Bolton and Hill

What is Mental Disorder?
Bolton

Delusions and Other Irrational Beliefs
Bortolotti

Postpsychiatry
Bracken and Thomas

Philosophy, Psychoanalysis, and the A-Rational Mind
Brakel

Unconscious Knowing and Other Essays in Psycho-Philosophical Analysis
Brakel

Psychiatry as Cognitive Neuroscience
Broome and Bortolotti (eds)

Free Will and Responsibility
A Guide for Practitioners
Callender

Reconceiving Schizophrenia
Chung, Fulford, and Graham (eds)

Darwin and Psychiatry
De Block and Adriaens (eds)

Oxford Handbook of Philosophy and Psychiatry
Fulford, Davies, Gipps, Graham, Sadler, Stanghellini, and Thornton

Nature and Narrative
An Introduction to the New Philosophy of Psychiatry
Fulford, Morris, Sadler, and Stanghellini (eds)

Oxford Textbook of Philosophy and Psychiatry
Fulford, Thornton, and Graham

The Mind and its Discontents
Gillett

Is Evidence-based Psychiatry Ethical?
Gupta

Thinking Through Dementia
Hughes

Dementia
Mind, Meaning, and the Person
Hughes, Louw, and Sabat (eds)

Talking Cures and Placebo Effects
Jopling

Philosophical Issues in Psychiatry II: Nosology
Kendler and Parnas

Philosophical Issues in Psychiatry III
The Nature and Sources of Historical Change
Kendler and Parnas

Discursive Perspectives in Therapeutic Practice
Lock and Strong (eds)

Schizophrenia and the Fate of the Self
Lysaker and Lysaker

Responsibility and Psychopathy
Malatesti and McMillan

Body-Subjects and Disordered Minds
Matthews

Rationality and Compulsion
Applying Action Theory to Psychiatry
Nordenfelt

Philosophical Perspectives on Technology and Psychiatry
Phillips (ed.)

The Metaphor of Mental Illness
Pickering

Mapping the Edges and the In-between
Potter

Trauma, Truth, and Reconciliation
Healing Damaged Relationships
Potter (ed.)

The Philosophy of Psychiatry
A Companion
Radden

The Virtuous Psychiatrist
Radden and Sadler

Addiction and Weakness of Will
Radoilska

Autonomy and Mental Disorder
Radoilska (ed.)

Feelings of Being
Ratcliffe

Recovery of People with Mental Illness
Philosophical and Related Perspectives
Rudnick (ed.)

Values and Psychiatric Diagnosis
Sadler

Disembodied Spirits and Deanimated Bodies
The Psychopathology of Common Sense
Stanghellini

One Century of Karl Jaspers' Psychopathology
Stanghellini and Fuchs

Emotions and Personhood
Stanghellini and Rosfort

Essential Philosophy of Psychiatry
Thornton

Empirical Ethics in Psychiatry
Widdershoven, McMillan, Hope, and Van der Scheer (eds)

The Sublime Object of Psychiatry
Schizophrenia in Clinical and Cultural Theory
Woods

Alternative Perspectives on Psychiatric Validation
DSM, ICD, RDoC, and Beyond
Zachar, Stoyanov, Aragona, and Jablensky

Alternative Perspectives on Psychiatric Validation

DSM, ICD, RDoC, and Beyond

Edited by

Peter Zachar

Drozdstoj St. Stoyanov

Massimiliano Aragona

Assen Jablensky

OXFORD
UNIVERSITY PRESS

OXFORD
UNIVERSITY PRESS

Great Clarendon Street, Oxford, OX2 6DP,
United Kingdom

Oxford University Press is a department of the University of Oxford.
It furthers the University's objective of excellence in research, scholarship,
and education by publishing worldwide. Oxford is a registered trade mark of
Oxford University Press in the UK and in certain other countries

First Edition published in 2015

Impression: 1

Published in the United States of America by Oxford University Press
198 Madison Avenue, New York, NY 10016, United States of America

British Library Cataloguing in Publication Data
Data available

Library of Congress Control Number: 2014940087

ISBN 978–0–19–968073–3

Printed and bound in Great Britain by
Clays Ltd., St Ives plc

Acknowledgments

The editors would like to thank Charlotte Green and Martin Baum for their support and assistance during the preparation of this volume. Kitty Walker also helped manage chapter submissions and deadlines. Thank you also to the entire production team employed by Oxford University Press. Thanks especially to Bill Fulford, who supported and encouraged this project from its inception.

Contents

List of Figures and Tables

List of Contributors

Massimiliano Aragona, MD, BPhil
Professor of General Psychology
Faculty of Philosophy and
Professor of Phenomenological Psychopathology
School of Psychiatry
La Sapienza University
Rome, Italy

Claudio E. M. Banzato, MD, PhD
Associate Professor of Psychiatry
Department of Psychiatry
University of Campinas (UNICAMP)
Campinas, Brazil

German E. Berrios, MD
Professor of the Epistemology of Psychiatry
Robinson College and Department of Psychiatry
University of Cambridge
Cambridge, UK

Stefan J. Borgwardt, MD
Professor of Neuropsychiatry
Department of Psychiatry (UPK)
University of Basel
Basel, Switzerland

Lisa Bortolotti, PhD
Professor of Philosophy
Department of Philosophy
University of Birmingham
Birmingham, UK

Matthew R. Broome, PhD, MRCPsych
Senior Clinical Research Fellow
University of Oxford
Department of Psychiatry
Oxford, UK

C. Robert Cloninger, MD, PhD
Wallace Renard Professor of Psychiatry, Genetics, and Psychology
Washington University School of Medicine
St. Louis, MO, USA

Assen Jablensky, MD
Winthrop Professor of Psychiatry
The University of Western Australia
Centre for Clinical Research in Neuropsychiatry (CCRN)
Perth, Australia

Kathryn L. Jacobs, BS
Department of Psychology
University of Minnesota
Minneapolis, MN, USA

Jared W. Keeley, PhD
Assistant Professor of Psychology
Department of Psychology
Mississippi State University
Starkville, MS, USA

Robert F. Krueger, PhD
Hathaway Distinguished Professor of Psychology
Department of Psychology
University of Minnesota
Minneapolis, MN, USA

Michael Loughlin, PhD
Professor of Applied Philosophy
Department of Interdisciplinary Studies
Manchester Metropolitan University Crewe
Cheshire, UK

Ivana S. Marková, MD
Reader in Psychiatry
Centre for Health and Population Sciences
Hull York Medical School
University of Hull
Hull, UK

Juan E. Mezzich, MD, PhD
Professor of Psychiatry
Icahn School of Medicine at Mount Sinai
Editor in Chief
International Journal of Person Centered Medicine
New York, NY, USA

Andrew Miles, MSc, MPhil, PhD
Professor
World Health Organization Collaborating Centre for Public Health Education and Training
Faculty of Medicine
Imperial College
London, UK

René J. Muller, PhD
Department of Psychiatry
The Johns Hopkins University School of Medicine
Baltimore, MD, USA

Dominic Murphy, PhD
Associate Professor of Philosophy
Unit for History and Philosophy of Science
University of Sydney
Sydney, Australia

James Phillips, MD
Clinical Professor of Psychiatry
Yale School of Medicine
New Haven, CT, USA

Adriano C. T. Rodrigues, MD, PhD
Lecturer in Psychiatry and Medical Psychology
Health Sciences Center
Federal University of Piaui
Teresina, Brazil

Nigel Sabbarton-Leary, PhD
Independent Researcher
UK

Ihsan M. Salloum, MD, MPH
Professor of Psychiatry and Behavioral Sciences
Miller School of Medicine
University of Miami
Miami, FL, USA

Drozdstoj St. Stoyanov, MD, PhD, PGCert
Professor
Department of Psychiatry and Medical Psychology
Medical University of Plovdiv
Plovdiv, Bulgaria

Somogy Varga, PhD
Assistant Professor of Philosophy
Department of Philosophy
University of Memphis
Memphis, TN, USA

Peter Zachar, PhD
Professor of Psychology
Department of Psychology
Auburn University Montgomery
Montgomery, AL, USA

Part 1

Prologue

Chapter 1

Introduction: The concept of validation in psychiatry and psychology

Peter Zachar and Assen Jablensky

1.1 Introduction

The roots of validity lie in logic, referring to whether an instance of reasoning conforms to correct rules (formal validity) and to whether the conclusion is true (material validity). How we progressed from logical validity to a problem about the validity of diagnostic constructs is not a simple tale. Although the path from logic to the current notions of validation in psychiatry travels through the science of psychological measurement, one has to be careful about construing parallel developments in psychiatry and clinical psychology as causally related and thereby inferring connections that never existed.

Psychologists began using reliability and validity to think about the technology of measuring inferred psychological attributes as two interchangeable terms for "adequacy" (Leuba 1899; Starch 1915). Subsequently, they employed them to distinguish measuring a psychological attribute consistently—*reliability*—from measuring it accurately—*validity* (Thurstone 1931; Adams 1936). As we shall see, in psychology the problem of accurately measuring psychological attributes came to be seen as the problem of measuring theoretical constructs, whereas in psychiatry the primary concern was one of confirming disease status. Over the years, however, the validity problem in psychiatry has also evolved into a problem about theoretical constructs.

1.2 Validity in Mid-Twentieth-Century Science and Philosophy

In the middle years of the twentieth century, the school of logical positivism was the dominant approach in the philosophy of science. One of the goals of this school was to elucidate the logical structure of scientific reasoning. It therefore made sense for the logical positivists to refer to the *validity* of scientific theories. According to them, validity was largely formal. For example, on the logical positivist's account, confirmation and explanation depended on conforming to proper logical syntax.

The positivist (or empiricist) aspect of this school held that science seeks to discover and systematize regularities in the network of observations that are part of experience. Logical positivism also updated empiricism to better conform to twentieth-century science (especially relativity theory and quantum physics). Networks of scientific concepts, the logical positivists agreed, also contain theoretical constructs such as force and electron (in physics) or general intelligence (in psychology).

What does psychiatric diagnostic classification look like from the perspective of such an empiricism? According to this particular empiricist view, in psychiatry a regular pattern of characteristic self-disturbances, hallucinations, delusions, and a decline in functioning is given a name such as "schizophrenia." In the most minimalist form of empiricism, schizophrenia is a descriptive term (or inductive summary) that refers only to the pattern of observed signs and reported symptoms.

Less minimally, schizophrenia is a theoretical construct that enables clinicians to organize signs and symptoms into a coherent framework. The construct of schizophrenia also has surplus meaning by virtue of its association with other theoretical constructs such "psychosis," and "disease." In general, empiricists are instrumentalist and anti-realist about theoretical constructs, viewing them like they do socioeconomic status. A person's socioeconomic status is not a cause of income level and educational attainment; rather, it is a handy abbreviation for income and educational attainment patterns in a population.

According to Markus and Borsboom (2013), the psychologists who introduced the notion of construct validity increasingly went beyond the empiricism of the logical positivists and adopted scientific realism about psychological attributes such as intelligence, extroversion, and schizophrenia. According to realism about constructs, differences in test scores are caused by people's position on the psychological attribute being measured. These attributes are considered to exist independently of being measured.

1.3 Science and Validity in Psychiatry

For nearly the entire twentieth century psychologists debated whether the latent variable of general intelligence is a real attribute/natural kind or a mathematical construct whose meaning changes depending upon how it is measured. Proposed mid-century largely to address the clinical constructs measured by instruments such as the *Rorschach Inkblot Test* and the *Minnesota Multiphasic Personality Inventory* (MMPI), the notion of construct validity redrew the lines of the ongoing debate. After the lines were redrawn, schizophrenia and hysteria were declared to be unproblematical constructs—but constructs that cannot be reduced to how they are measured and that can refer to something real.[1]

The term construct validity was introduced in an American Psychological Association Technical Report in 1954. The committee that prepared this report was chaired by Lee Cronbach. According to Cronbach (1989), the idea of construct validation was proposed by committee member Paul Meehl. It had been worked out in cooperation with Meehl's colleagues at the Minnesota Center for the Philosophy of Science. Meehl expanded on these ideas with Cronbach in a 1955 article titled *Construct validity in psychological tests*. One of the main ideas of this article was that the validation of a test is analogous to the validation of a theory in science (according to the strictures of logical positivism/empiricism with scientific realism tacked on).

If Cronbach and Meehl's article was a watershed event for construct validity in psychology, Robins and Guze's (1970) article *The establishment of diagnostic validity in psychiatric illness: Its application to schizophrenia* played a similar role in psychiatry. In their article Robins and Guze said that diagnosis must be a scientific classification, and valid classification is essential to science. Rather than worry about the validity of the diagnosis of a single patient as would be typical in medicine, they were concerned about the validity of schizophrenia—and later about classification in general (Woodruff et al. 1974).

Most commentators consider this article to be an attempt to resurrect a psychiatry of disease entities similar to that advocated by Emil Kraepelin. Kraepelin proposed that dementia praecox (renamed schizophrenia by Bleuler in 1908) and manic depressive illness were two different entities, with the first having a deteriorating course and the second involving recovery and re-occurrence over time. In this tradition, Robins and Guze's over-arching construct was "psychiatric illness." They proposed five groups of validators—clinical description, laboratory studies, differentiation from other disorders, studies of outcome, and family studies—each of which were predictions about what would be observed if a diagnostic construct such as schizophrenia conformed to their illness construct.

In the 1950s there was little interest in diagnosis among American psychiatrists, with one important exception being a group of scientifically oriented psychiatrists at Washington University in St. Louis. Subsequently named the neo-Kraepelinians, this group included Robins and Guze. They introduced the concept of *diagnostic validity* to describe the research programs that nosologically oriented psychiatrists were already conducting (Goodwin et al. 1969; Purtell et al. 1951; Robins and Mensh 1954). Validity was also a helpful term for encouraging psychiatrists to conduct research that could disprove Szasz's (1961) claims about mental illness being a myth (or a theoretical fiction).

To what extent did the articulation of construct validity in clinical psychology influence the conceptualization of *diagnostic validity* in psychiatry? It is worth noting that Samuel Guze's early research included a study of the validity of *The Taylor Anxiety Scale* (Matarazzo et al. 1955). In that article Guze and his

colleagues claimed to be evaluating construct validity (as described in the 1954 technical report), defining validity as confirming theory-based predictions. This mingles Robins' natural history of disease approach, the predictive validity notion that preceded the work of Cronbach and Meehl, and construct validation.

Another factor influencing the establishment of a psychiatric research program on *diagnostic validity* was the emphasis in the 1970s placed on the evaluation of reliability as it was assessed statistically by psychologists (Ash 1949; Kendler et al. 2010). The Washington University group's operationalization of diagnostic constructs (called the Feighner criteria) and Columbia University psychiatrist Robert Spitzer's advocacy of measuring reliability using Cohen's kappa culminated in the publication of the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III) in 1980 (Feighner et al. 1972; Spitzer et al. 1967).[2]

After the DSM-III was published its proponents claimed that reliability had been achieved. Once the psychologists' more scientific approach to reliability was implemented in psychiatry, the reliability–validity distinction came for free, and with it came the notion that securing validity is the next task.

> In principle, the problem of reliability has been solved. . . . However, reliability does not guarantee validity. While reliability is a necessary precursor to establishing the validity of psychopathologic classes, special efforts are required for validity research (Klerman 1986).

In making this claim, Klerman was relying upon the psychometric principle that reliability sets a ceiling on validity since the latter cannot be meaningfully explored unless the variable or entity under consideration can be defined in robust and reproducible terms. Despite this bridge back to psychometrics, the meaning of *construct validity* for psychologists and *diagnostic validity* for psychiatrists continued to differ. The nuts and bolts language of measuring abstract constructs (or latent variables) is not one that psychiatrists tended to use (Blashfield and Livesley 1991). Meehl's metaphysical elaborations such as "surplus meaning" and "nomological networks" were not carried over to psychiatry. Inspired by biomedicine and not physics, for psychiatrists diagnosis was about identifying disease *entities* (nosological realism in Rodrigues and Banzato's terms in Chapter 3). Yet as we will see, the notion that psychiatric classifications are constructs that may or may not represent real clinical entities has gradually been working its way into psychiatric thinking.

1.4 The Failure to Validate as Expected

Each of Robins and Guze's validators can be considered a standard of adequacy that a diagnostic construct must meet. Documenting that the construct does meet a standard is called validation. In recent years a variety of standards have been articulated.

1. A diagnosis is valid if it can be confirmed by a test that is independent of the diagnostic criteria (e.g., a biopsy validates a diagnosis of cancer).
2. A diagnostic criterion is valid if it is a sensitive indicator of a disorder.
3. A diagnostic criterion is valid if it is a specific indicator, distinguishing true cases of the disorder from other disorders.
4. A diagnostic construct is valid if it representatively samples the psychological and behavioral features of the disorder.
5. A diagnostic construct is valid if it refers to an integrated syndrome (a pattern of intercorrelated symptoms and a predictable time course) that supports a distinction between cases and non-cases (i.e., a natural clinical grouping).
6. A diagnostic construct is valid if it allows professionals to make non-trivial inferences about patients that contribute to the description, management, or treatment of the problem. Non-trivial means the inferences are not deducible from the definition of the construct.
7. A diagnostic construct is valid if it is psychometrically unidimensional.
8. A diagnostic construct is valid if it corresponds to a unique (identity-determining) etiology (preferably biological).
9. A diagnostic construct is valid if it refers to an objective dysfunction that is harmful to its bearer.
10. A diagnostic construct is valid if its internal structure corresponds to how symptoms are structured in the population.

Arguably, one of most significant developments of the past 30 years is how difficult it has been to decisively validate the constructs of the DSM using the Robins and Guze standards. As regards the mental disorders section of the *International Classification of Diseases* (ICD-10), such validation has not been attempted.

For instance, doubt has been raised about the distinction between schizophrenia and manic depression (bipolar disorder) as assessed by every Robins and Guze standard (Kendell 1991; Bentall et al. 1988). Even when these constructs are carefully defined to emphasize severe pathology, symptom overlap is extensive (description, differentiation), clinical course is highly variable (outcome), they share genetic vulnerabilities not only with each other but with other disorders as well (family studies, differentiation), and no identity-determining biological pathology has been identified (lab studies) (Jablensky 2010; Greene 2007; Craddock and Owen 2005).

What complicates this issue is that rather than being conclusively falsified, Kraepelin's original dichotomy (dementia praecox versus manic

depressive insanity) has garnered mixed support. Kraepelin's data set consisted of semi-structured case summaries written on *Zählkarten* (counting cards). Jablensky et al. (1993) obtained access to all the *Zählkarten* for the year 1908. They recoded them using the symptoms and syndromes assessed in the *Present State Examination* (PSE) with the goal of studying what groups would emerge from objective statistical analyses of the original clinical data. Using both discriminant function analysis and cluster analysis, Jablensky et al. were able to corroborate Kraepelin's dichotomy using PSE symptoms. They found that the dementia praecox category was more restrictive and homogeneous than ICD-9 schizophrenia, placing greater emphasis on alteration of personality and disorders of affect, speech, movement, and volition. On the other hand, the manic depressive category was very broad, consisting of typical cases as well as a residue of mixed and borderline cases. Using grade of membership analysis, Jablensky and Woodbury (1995) found that pure types of dementia praecox, bipolar affective disorder, and unipolar affective disorder could be detected, but 30 percent of these cases had significant symptom overlap with one of the other types. For all types, the presence of catatonia was associated with overlap.

Kendell and Jablensky (2003) claimed that when the Robins and Guze validators were first proposed, it was assumed that the delineation of clinical entities would readily follow. However, they noted, this assumption proved to be unfounded. Kendell and Jablensky worried that this troubling outcome was being minimized in part because of ambiguity in the use of the term "valid." In practice, to say that the concept of schizophrenia is valid had come to mean that its use in psychiatry is clinically justified. In their view, such a broad use of the term validity encouraged the reification of constructs in which anything that was clinically useful would be considered to be a distinct entity.

According to Kendell and Jablensky, whether something such as schizophrenia is what psychometricians call a taxon is an empirical question. It depends on whether a syndrome has natural boundaries with other disorders and with normality, and if no boundary exists whether it possesses defining characteristics (pathognomic signs) that are qualitatively distinct. Although the "taxonicity" of disorders had not yet been confirmed, Kendell and Jablensky believed that the methodology to do so was available. They proposed that the concept of validity be limited to the assessment of taxonicity, whereas the degree to which a diagnostic construct was clinically informative be termed utility.[3]

Kendell and Jablensky's article was published 33 years after Robins and Guze. Only seven years later Steven Hyman (2010) claimed that it was time to acknowledge that the DSM and ICD classifications are interfering with making progress on a more scientifically valid classification system. Illustrating *construct-oriented thinking*, Hyman noted that classifications are cognitive

structures imposed on data to achieve important goals. According to Hyman, the DSM classifications, which are the products of the attempt to increase inter-rater reliability with reference to surface characteristics (observable signs and reportable symptoms), have not resulted in the discovery of etiology and pathogenesis, leave many cases unclassified despite a proliferation of specific diagnostic categories, and have produced confusing comorbidities.

Although Hyman has not proposed immediately replacing the current system with an alternative, he does not believe that it can be made valid by incremental changes. In agreement with his colleagues at the U.S. National Institute of Mental Health (NIMH), Hyman believes that a classification derived from the Research Domain Criteria Project (RDoC) could eventually replace or complement the DSM and ICD classifications. A collaborative effort between psychiatrists and psychologists, the ambitious RDoC program aims to bridge the knowledge gap between psychiatry and the recent groundbreaking advances in neuroscience and genomics (such as the ENCODE project, the Brain Activity Map project, and the next-generation sequencing of whole genomes of psychiatric patients), and to translate them into "personalized" diagnostic formulations and targeted prevention or treatment. Ideally, this will result in a "functional psychopathology" that may lead to recasting the taxonomy of mental disorders (Jablensky and Waters 2014). If successful, psychiatric *constructs* will be more amenable to being explained with reference to genetic and physiological mechanisms (Cuthbert and Insel 2010; Insel and Cuthbert 2010; Sanislow et al. 2010).

1.5 **Extraordinary Science**

Massimiliano Aragona (2009) has written about how the problem of extensive comorbidity in the DSM can be conceptualized in Kuhnian terms. More generally, the neo-Kraepelinian paradigm established by Robins and Guze and institutionalized in the DSM has resulted in so many problems and inconsistencies that a crisis of confidence has become widespread, as indicated by the many criticisms that DSM has attracted from inside the field. The problems with the current diagnostic paradigm, some psychiatrists believe, has become so great that a significant paradigm shift may be required (Kupfer et al. 2002; Hyman 2010; Frances 2009).

According to Kuhn (1962), although a crisis of confidence does not invariably result in paradigm shifts, it is reliably associated with a transition from a period of normal science (where the paradigm serves as an integrating framework in which questions are asked and answered) to a period of *extraordinary science*. The defining features of the fragmented periods called extraordinary science include a) a lack of agreement on what are the most appropriate methodologies,

b) magnification of the problems that define the crisis into the most important problems of the discipline, c) the generation of speculative new theories, and d) a dramatic increase of interest in exploring the philosophical assumptions of the discipline.

In the course of his own career, Kraepelin came to doubt the usefulness of the disease entity model for psychiatry—a model which he himself had borrowed from Karl Kahlbaum (Kendler and Jablensky 2011). For instance, the failure to discover etiology and pathogenesis, and even to establish a firm boundary between the normal and the abnormal, was of great concern. In 1920 Kraepelin acknowledged that schizophrenia and manic depression were not separate entities as originally proposed. To some extent, Eugen Bleuler's early work on the group of schizophrenias in 1908, the establishment of Adolf Meyer's psychobiology in 1910–30, and Karl Jaspers' pluralistic model of general psychopathology in 1913 can also be seen as responses to this earlier crisis.

In fact, the neo-Kraepelinian paradigm itself was promulgated as a Kuhnian revolution that sought to replace the DSM systems that were developed using the nosological theories of Meyer and Freud (Klerman 1986). Aragona in Chapter 2, however, argues that the DSM-I and the DSM-II were both more Kraepelinian than not in many respects. The same might be said for many currently proposed alternatives. For example, a renewed commitment to Robins and Guze's emphasis on laboratory studies (for etiology and pathogenesis) is a primary justification for claiming that a paradigm shift is needed. Attempts to take psychiatry beyond the traditional medical model are probably more thoroughly revolutionary, but this too is not a new development—nor even anti-Emil Kraepelin. Trained in experimental psychology under the scientific pluralist Wilhelm Wundt, Kraepelin rejected the reductive analysis of psychiatry associated with Greisinger and Wernicke, asserting that psychiatry was inherently psychological and best kept distinct from neurology (Kendler and Jablensky 2011; Engstrom forthcoming).

The important question is whether basic science has become (or soon will be) advanced enough to make this current crisis more than an expectable oscillation in a long historical cycle. We should all hope so—especially if it leads to progress in the treatment and management of psychiatric distress.

Before bringing this introduction to a close and proceeding to our chapter summaries (the final chapter by Stoyanov and Aragona offers an integrative overview of the book), we would like to say more about extraordinary science and the exploration of philosophical assumptions. It is our view that extraordinary science *should* explicitly include a scholarly attempt to examine the history and the philosophy of the discipline. This requires not only the increased interest in historical and philosophical questions on the part of those inside the

discipline as Kuhn described, but the inclusion of philosophical thinking drawn from sources outside the discipline.

In addition, we suggest that the philosophical problems of psychiatric classification and nosology are important not only for psychiatry and clinical psychology, but for philosophy itself. According to Popper (1963), from Plato onward the genuine problems of philosophy have originated in the philosophical problems of other disciplines, particularly the sciences.

> The degeneration of philosophical schools [into pseudo-problems and meaningless babble about words] in its turn is the consequence of the mistaken belief that one can philosophize without having been compelled to philosophize *by problems which arise outside philosophy. . . . Genuine philosophical problems are always rooted in urgent problems outside philosophy, and they die if these roots decay* (p. 95).

Although helpful to psychiatry, the resulting philosophical work should be more than a handmaiden to psychiatry. Ideally it would be formulated to also make a contribution to philosophy.

In conclusion, this volume in the *International Perspectives in Philosophy & Psychiatry* book series is offered neither as a comprehensive handbook on the problems of psychiatric validation, nor as a partisan view on how this most recent crisis of confidence should be resolved. It is more along the lines of an invitation for those with different scholarly skills to participate in a debate on a significant scientific, philosophical, and sociocultural problem—how to classify those signs and symptoms that constitute what we call psychiatric distress and impairment.

1.6 Chapter Summaries

In Chapter 2, **Massimiliano Aragona** critically analyzes several received views about important historical changes in psychiatric classification, especially the DSM-III revolution and the hoped for DSM-5 and RDoC revolutions. With respect to the DSM-III's so-called neo-Kraepelinian revolution, he shows that, despite their usual association with the theories of Meyer and Freud, the DSM-I and the DSM-II were developed in conformity with Kraepelinian (or conventional European) assumptions. In fact, an examination of the disorders listed in the first two editions of the DSM reveals that they were more similar to Emil Kraepelin's system than were DSM-III and its successors. Even the DSM-III's emphasis on diagnostic reliability was not new; rather, what was new was the neopositivist attempt to establish reliability by using operational criteria that can be algorithmically applied.

Aragona argues that because each new manual is contrasted with its predecessor in order to establish its superiority, the fundamental continuity between all the manuals tends to be ignored. What has remained the same throughout

the various revisions is a Kraepelinian model of validity *par excellence*, which holds that similar cases should ideally be grouped together with respect to shared underlying biopathological processes. The difference between older and the newer approaches is whether the organization of the phenomenal/descriptive level of analysis is expected to lead to an underlying etiology or to follow from it. He closes by suggesting an alternative to the Kraepelinian model of validity that is more constructivist and less realist in its aspirations.

Adriano Rodrigues and **Claudio Banzato**, in Chapter 3, demarcate two domains of validation—the diagnostic and the nosological. In the diagnostic domain they place the various types of validity assessed in psychometrics such as content, concurrent, predictive, and construct validity. The goals of diagnostic validation are to assess how well the diagnostic criteria represent the construct of interest and the extent to which they are able to correctly sort people into cases and non-cases. In their view the methodology for assessing *diagnostic validity* is well understood and should be applied to every disorder construct.

Nosological validation, however, is a different matter. By nosological validation they mean the extent to which it is reasonable to include a diagnostic category in the classification system. The best methodology for assessing nosological validity, they note, is more subject to debate. In this domain they distinguish two strategies which they name the pragmatic conception and the realistic conception. According to the pragmatic conception, a disorder category is valid if it is clinically useful. According to the realistic conception, a disorder category is valid if it is a real kind in J. S. Mill's sense of the term, i.e., instances of the kind systematically share features that are not included in the definition of the kind. The defining features of real kinds are also expected to cluster together more often than they would be expected to by chance.

They argue that both the pragmatic and realistic approaches should be applied to the validation of every disorder because they are non-redundant—one cannot be reduced to the other. Categories can have clinical utility without being real kinds and a category may be a real psychiatric kind, but its clinical usefulness does not depend upon its reality (i.e., what makes it real, such as having a specific genetic etiology, provides no guidance on how to manage or treat cases). In their view, clinical utility is an important consideration in both the pragmatic conception and the realist conception of validation and is therefore the most important validator. With some caveats as to whether the abstract psychological constructs of psychiatry can be squeezed into the notion of a real kind without loss of information, they also acknowledge that a system containing only real kinds may prove to be the most useful in the long run.

For those of us who have been working in the philosophy of psychiatry area over the past decade, the National Mental Institute of Health's Research Domain

Criteria (RDoC) could be considered to be an implementation of **Dominic Murphy**'s (2006) vision for a scientific psychiatry. Therefore, Murphy's evaluation of the RDoC approach to validation in Chapter 4 is a significant statement. Murphy argues that validation in psychiatry should involve discovering how disorders are produced by the causal structure of the world, and that these causal structures (preferably mechanistic in nature) can be interpreted as being real. This, he says, is a kind of Big-V validity, or the view that to validate a disorder is to show that it is real.

He contrasts his views with those of pragmatists such as Kenneth Schaffner. Pragmatists often say that valid classifications are derived from utility considerations, the articulation of goals and purposes, and background assumptions rather than the discovery of what is objectively there independent of human interests. Murphy argues that utility, purposes, and conceptual interpretations are all important in the development of psychiatric constructs as the pragmatists claim, but the resulting constructs are not necessarily incompatible with a scientific realism that seeks to discover what is really there.

However, in contrast to another important feature of the RDoC vision, Murphy articulates why the legitimacy of scientific realism about causal structures does not confer a similar legitimacy upon scientific realism about psychopathology. The concept of dysfunction/pathology, he argues, depends on normative assumptions. Dysfunctions are unwanted conditions similar to how weeds are unwanted plants. Why they are unwanted depends on contingent human interests. In fact, it is impossible to empirically (Big-V) validate a claim that a particular condition is really a dysfunction. This problem, he argues, is not specific to psychiatry, but also holds for other types of pathology, including cancer.

In Chapter 5 **Nigel Sabbarton-Leary, Lisa Bortolotti**, and **Matthew Broome** explore validity with respect to the concept of a natural kind. According to them, a natural kind is a part of *the furniture of reality* that reflects divisions in the world that can be considered to exist independently of human classification practices. Examples of natural kinds include electrons, carbon, and physical diseases such as syphilis. Because many of the features we want from a valid disorder construct are possessed by natural kinds, including uniformity among cases with respect to causal origin (etiology), development (time course), and response to intervention (treatment strategies), one way to validate a mental disorder construct is to show that it refers to a natural kind. It is thought by some (especially Szaszians) that only natural kinds are appropriate objects for medical attention.

Some arguments against the existence of natural kinds in psychiatry refer to lack of known etiology for most disorders. Others arguments point to different possibilities for lumping and splitting disorders based on extra-empirical

considerations. Sabbarton-Leary, Bortolotti, and Broome, however, argue that clear examples of natural kinds in the rest of medicine do not require that there be a confirmed (and specific) etiology or even a firmly decided lumping and splitting of cases, nor should there be for natural kinds in psychiatry. Without taking a stance on best ontological theory of natural functions, they argue that valid mental disorders represent objective biological dysfunctions (or failures of natural functions). On any construal, objective biological dysfunctions are natural kinds. They are proper objects of scientific investigation and good targets for pharmacological treatment.

The problem is what to do about cases that refer not to objective biological dysfunctions, but to harmful (yet natural) alterations in functioning such as persistent bereavement or maladaptive and sub-optimal functioning that represents a violation of norms (such as conduct disorder). These too, seem like allowable targets for psychiatric attention, but often of a non-pharmacological nature. Given that these alterations are parasitic on natural functions, Sabbarton-Leary, Bortolotti, and Broome claim that they can be classified as para-natural kinds. In the philosophy of science, a para-natural kind refers to something like an electron hole—the lack of an electron in a shell where the laws of physics would allow one to be. The electron hole is not an entity in the world, but it is a mind-independent regularity about which reliable inferences can be made (parasitic upon the nature of electrons). Another example of a para-natural kind is cold, which, according to the kinetic theory of thermodynamics, is the absence of heat. Likewise, the absence of normal function represented by bereavement can also be considered a para-natural kind.

In summary, they claim that two categories of mental kind should be recognized in psychiatry. *Mental disorders* refer to sub-optimal functioning that represents objective biological dysfunctions. *Mental harms* refer to cognitive-emotional states that are harmful to their bearers, but that lack a causally potent biological etiology. These harms too can be subject to a validation process, but for para-natural kinds the boundaries between suboptimal and optimal may be fuzzy. When this is the case, additional conceptual analysis is needed to decide if and when a particular alteration should be subject to clinical attention.

Jared Keeley, in Chapter 6, explores what he calls the *ontological loop* that exists between the practice of diagnosis/assessment and our ideas about the nature of psychopathology. This loop, argues Keeley, is one of the sources of reification in psychiatry and psychology. Keeley illustrates how our assumptions about the nature of psychopathology influence the diagnostic and assessment strategies we use, and how the results obtained from using these strategies are taken to represent a validation of those ontological assumptions.

Furthermore, some of the ontological assumptions we bring to the study of psychopathology are imparted to us during our education, rooted in the

measurement techniques available to us. Although measurement can occur in many ways, Keeley focuses on the ontological assumptions encoded in qualitative interviews, structured diagnostic interviews, and self-report psychological tests. To take one example, those who study depression using psychological tests weight every item equally and treat them as additive, making it likely that in using the test they will validate the theory that depression in the population varies continuously from lower to higher scores. A different measurement ontology in which not all symptoms are treated as equal and are assigned different weights when they interact with other symptoms under specific conditions would prescribe and subsequently validate a different ontology for depression. If all we need is severity information, then the additive ontology is perfectly adequate—but it may not be adequate for all purposes we may adopt.

In Chapter 7 **Ivana Marková** and **German Berrios** critically examine the assumption that progress in the validation of psychiatric symptoms can invariably be made by using neuroimaging data to localize symptoms onto underlying neural structures and pathways. According to this assumption, those symptoms that cannot be captured using the gold standard of imaging data are likely invalid.

Focusing on subjective complaints reported by the patients, Marková and Berrios refer to these kinds of symptoms as *hybrid objects*. By hybrid object they mean that the conversion of a raw experience into a report of a subjective mental symptom gains structure from multiple sources.

These sources can roughly be grouped into the categories of the biological and semantic. The biological refers to neural signals and the semantic refers to an envelope of meaning that symptoms gain from concepts, communities, comrades, cultures, and so on. The upshot or *sense* of the symptom as a whole, they say, is sometimes located in the neural signaling, but at other times is located in the semantic element. When the *sense* of the symptom is located in the semantic element, imaging will be an inadequate validator. As a consequence, psychiatric symptoms are not all equally appropriate for imaging research, and it would behoove researchers to gain a better understanding of symptom structure in order to avoid continually entering blind alleys.

Drozdstoj Stoyanov, **Stefan Borgwardt**, and **Somogy Varga** begin Chapter 8 by exploring the explanatory gap that exists between the underlying metaphysics of clinical psychology–psychopathology and the neurosciences, which together comprise the amalgam discipline of psychiatry. In their telling the tests of the clinical psychologists and structured interviews of the psychiatrists represent decontextualized excerpts drawn from patient narratives (i.e., "I feel tired most mornings"). On the level of meaning these reports are much like the hybrid objects described by Marková and Berrios in their chapter and therefore are closely allied with the hermeneutical approaches of the humanities.

As measurements they are typically conceptualized within the instrumentalist framework of scientific empiricism.

In contrast, the data of the neurosciences are not embedded in a hermeneutical space of reasons and narrative meanings. Additionally, the neurosciences are typically thought of using the assumptions of scientific realism rather than instrumentalism. The problem emphasized in this chapter is that the validation of psychological tests in particular occurs internal to the testing domain. Test scales are validated with reference to clinical interviews or other tests. It would be better to validate these reports by reference to something external to the framework such as the data of neuroscience, but it has not been possible to cross the explanatory gap using the kind of data that is conventionally collected by researchers. What is needed, they claim, is a program of translational validation.

One way to do this, they argue, is to align the information in the two domains by focusing on the simultaneous measurement of occurrent states by both self-report and imaging modalities. If some key methodological guidelines are followed, they claim that the imaging data would represent instantaneous states. The analog in the testing framework would not be an aggregate multi-item measure like those preferred by psychologists nor a general claim like "I feel tired most mornings," but a report of an occurrent state such as "I feel restless." Their hypothesis is that this kind of report might be better validated by imaging data, and if the translation is successful, the imaging data would itself gain clinical meaning.

The second section of the book is brought to a close in Chapter 9, where **Michael Loughlin** and **Andrew Miles** explore our inevitable reliance on norms in making distinctions between health and illness. They argue that we cannot validate a particular condition as truly disordered if our normative judgments about "disorder" cannot also be true or false. They refer to this position as *realism about value*. One of the reasons that progress on the validity problem has stalled, they claim, is that psychiatrists have accepted a modern philosophical framework in which "truth aptness" is classified with "observable and measurable" under the auspices of *the objective*. This leaves the contrasting concept of *the subjective* with "values," "opinion," and "preference." Identifying psychiatric disorders, they say, is irreducibly moral (value-laden), but moral reasoning is not just a matter of subjective preference. It too, can be correct or incorrect.

One influential manifestation of this modern framework, they suggest, is scientism—which they define as the philosophical doctrine that those approaches to knowledge generation that want to be considered as discovering truth must establish their scientific credentials, and that subjective values must be minimized if we want to know what is true. In their view, this framework is a philosophical mistake that needs to be rectified. In fact, psychiatrists may learn to accept this value-free view of truth and the objective intellectually, but cannot actually do so when practicing psychiatry.

They close by addressing the criticism that their view may encourage authoritarian practice, justifying the use of psychiatry to impose values on others. While they accept that such "totalitarian practice" has occurred in the history of psychiatry, their view, they maintain, does not justify such practice. They argue that tolerance is also an objective value—and a good. They agree that the practices of authoritarian or totalitarian psychiatry are wrong, but not just because we do not like them. They are objectively and truly wrong.

Bridging the more philosophical chapters in the second section with those in the third section that are progressively more concerned with the everyday work of clinicians, **James Phillips** begins Chapter 10 by observing that with the recent publication of the DSM-5, *diagnostic validity* as conceptualized in the Robins and Guze paradigm still has not been secured. He also notes, with some puzzlement, that the DSM-5 process began with the hope that validity might be secured with a paradigm shift, but ended with the marketing of an evaluation of reliability. Even more puzzling, the DSM-5 architects claimed that they could study reliability because adequate validity had been established. Of this, Phillips is not convinced.

He proceeds to describe three developments in the Robins and Guze paradigm, which he calls *strong syndromal validity, weak syndromal validity*, and the *RDoC* project. Strong syndromal validity refers to Kendell and Jablensky's emphasis on syndromes that possess natural boundaries with other syndromes with normality—a feature which is either present or not. Weak syndromal validity refers to Kenneth Kendler's emphasis on examining a plurality of possible validators, which can result in there being degrees of validity. One of the issues with pluralism is that the validators may not agree on the placement of category boundaries, and selecting which validator to weight higher is often a non-empirical decision. In RDoC the validator that is given the most weight is the discovery of underlying neural mechanisms. RDoC jettisons the description of syndromes in favor of studying symptomatic expressions of general psychological processes.

Next Phillips turns to a more philosophical evaluation of these *diagnostic validity* paradigms as expressions of the medical model. First, in all three paradigms etiology is a potentially important validator. They all assume that etiology is to be understood in a bottom-up fashion—from cause to phenomenon—but this may not be justified when one considers systems theory. In systems theory etiological factors interact in complicated ways, and their causal roles can sometimes only be understood in terms of the larger system in which they are embedded.

This complexity is most problematic for the RDoC matrix which attempts to regiment the domain into distinct symptom boxes that are then explained at multiple levels of analysis. From an integrated systems approach, however,

these boxes are defined by somewhat artificial boundaries. Second, despite the medical model's use of psychological symptoms, the medical model typically does not consider the implications of the psychological being emergent. One example is identity disturbance. As an emergent process, identity disturbance is both a symptom and a pathological process. The best treatment strategies may not involve direct interventions on underlying biological realizers. Even when medication is used, its role in the complex system cannot be understood in a bottom-up fashion.

In Chapter 11, **Kathryn Jacobs** and **Robert Krueger** argue that diagnostic constructs (and diagnostic systems) should be assessed by examining their structural validity. Structural validity refers to how well the internal structure of a construct (or system) corresponds to how symptoms are actually structured in the population. They contrast this data-driven approach with the expert-driven approach that is more commonly used in psychiatry. In the expert-driven approach, one or more clinicians group a number of cases together based on noticed similarities. They give that symptom pattern a name and then study it by *looking for* other cases that match that pattern. The problem is that validation research becomes an exercise in confirmatory hypothesis testing and is oriented to what clinicians expect to find. For instance, often a disorder such as depression is considered validated when it is shown to be associated with some external criterion such as work impairment. Two problems are that this so-called "external criterion" was likely one of the implicit considerations used in identifying cases (so validation is circular) and it tends to be associated with many conditions, not only depression (so validation is unspecific). This makes validation somewhat illusory.

Furthermore, if depression and generalized anxiety disorder are defined as separate and distinct entities and their defining symptoms are those which support diagnostic specificity, then actual and important overlap between diagnosed cases will not be noticed, or if noticed considered to be perplexing. When a diagnostic system is structurally invalid clinicians will be constantly confronted with cases that do not fit any known disorder, or that reflect a confusing mix of disorders. The problem is that once people learn common diagnostic distinctions, such as major depression versus generalized anxiety and schizophrenia versus bipolar disorder, they will see them out there in the world, even though the world is not structured in that way. With increased structural validity, what mental health professionals expect and what they actually find would be more closely aligned.

How can such a lofty goal be accomplished? Jacobs and Krueger suggest that the methods psychologists use to develop psychological tests offer a readily available technology for enhancing the structural validity of psychiatric diagnoses. In the concluding sections of the chapter they describe the attempt to develop a more

structurally valid model for diagnosing personality disorders in DSM-5 and suggest how something similar could be implemented for depression and generalized anxiety disorder.

Robert Cloninger, in Chapter 12, argues that psychiatrists' assumptions about how to validate a classification system have been at one time or another mistaken, partial, and lacking in vision. According to him, simplified assumptions about causality, arm-chair debates between advocates of categorical approaches and dimensional model monists, and losing sight of the persons who are subject to classification, characterize much of the recent validation literature.

As an alternative he offers a comprehensive theoretical and scientifically informed approach called the psychobiological model of personality. One of the advantages of this model, he claims, is that it contains the resources to systematically validate the distinction between psychiatric health and illness, and to ground that distinction in a model of personality functioning in general. To do so, Cloninger distinguished three domains: temperament, character, and the narrative self. Personality is what emerges from the interaction of these three domains in dynamic social contexts.

The four temperaments are basic to all vertebrate life forms and evolved early on. They are harm avoidance, novelty seeking, reward dependence, and persistence. Character evolved later in mammals and includes cooperativeness, self-directedness, and self-transcendence (in modern humans). Both temperament and character are moderately heritable and both interact in development, although character is more plastic and culturally embedded. The healthy personality is the result of being at a "golden mean" on the four temperaments combined with high values on the three character traits.

In his telling, the key feature of psychopathology is a deficiency in one or more character traits. Together, temperament and character influence, but do not fully determine, the narrative self. When healthy, the narrative self utilizes the creative capacity of self-transcendence to evolve in unique ways and is more closely associated with the concept of flourishing. He concludes the chapter by describing the ways in which the psychobiological model is superior to the factor analytic models that dominate contemporary psychopathology.

One of the new features of the DSM-III in 1980 was the introduction of multiaxial diagnosis. In addition to identifying a patient's psychiatric disorder, mental health professionals were asked to identify personality traits and types, other medical conditions, and psychosocial stressors and then to rate the patient's general functioning. The goal was to convert the more narrow activity of diagnosis into conceptual case formulation. One of the major changes in the DSM-5 was that the multiaxial system has been eliminated. In opposition to this change, in Chapter 13 **Juan Mezzich** and **Ihsan Salloum** argue that the

multiaxial model was itself too narrow. Valid case conceptualization can only be achieved in a widening of diagnostic scope. Their proposed widening is termed *Person-centered Integrative Diagnosis*. The conceptual foundation of this more comprehensive approach is that of person-centered medicine, a holistic model that rejects the assumption that one can understand the nature of a disorder independently of the person who has the disorder. The fragmentation of mental health care, they argue, is one consequence of the DSM goal of diagnosing disorders, not persons. What would be better is to diagnose a person's whole health.

Their development of an explicit and useable new diagnostic paradigm that is person-centered and integrative was informed by surveys of what clinicians, patients, and third-party stakeholders want from a diagnostic model. Their proposed model is divided up into three pillars. The first pillar is named Broad Informational Domains, of which there are three: health status, the experience of health, and contributors to health. Each domain is divided into "ill health" and "positive health." Included in this first pillar are psychiatric diagnostic categories and states of health, but also contributors to illness and health, and an attempt to understand what it is like for the person to be sick or well, and how they understand the condition themselves. This aspect of the model was the basis of the Latin American Guide of Psychiatric Diagnosis, Revised Version (GLADP-VR), which has been developed by the Latin American Psychiatric Association. The second pillar is named Pluralistic Descriptive Procedures. In addition to categories and dimensions, it uses narratives to depict an individual patient. The third pillar is named Partnership for Evaluation. In this pillar the person who is being evaluated is seen as a participant in the diagnosis process, and whose values and preferences help determine the clinical recommendations.

In Chapter 14, **René J. Muller** contends that the DSM-5 is an invitation to get a psychiatric diagnosis wrong. The problem, he says, is that this approach conceptualizes all mental illnesses as natural disease entities and it assumes that people with the same clinical presentation share the same underlying pathological process. He argues that a better alternative would be the diagnostic model promulgated by Adolf Meyer at the Johns Hopkins University School of Medicine during the first four decades of the twentieth century, and more recently by Paul R. McHugh, chair of the Hopkins psychiatry department from 1975 to 2001.

According to Meyer, a diagnostic assessment should focus on the person, and not attempt to fit symptoms into categories of psychopathology, though these categories can be useful when not taken too literally. Influenced by William James and John Dewey (Dewey was a personal friend), Meyer believed that a person's

psychology and biology are inseparable, and that persons are inseparable from their environments. He held that most psychiatric illnesses can be understood as psychological *reactions* to negative life-events that a person refuses to—or is unable to—work through. These reactions are defensive alterations in thought, emotion, and behavior, and the diagnostician's task is to understand their psychobiological origin and meaning.

McHugh and Slavney (1998) have partially systematized Meyer's psychobiology by identifying four "perspectives of psychiatry" from which a patient's pathology may be viewed. Each perspective offers a different conceptual model for evaluating a mental illness. Starting with the McHugh–Slavney perspectives and Meyer's psychobiology, Muller has developed a guide to classification and diagnosis that redubs the four perspectives *domains* to emphasize the existential niches inhabited by those who are mentally ill.

The first domain includes maladaptive reactions to challenge and stress, where a return to normalcy is delayed by a defensive stance. The varied manifestations of depression and anxiety fall here, along with dissociative disorders and some types of psychosis, as well as severe obsession–compulsion. These problems may be biologically abetted, but are ultimately the result of a person succumbing to what Meyer called the "bad habits" of mental life.

Illnesses of the second domain derive from psychobiological deficits of intellect and personality. The personality disorders and autism reside here. So do impediments to impulse control, and to reading and learning. It seems plausible, claims Muller, that deficits in the second domain could make a person more likely to succumb to maladaptive reactions of the first domain.

Third domain reactions are actively acquired and self-destructive habits. Addictions, psychosomatic conversion, sexual paraphilia, anorexia, bulimia, and self-injury are the major exemplars. Those with deficits in the second domain are also more susceptible to acquiring bad habits of the third domain.

Only in the fourth domain do we find the type of psychopathology that, to use Meyer's term, "impinges" on a person from the outside, as happens with diseases such as epilepsy. Some psychotic deviations from normality are undoubtedly brain diseases, though many are due to first domain reactions—a distinction to which the DSM-5 is blind.

Muller argues that in the DSM-5, the emphasis on reliability (agreement among clinicians) leaves out so much of what is contributing to a person's illness that validity (the truth about the illness) is compromised, and often sacrificed altogether. The psychobiological tack taken in the FDMI implicitly follows the conviction that the etiology and meaning of symptoms can be understood—and that this understanding can be validated—using existential and pragmatic criteria.

The book concludes in Chapter 15 with an overview by **Drozdstoj St. Stoyanov** and **Massimiliano Aragona.** Referring to the ideas of the Bulgarian philosopher Azarya Polikarov, they seek a middle ground between a radical pluralist approach to validation and a radical unificationist approach. Their middle ground is one in which diverse notions of validation can work together and, in some cases, be combined into a more parsimonious menu of approaches.

Notes

1. Borsboom et al. (2003) and Murphy (2006) critique Cronbach and Meehl for being too wed to logical positivism and not adequately realist. This is a fair reading, but if one considers concurrent work by Meehl, such a reading is harder to sustain (Maccorquodale and Meehl (1948), Meehl (1962)). The first part of Cronbach and Meehl's article was largely realist in tone, referring to the psychological attributes that account for test performance.
2. The DSM-III of 1980 was preceded in 1978 by the U.S. National Institute of Mental Health's Research Diagnostic Criteria (RDC), which was the result of a collaborative effort by Spitzer, Robins, and colleagues.
3. Similar to Kendell and Jablensky (2003), Borsboom et al. (2004) are critical of how the term validity is used. They claim that in psychological testing the concept of validity has become so broad that every important test-related issue is relevant to validity. Instead, they propose making the term more precise. According to them, validity should refer to whether the variations in the psychological attribute in question causally produce the variations in the measured outcomes.

References

Adams, H. F. (1936). Validity, reliability, and objectivity. *Psychological Monographs*, **47**, 329–50.

American Psychological Association. (1954). Technical recommendations for psychological tests and diagnostic techniques. *Psychological Bulletin*, **51**, 1–38.

Aragona, M. (2009). The role of comorbidity in the crisis of the current psychiatric classification system. *Philosophy, Psychology, Psychiatry*, **16**, 1–11.

Ash, P. (1949). The reliability of psychiatric diagnoses. *The Journal of Abnormal and Social Psychology*, **44**, 272–6.

Bentall, R. P., Jackson, H. F., and Pilgrim, D. (1988). Abandoning the concept of "schizophrenia": some implications of validity arguments for psychological research into psychotic phenomena. *The British Journal of Clinical Psychology/The British Psychological Society*, **27** (Pt 4), 303–24.

Blashfield, R. K., and Livesley, W. J. (1991). Metaphorical analysis of psychiatric classification as a psychological test. *Journal of Abnormal Psychology*, **100**, 262–70.

Borsboom, D. G., Mellenbergh, G. J. and Van Heerden, J. (2003). The theoretical status of latent variables. *Psychological Review,* **110**, 203–19.

Borsboom, D., Mellenbergh, G. J., and van Heerden, J. (2004). The Concept of Validity. *Psychological Review,* **111**(4), 1061–71.

Craddock, N., and Owen, M. J. (2005). The beginning of the end for the Kraepelinian dichotomy. *The British Journal of Psychiatry*, **186**, 364–6.

Cronbach, L. J. (1989). Lee J. Cronbach. In G. Lindzey (ed.), *A History of Psychology in Autobiography* (pp. 62–93). Stanford, CA: Stanford University Press.

Cronbach, L. J., and Meehl, P. E. (1955). Construct validity in psychological tests. *Psychological Bulletin*, **52**, 281–302.

Cuthbert, B., and Insel, T. (2010). The data of diagnosis: New approaches to psychiatric classification. *Psychiatry*, **73**, 311–14.

Engstrom, E. J. (forthcoming). The history of psychiatry as interdisciplinary history: on attitudes toward philosophy and psychology in German psychiatry 1867–1917. In K. S. Kendler and J. Parnas (eds), *Philosophical Issues in Psychiatry III: Nature and sources of historical change*. Oxford, UK: Oxford University Press.

Feighner, J. P., Robins, E., Guze, S. B., et al. (1972). Diagnostic criteria for use in psychiatric research. *Archives of General Psychiatry*, **26**, 57–63.

Frances, A. (2009). Whither DSM-V? *The British Journal of Psychiatry*, **195**, 391–2.

Goodwin, D. W., Guze, S. B., and Robins, E. (1969). Follow-up studies in obsessional neurosis. *Archives of General Psychiatry*, **20**, 182–7.

Greene, T. (2007). The Kraepelinian dichotomy: The twin pillars crumbling? *History of Psychiatry*, **18**, 361–79.

Hyman, S. E. (2010). The diagnosis of mental disorders: The problem of reification. *Annual Review of Clinical Psychology*, **6**, 155–79.

Insel, T., and Cuthbert, B. (2010). Research Domain Criteria (RDoC): Toward a new classification framework for research on mental disorders. *American Journal of Psychiatry*, **167**, 748–50.

Jablensky, A. (2010). The diagnostic concept of schizophrenia: its history, evolution, and future prospects. *Dialogues in Clinical Neuroscience*, **12**, 271–87.

Jablensky, A., Hugler, H., Von Cranach, M., et al. (1993). Kraepelin revisited: A reassessment and statistical analysis of dementia praecox and manic-depressive insanity in 1908. *Psychological Medicine*, **23**, 843–58.

Jablensky, A., and Waters, F. (2014). RDoC: a roadmap to pathogenesis? World Psychiatry, **13**, 43–4.

Jablensky, A., and Woodbury, M. A. (1995). Dementia praecox and manic-depressive insanity in 1908: A Grade of Membership analysis of the Kraepelinian dichotomy. *European Archives of Psychiatry and Clinical Neuroscience*, **245**, 202–9.

Kendell, R. E. (1991). The major functional psychoses: Are they independent entities or part of a continuum? Philosophical and conceptual issues underlying the debate. In A. Kerr and H. McClelland (eds), *Concepts of Mental Disorder: A Continuing Debate* (pp. 1–16). London England: Gaskell/Royal College of Psychiatrists.

Kendell, R. E., and Jablensky, A. (2003). Distinguishing between the validity and utility of psychiatric diagnoses. *American Journal of Psychiatry*, **160**, 4–12.

Kendler, K. S., and Jablensky, A. (2011). Kraepelin's concept of psychiatric illness. *Psychological Medicine*, **41**, 1119–26.

Kendler, K. S., Muñoz, R. A., and Murphy, G. (2010). The development of the Feighner criteria: a historical perspective. *The American Journal of Psychiatry*, **167**, 134–42.

Klerman, G. L. (1986). Historical perspectives on psychopathology. In T. Millon and G. L. Klerman (eds), *Contemporary Directions in Psychopathology: Toward the DSM-IV* (pp. 3–28). New York: Guilford Press.

Kuhn, T. S. (1962). *The Structure of Scientific Revolutions*. Chicago: University of Chicago Press.

Kupfer, D. J., First, M. B., and Regier, D. A. (2002). *A Research Agenda for DSM-V*. Washington, DC: American Psychiatric Association.

Leuba, J. H. (1899). On the validity of the Griesbach method of determining fatigue. *Psychological Review*, **6**, 573–98.

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

Markus, K. A., and Borsboom, D. (2013). *Frontiers of Test Validity Theory*. New York: Routledge.

Matarazzo, J. D., Guze, S. B., and Matarazzo, R. G. (1955). An approach to the validity of the Taylor Anxiety Scale: scores of medical and psychiatric patients. *The Journal of Abnormal and Social Psychology*, **51**, 276–80.

McHugh, P. R. and Slavney, P. R. (1998). *The Perspectives of Psychiatry*. Baltimore: Johns Hopkins University Press.

Murphy, D. (2006). *Psychiatry in the Scientific Image*. Cambridge, MA: The MIT Press.

Popper, K. (1963). *Conjectures and Refutations: The Growth of Scientific Knowledge*. London: Routledge.

Purtell, J. J., Robins, E., and Cohen, M. E. (1951). Observations on clinical aspects of hysteria. *Journal of the American Medical Association*, **146**, 902–9.

Robins, E., and Guze, S. B. (1970). Establishment of diagnostic validity in psychiatric illness: Its application to schizophrenia. *American Journal of Psychiatry*, **126**, 983–6.

Robins, E., and Mensh, I. N. (1954). Prediction in the clinical method and interrelations of biochemistry, psychiatry, and psychology. *The Journal of Abnormal and Social Psychology*, **49**, 435–42.

Sanislow, C. A., Pine, D. S., Quinn, K. J., et al. (2010). Developing constructs for psychopathology research: Research domain criteria. *Journal of Abnormal Psychology*, **119**, 631–9.

Spitzer, R. L., Cohen, J., and Fleiss, J. L. (1967). Quantification of agreement in psychiatric diagnosis: a new approach. *Archives of General Psychiatry*, **17**, 83–7.

Starch, D. (1915). The measurement of efficiency in reading. *Journal of Educational Psychology*, **6**, 1–24.

Szasz, T. S. (1961). *The Myth of Mental Illness.* New York: Harper & Row.

Thurstone, L. L. (1931). Relation between reliability and validity of a test. *The reliability and validity of tests: Derivation and interpretation of fundamental formulae concerned with reliability and validity of tests and illustrative problems.* Ann Arbor, MI US: Edwards Brothers.

Woodruff, R., Goodwin, D. W., and Guze, S. (1974). *Psychiatric Diagnosis.* New York: Oxford University Press.

Part 2

Matters more philosophical

Chapter 2

Rethinking received views on the history of psychiatric nosology: Minor shifts, major continuities

Massimiliano Aragona

2.1 Introduction

This chapter examines the epistemological history of the American Psychiatric Association's *Diagnostic and Statistical Manual* (DSM), which was developed to classify mental disorders. The chapter will be organized around main epistemological questions that have come to the fore in recent nosological debates. In so doing, the historical documents will be respected and considered in their proper historical context.

The main epistemological questions posed by recent debate about psychiatric nosology in general, and the DSMs in particular, are the following:

(a) What does it mean to assert that a psychiatric classification is atheoretical? Are there theoretical stances subtending the DSMs? And what kind of theories are they?

(b) Beginning with the DSM-III, the manuals are usually considered neo-Kraepelinian. What exactly is meant by Kraepelinian, and how was the DSM-III more Kraepelinian than its predecessors?

(c) It is usually claimed that the DSM-III differed from its predecessors by being concerned with the problem of reliability. To what extent was this a new concern, and how original was the proposed solution?

(d) Why has the problem of validity evolved into the main concern of current debates in psychiatric nosology?

2.2 The Theoretical Assumptions of the DSMs

2.2.1 The DSM-I

Although the first edition of the DSM (American Psychiatric Association 1952) does not acknowledge any particular theoretical source, the psychobiological theory of the psychiatrist Adolf Meyer is largely credited as inspiring

its structure and contents (American Psychiatric Association 1994, 2000; Grob 1991; Kawa and Giordano 2012; Starks and Braslow 2005; Mayes and Horwitz 2005; Spitzer 1980; Spitzer and Williams 1987).

Meyer imported many themes from European debates in psychiatry to the U.S. His main thesis was that mental symptoms are not the direct effect of biological diseases but complex *reactions* of the total person (the psychobiological unit) facing particular life circumstances (Lidz 1966; Peters 1990). This fundamental conceptualization molded the DSM-I as is clear from the section "Disorders of psychogenic origin or without clearly defined physical cause or structural change in the brain", which included "affective *reaction*," "schizophrenic *reaction*," "psychophysiologic cardiovascular *reaction*," and so on.

Many scholars have also stressed the influence of psychoanalysis on the DSM-I (Grob 1991; Kawa and Giordano 2012; Starks and Braslow 2005; Mayes and Horwitz 2005). In many cases Meyer's approach is considered psychoanalytic in spirit, but Meyer was not a psychoanalyst and as the years went on, he increasingly distanced himself from Freud's ideas. However, many of the founders of the New York Psychoanalytic Society had been Meyer's students, so Meyer indirectly influenced psychoanalysis in the U.S. The overlap between Meyer's ideas and psychoanalysis can be seen in the concrete definition of the syndromes listed in the Section on the "Diseases of the Psychobiologic Unit." For example, the "psychoneurotic disorders" were "those disturbances in which 'anxiety' is a chief characteristic, directly felt and expressed, or automatically controlled by such defenses as depression, conversion, dissociation, displacement, phobia formation, or repetitive thoughts and acts" (American Psychiatric Association 1952: 12).

Although it is clear that both Meyer's ideas and psychoanalytic ideas were important, to say that the publication of DSM-I mirrored the growing dominance of psychodynamic and psychoanalytic psychiatry and the relative weakness of the biological tradition (Grob 1991) is historically misleading. For instance, a) the aim of the DSM-I was "to provide a classification system consistent with the concepts of modern psychiatry *and neurology*" (American Psychiatric Association 1952: 9, my emphasis); b) it does not restrict mental disorders to the Meyerian "diseases of the psychobiologic unit," but also considers in great detail the mental disorders associated with organic brain disturbance. Accordingly, attention is explicitly called to the fact that "the Section on Diseases of the Psychobiologic Unit *is only one* section of the Standard Nomenclature of Diseases and Operations" (American Psychiatric Association 1952: 11, my emphasis); c) the mental disorders associated with organic brain disturbance *are not* psychodynamic reactions but "disturbance of mental function resulting from, or precipitated by, a primary impairment of the function of the brain" (American Psychiatric Association 1952: 9).

To sum up: the DSM-I was a descriptive classification of mental syndromes in which the most advanced available data on their etiology were included. Etiologies were either neurological or psychobiological depending on the knowledge of the time. Finally, main etiological concepts, such as *reaction types*, and psychodynamic ideas such as defense mechanisms, were utilized only in the domain of the psychotic, neurotic, and personality disorders.

2.2.2 The DSM-II

The DSM-II is often presented as being *in continuity* with the first edition by also reflecting the psychodynamic tradition (Kawa and Giordano 2012; Kubota and Matsuishi 2003; Mayes and Horwitz 2005). It is also claimed that the psychodynamic orientation was the reason why the DSM-II made little effort to provide elaborate classification schemes (Mayes and Horwitz 2005). However, the historical evidence shows that although the concept of neurosis was important in the DSM-II, the definitions of the specific neuroses (anxiety neurosis, hysterical neurosis, and so on) were basically *descriptive*; in fact, "psychodynamic" traces only persisted in the general definition of neurosis, where anxiety was considered "the chief characteristic." In addition to the *description* of how anxiety was perceived by the patient, it was also stated that "[i]t may be felt and expressed directly, or it may be controlled unconsciously and automatically by conversion, displacement and various other psychological mechanisms" (American Psychiatric Association 1968: 39). It is worth noting that the concept of neurosis preexisted the birth of psychoanalysis and in its long history underwent several semantic shifts, the psychodynamic sense being only one of its meanings. Moreover, for non-organic mental disorders the DSM-II had "used diagnostic terms that by and large did not imply a particular theoretical framework" (Spitzer 1980: 1–2), the principal basis for the DSM-II being the WHO 8th Edition of the ICD, to which it was closely aligned (Kawa and Giordano 2012; Spitzer 1980). The use of ICD-8 was based on the acknowledgment that "the people of all nations live in one world" (Gruenberg 1968: vii), and the ICD-8 glossary definitions were not psychodynamically oriented but the direct expression of European psychopathological research, a tradition that with few exceptions was very critical of psychoanalytic views.

2.2.3 The DSM-III

There is a large agreement that the DSM-III (American Psychiatric Association 1980) represented something really new: e.g., it has been considered "revolutionary" (Kawa and Giordano 2012; Mayes and Horwitz 2005), a "turning point" (Kawa and Giordano 2012), a "major shift in diagnostic style" (Compton and Guze 1995), a "paradigm shift" (Kawa and Giordano 2012; First 2010, 2012a;

Pincus 2012), a "paradigm-setting document" (Kendler 2012), "the construction of the first scientific paradigm in psychiatric nosology" (Aragona 2006).

The DSM-III self-defined its enterprise as "atheoretical," a contradiction in terms if, as early critics stressed, "it is not possible to be conceptually atheoretical unless one is mute" (Michels 1984: 550). However, Spitzer did not intend the DSM-III to be radically atheoretical; he simply supported the pragmatic view that the inclusion of speculative etiological theories had to be avoided because including them served as an obstacle to the manual's being used by clinicians of different theoretical orientations (Spitzer 1980). Accordingly, the DSM-III atheoretical claim should not "be intended as absence of theories but as the stance of suspending the judgment about the possible etiology of the phenomenally-defined disorders" (Aragona 2006: 48). For those disorders whose etiology or pathophysiological process were well established, this information was included in the definition of the disorder itself (Spitzer 1980).

The return of a descriptive approach against speculative psychodynamic etiologies is often considered to be a significant change introduced by the DSM-III (Compton and Guze 1995; Mayes and Horwitz 2005; First 2012a). However, the adoption of a European descriptive stance in the DSM-II was what led to the elimination of Meyer's reactions from the manual. Indeed, it is not description *per se* but other interconnected theoretical implications subtending the DSM-III that made it different from its predecessors. Two theoretical assumptions were particularly relevant. The first one was *a neopositivist theory of classification* reflected in the following DSM-III features (Aragona 2013): a) a distinction between scientific and non-scientific diagnoses; b) the exclusion of the latter as nonsensical (i.e., those relying on the last few untestable psychodynamic residuals still present in the DSM-II general concept of neurosis); c) faith in the existence of a purely observable basis for diagnosis; and d) the introduction of the operational diagnostic criteria as rules of correspondence linking the observational level of symptoms to the abstract level of mental disorders. The second main implicit theoretical stance was *a neo-Kraepelinian, biomedical assumption*, conceiving the essence of psychiatry as that of being a medical discipline.

2.2.4 DSM-III-R, DSM-IV, and DSM-IV-TR

The next three editions of the DSM were considered to be slight revisions of the DSM-III, i.e., as part of the same dominant paradigm, a progressive refinement of its concepts and achievements. The main changes in the DSM-III-R (American Psychiatric Association 1987) were the extensive use of polythetic criteria (while the DSM-III also used monothetic criteria) and the deletion of the term "neurosis" (which was retained in brackets in the DSM-III). The major change in the DSM-IV (and DSM-IV-TR) was a shift in emphasis regarding the revision: the DSM-III was a mix of conventionalism (the disorders and criteria

were agreed upon by experts, who voted to resolve disagreements) and empiricism (the famous "field trials" to test the reliability of the proposed diagnoses). The DSM-IV and subsequent editions placed more emphasis on empirical evidence, reflecting the rise of the "evidence-based medicine" (Fischer 2012).

2.2.5 DSM-5

The early hope for the DSM-5 was that it would introduce a "paradigm shift" (Kupfer et al. 2002). However, even those who believed that the DSM system faces a paradigmatic crisis that requires a revolutionary solution, stressed that the proposed revolutionary models (e.g., dimensional diagnosis, spectra, etiologically based diagnosis) were not yet ready to be fully implemented (Aragona 2006). In fact, the most revolutionary DSM-5 proposals were rejected. Hence, the DSM-5 retains the syndromal approach of the DSM-III, with the exception of: a) a limited dimensionalization: e.g., the general suggestion that the organization of chapters reflects the underlying dimensions of internalizing and externalizing; the inclusion of cross-cutting measures of symptom severity; and the provisional dimensional profile of pathological personality traits in Section III: Emerging measures and models; b) the introduction of the Autistic spectrum disorder; and c) the claim that the DSM-5 organization is consistent with the initial overall structure of the NIMH Research Domain Criteria (RDoC) project (First 2012b). In sum, although the DSM-5 self-defines itself as "a bridge to new diagnostic approaches without disrupting current clinical practice or research" (American Psychiatric Association 2013: 13), the implementation of alternative diagnostic procedures is marginal and its core remains "The individual disorder definitions that constitute the operationalized sets of diagnostic criteria" (American Psychiatric Association 2013: 10).

2.3 Emil Kraepelin, the Neo-Kraepelinians, and the DSMs

According to the received view, the DSM-III implemented the neo-Kraepelinian approach to diagnoses as it was developed at Washington University in Saint Louis. This is substantially true, but there are important caveats.

2.3.1 Contents Comparison

Table 2.1 compares the diagnoses listed in the DSMs and in Kraepelin's "Lehrbuch der Psychiatrie" (Kraepelin 1907). As shown, the DSM I and II more strictly parallel the Kraepelinian textbook, particularly in the large space dedicated to many distinct organic syndromes, while the DSM-III and DSM-5 combine them in general categories.

Table 2.1 Contents comparison between Kraepelin and the DSMs

Kraepelin	DSM-I	DSM-II	DSM-III	DSM-5
Infection psychoses	Disorders due to or associated with infections	Psychosis associated with intracranial infection Psychosis associated with systemic infection	Organic brain syndromes whose etiology or pathophysiological process is either noted as an additional diagnosis from outside the mental disorders section of ICD-9-CM or is unknown	Neurocognitive disorders (due to HIV infection, to Prion disease, or to another medical condition)
Exhaustion psychoses (collapse delirium, amentia, acquired neurasthenia)	Psychophysiologic nervous system reactions	Reactive confusion Neurose (Neurasthenic neurosis) Psychophysiologic disorders	Somatoform disorders Dissociative disorder Organic brain syndromes whose etiology or pathophysiological process is either noted as an additional diagnosis from outside the mental disorders section of ICD-9-CM or is unknown	Neurocognitive disorders (delirium) Somatic symptom and related disorders
Intoxication psychoses	Disorders due to or associated with intoxication	Alcoholic psychosis Psychosis with drug or poison intoxication	Organic mental disorders (substance-induced)	Neurocognitive disorders (substance intoxication delirium)
Thyroigenous psychoses	Disorders associated with disturbance of metabolism	Psychosis with endocrine disorder Psychosis with metabolic or nutritional disorder	Organic brain syndromes whose etiology or pathophysiological process is either noted as an additional diagnosis from outside the mental disorders section of ICD-9-CM or is unknown	Neurocognitive disorders (due to another medical condition)
Dementia praecox	Schizophrenic reactions	Schizophrenia	Schizophrenia	Schizophrenia

Dementia paralytica	Disorders due to or associated with infections (syphilis)	Psychosis associated with intracranial infection (general paralysis)	Organic brain syndromes whose etiology or pathophysiological process is either noted as an additional diagnosis from outside the mental disorders section of ICD-9-CM or is unknown	Neurocognitive disorders (due to another medical condition)
Organic dementias	Disorders due to or associated with trauma Disorders due to or associated with circulatory disturbance Disorders due to or associated with new growth (neoplasm) Disorders due to prenatal (constitutional) influence Disorders associated with disturbance of metabolism, growth, or nutrition	Psychosis with brain trauma Senile and pre-senile dementia Psychosis with cerebral arteriosclerosis Psychosis with other cerebrovascular disturbance Psychosis with intracranial neoplasm Psychosis with degenerative disease of the central nervous system Psychosis with childbirth	Organic mental disorders (dementias arising in the senium and presenium)	Neurocognitive disorders (major, due to Alzheimer's disease, frontotemporal lobar degeneration, etc.)
Involution psychoses	Involutional psychotic reactions	Involutional melancholia Involutional paranoid state		

(continued)

Table 2.1 (continued)

Kraepelin	DSM-I	DSM-II	DSM-III	DSM-5
Manic-depressive insanity	Affective reactions	Major affective disorders Psychotic depressive reaction	Affective disorders	Bipolar and related disorders Depressive disorders (major depressive disorder)
Paranoia	Paranoid reactions	Paranoia	Paranoid disorders	Delusional disorder
Epileptic insanity	Chronic Brain Syndrome associated with convulsive disorder	Psychosis with epilepsy		
Psychogenic neuroses	Psychoneurotic disorders Transient situational personality disorders	Neuroses	Anxiety disorders Somatoform disorders Dissociative disorders	Anxiety disorders Obsessive-compulsive-related disorders Trauma- and stressor- related disorders Dissociative disorders Somatic symptom and related disorders
Constitutional psychopathic states	Psychoneurotic disorders Personality disorders	Neuroses Personality disorders Sexual deviations	Disorders of impulse control Psychosexual disorders	Disruptive, impulse-control, and conduct disorders Paraphiliac disorders
Psychopathic personalities	Personality disorders	Personality disorders	Personality disorders	Personality disorders
Defective mental development	Mental deficiency	Mental retardation	Disorders usually first evident in infancy, childhood, or adolescence (mental retardation)	Neurodevelopmental disorders (intellectual disabilities)

Notes: a) DSM III-R, IV, and IV-TR are omitted because they are similar to DSM-III; b) disorders not yet classified by Kraepelin but included in one or more DSMs are not considered; c) due to changing organizational criteria with time, the diagnoses listed in the same row are not coextensive but only partly overlapping.

2.3.2 The Nosological Approach

Therefore, it is not the list of diagnostic categories that made DSM-III more Kraepelinian than previous editions. What, then, did make it more Kraepelinian?

First, as previously noted, the DSM-I had a Meyerian component. Meyer was critical of Kraepelin's views, in particular because Meyer's concept of "reaction" required an emphasis on the assessment of the patient as a person more than on the diagnosis of a disease. Nevertheless, Meyer's own emphasis on the importance of a general diagnosis contributed to the diffusion of Kraepelin's ideas in the United States. For example, in the preface to the first English edition (1902) of Kraepelin's textbook, the translator A. Ross Diefendorf writes that he is "particularly indebted" to Adolf Meyer for his "continued inspiration and critical assistance" (Kraepelin 1907: vi).

Second, as also noted previously, the DSM-II was based on the ICD-8, which was much indebted to the European tradition that was itself much indebted to Kraepelin.

Third, Kraepelin's ideal was to enucleate "disease entities," i.e. disease processes producing "identical symptom pictures, identical pathological anatomy, and an identical etiology [. . .] to which should be added the experience derived from the observation of the course, outcome, and treatment of the disease" (Kraepelin 1907: 117). However, Kraepelin recognized that "mental diseases thus far present but very few lesions that have positively distinctive characteristics [and that] [l]ikewise it has been impossible thus far to establish a classification upon an etiological basis" (Kraepelin 1907: 116). As a consequence, very similarly to what was implemented in the DSM-III, Kraepelin admitted that, etiopathogenic information being lacking in many cases, a systematic classification of psychiatric diseases was not achievable. His disease pictures were just "attempts to present a part of our observations in a form suitable for teaching purposes" (Kraepelin 1907: 119).

Fourth, the neo-Kraepelinian diagnostic criteria (or Feighner criteria) were based on five "phases" that were intended to validate a diagnostic category. These are clinical description (based on striking clinical features), laboratory tests, differential diagnosis, follow-up studies (prognosis), and family studies (heredity). Like for Kraepelin, it was acknowledged that there were not yet "consistent and reliable findings" concerning the laboratory tests (Feighner et al. 1972: 57) and that descriptive features were still the main defining characteristics.

Fifth, the DSM-III emerged from a "felicitous union" between the neo-Kraepelinian ideas of the Washington University school and the biometric and statistical approach of Spitzer and Endicott. According to Klerman (1984: 541), "Spitzer's appointment to the APA Task Force on Nomenclature and Statistics was the final pathway for funneling these trends into DSM-III." On the road from the Feighner's criteria to the DSM-III, laboratory tests and family studies were dropped as explicit diagnostic criteria. However, the principle Feighner innovation (the operational diagnostic criteria) and the general neo-Kraepelinian

categorical model were retained. So, when Spitzer (1980) writes that the DSM-III takes an atheoretical approach because the etiology is unknown for most of the DSM-III disorders, his position echoes Kraepelin's similar admission.

In conclusion, the received view that the first two editions of the DSM were not influenced by Kraepelin's nosography resulted from the neo-Kraepelinians' need to have a contrast against which they could argue for their supposed novel features. Indeed, there are more similarities between the DSM-I and DSM-II and Kraepelin's list of mental diseases, than between the latter and the disorders listed in the DSM-III (Table 2.1). The overlap between the early DSMs and Kraepelin can partly be explained by their proximity in time. What the DSM-III adds to the Kraepelian approach of its predecessors to make it more Kraepelinian are the following features: a) internal medicine as the aspirational medical model; b) the causal priority of brain mechanisms; c) etiology as the ideal end of the scientific process; d) emphasis on rigorous clinical description and differential diagnosis to identify mental disorders (Compton and Guze 1995). The fundamental commonality between Kraepelin, the neo-Kraepelinians, and the DSM-III was the assumption that by improving the diagnostic procedures, real entities of nature could be enucleated in a way that would support the discovery of the underlying etiopathologies.

Although the proposed DSM-5 revolution rejected the assumption that reliable disorders would be causally homogeneous disorders, as published the DSM-5 still exemplifies the belief that the diagnostic criteria sets "are intended to summarize characteristic syndromes of signs and symptoms that point to an underlying disorder with a characteristic developmental history, biological and environmental risk factors, neuropsychological and physiological correlates, and typical clinical course" (American Psychiatric Association 2013: 19).

2.4 **Reliability: Problems and Solutions**

DSM history converges on the claim that the innovations of the DSM-III were driven by the need to restore the credibility of the psychiatric profession which came under severe challenge in the 1970s. For example, it was reported by several studies that clinicians holding different psychiatric theories had very low agreement when diagnosing patients. It was also shown that psychiatrists in the U.S. used different criteria to diagnose schizophrenia than those in the UK. Such poor "inter-rater reliability" made it impossible to compare studies conducted across settings (Kendell et al. 1971). In other words, the reliability of psychiatric diagnosis, the minimal basis for any scientific activity (namely, the use of technical words to mean the same things/phenomena), was seriously challenged (Aragona 2009a).

This concern for lack of consensus and unreliability was not new. For example, according to Raines (1952), the 1933 Standard Classified Nomenclature of

Disease was developed to eliminate the chaos originating from the fact that any large teaching center employed its own system of classification. By 1948 three nomenclatures (Standard, Armed Forces, and Veterans Administration) were in general use, and none of them was in line with the International Statistical Classification. So, according to Raines, a major goal of the DSM-I was to establish some uniformity in the nomenclature of diseases in the United States. A similar but more international perspective was adopted by the DSM-II, whose major aim was the integration with the WHO ICD-8 which was claimed to be "indispensable for international communication and data collection" (Kramer 1968: XII).

In summary, low reliability was not a newly recognized problem in the DSM-III. The difference was that by the time of the DSM-III low reliability was perceived not as just a technical problem but as discrediting the scientific status of psychiatry in general. This motivated the search for *new* solutions to a problem that previous DSMs had failed to solve. The Washington University group introduced operational diagnostic criteria in order to ensure the comparability of data gathered in different centers and to promote communication between investigators (Feighner et al. 1972). The DSM-III architects believed that the inclusion of such criteria for every DSM-III category would ensure the scientific credibility of psychiatric nosology.

It is noteworthy that the ICD-8 (and the derived DSM-II) had already claimed to use operational definitions to improve diagnostic reliability (Kramer 1968: XIV–XV), but such definitions were glossary definitions and not, as in the DSM-III and following editions, behaviorally defined diagnostic criteria, each with a clear, explicit definition of its satisfaction criteria. Of the three sources of unreliability analyzed by Spitzer and colleagues (1979) (information, interpretation, and criterion variance[1]), it was the criterion variance (i.e., the same symptoms seen as part of different diagnoses) that could be enhanced by using formalized operative diagnostic criteria (see also First 2012a). The main characteristic discriminating the glossary definitions of the DSM-II from the operational diagnostic criteria of the DSM-III is that in the first case, the diagnostic act was largely based on the clinician's subjective judgment about the similarity between the definition and the individual patient, while in the latter the physician could only decide if the enlisted phenomena (the input) were present or not, the diagnostic rules being decided a priori and leading to the final diagnosis (the output) in a mechanistic way (Aragona 2009a).

2.5 Validity

2.5.1 From Reliability to Validity: A Failure

As seen in the previous section, although reliability was not a new problem, it was nonetheless highly weighted in the DSM-III. The manual's claim for scientific credibility largely rested upon the field-trials' demonstration of improved

inter-rater reliability. Such a "reliability first" stance is best illustrated by Spitzer's (1984: 547) assertion that "[a]lthough reliability does not guarantee validity, to the extent that diagnostic criteria are unreliable they cannot be valid." Accordingly, validity was perceived as important, but due to lack of information about many validators (etiology *in primis*) it remained as a future goal. There was great confidence that by using the DSM-III diagnoses, researchers would rapidly increase their knowledge of the important validators. Consequently, it was expected that the DSM-IV would replace the provisional descriptive DSM-III diagnoses with more valid ones, based on new evidence. Such a confidence is clearly expressed by Klerman (1984: 542): "The victory of the DSM-III has already been acknowledged [. . .] I invite our colleagues to acknowledge the achievements of DSM-III and to join with us in gathering data based on science to revise it—on to DSM-IV!"

As is now well known, the lack of significant achievements over 40 years in progressing from reliability to validity is responsible for the present-day crisis of confidence in the DSM-III project. This was acknowledged by the editors of the DSM-5 themselves: "Research exclusively focused on refining DSM-defined syndromes may never be successful in uncovering their underlying etiologies. For that to happen, an as yet unknown paradigm shift may need to occur" (Kupfer et al. 2002: xix).

Recent history has also shown that the field is not ready to change its paradigm. As officially published, the DSM-5 is a rather conservative document: "Although the need for reform seemed apparent, it was important to respect the state of the science as well as the challenge that overly rapid change would pose for the clinical and research communities. In that spirit, revision of the organization was approached as a conservative, evolutionary diagnostic reform" (American Psychiatric Association 2013: 10).

2.5.2 What is Meant by "validity" in the DSMs?

The lack of validity for most DSM disorders is commonly acknowledged (Phillips et al. 2012a), suggesting that "past science was not mature enough to yield fully validated diagnoses" (American Psychiatric Association 2013: 5). However, what kind of validity is at issue? As seen, the "atheoretical" DSM-III was in line with the neo-Kraepelinian view that course, familial pattern, and treatment planning are the important validators for justifying the addition of new disorders to the classification (Spitzer 1980). At the same time, due to its underlying biomedical stance, the DSM-III considered etiology as the validator "par excellence," i.e. the validator transforming provisional mental disorders into "real" biomedical diseases. Hence, etiology was the validator yet not available (and thus practically not involved in the phenomenally based definition of

DSM mental disorders), but the ultimate goal of the DSM-based validation program. It is noteworthy that, despite its initial revolutionary claims, the DSM-5 still holds a neo-Kraepelinian ideal of validity: "antecedent validators (similar genetic markers, family traits, temperament, and environmental exposure), concurrent validators (similar neural substrates, biomarkers, emotional and cognitive processing, and symptom similarity), and predictive validators (similar clinical course and treatment response)" (American Psychiatric Association 2013: 20). The only difference is that the authors of the DSM-5 no longer expect that the extant syndromes of the DSM will be validated in this way. Rather, they believe that such validators cross diagnostic boundaries and "tend to congregate more frequently within and across adjacent DSM-5 chapters groups" (American Psychiatric Association 2013: 20). Significant as this difference might be, a line of continuity clearly prevails. In fact, both the neo-Kraepelinian DSM-III and the DSM-5 are based on the same assumption about validity: i.e., the provisional phenomenal descriptions (the individual mental disorders or, as in the DSM-5, the larger grouping of adjacent disorders) will be fully validated when some specific neurobiological factors are "discovered" that confirm that the phenomenal description *really* corresponds to a neurobiological disease.

2.5.3 Recent Etiopathogenic Proposals

When the Research Agenda for DSM-V was launched, one of the most revolutionary proposals was the Five-Axis "pathophysiologically based classification system." In this model, Axis I was the genotype, and Axis II the "neurobiological phenotype" (i.e., intermediate phenotypes such as neuroimaging data, cognitive function, and emotional regulation). Phenomenal description, significantly redefined as "behavioral phenotype," was relegated to Axis III (Charney et al. 2002). As expected (Aragona 2006), and as witnessed by the DSM-5 itself (American Psychiatric Association 2013), it has been impossible to apply this proposal to present-day psychiatric nosology.

Somewhat later a less reductionist cognitive neuropsychiatric taxonomy was proposed in which mental disorders were seen as the result of breakdowns of neurocomputational mechanisms. It was also acknowledged that such an approach was not ready to provide a systematic reformulation of psychiatric classification, although it could suggest interesting directions for future psychiatric research (Sirgiovanni 2009). More recently, in the U.S. the National Institute of Mental Health's Research Domain Criteria (RDoC) project focuses on cognitive domains as the key constructs around which available evidence (from different sources, from genes to self-observation) should be trans-nosographically organized (First 2012b; Cuthbert and Insel 2013). The RDoC project, which is considered the most promising etiopathogenic approach at play in current

debates,[2] shares with the other etiopathogenic approaches a revolutionary shift. While in the neo-Kraepelinian approach validation research is expected to proceed from phenomenally defined disorders *back* to the discovery of their etiology, in the etiopathogenic approaches the direction is expected to be from "subpersonal" dysfunctions (of genes, brain processes, or cognitive mechanisms) *ahead* to the resulting phenomenal picture. As seen, the DSM-5 was not ready to include these new views, although in presenting itself as a "bridge" it explicitly opens future revisions to the RDoC project. Despite this expression of faith, a cautious position is also required regarding the NIMH's RDoC project. Indeed, as suggested by Frances,

> the obstacles are huge. The complexity of the brain has dwarfed the reach of even our most powerful research tools. Our science will advance, but probably will uncover vast new territories of our ignorance for every new beachhead of new knowledge. It may take decades of concerted effort for this project to bear clinical fruit and impact on the diagnostic system. It is an open question whether NIMH will be able to mount the necessary sustained commitment. [. . .] RDoC is indeed our most promising seed—let us hope it grows and thrives. But the prospects for its future success are unpredictable in these early days (Phillips et al. 2012b: 11).

Finally, despite their recognized revolutionary potential, the etiopathogenic approaches also show some continuity with the DSMs' neo-Kraepelinian assumptions. In particular, they all see mental pathologies as biomedical entities resulting from a dysfunction of physiological processes; hence, the difference is just in the direction of the discovery enterprise, i.e., from the syndrome to the underlying pathophysiology in the DSMs, from pathophysiology to the resulting syndromes in the etiopathogenic approaches.

2.6 Conclusions

This chapter shows that although many epistemological shifts occurred during the history of the DSMs, there was also a significant degree of continuity. Every edition introduced something new, but always remained conservative regarding other features. Accordingly, saying that one edition was revolutionary and the other conservative largely depends on the emphasis allotted to this or that element. The present writer chose to view the DSM-III as the birth of a new paradigm, which has not changed until now, even though it is currently in a Kuhnian crisis (Aragona 2006; 2009b).

The following are the major continuities and shifts discussed in this chapter.

The "Kraepelinian" stance. In essence, the Kraepelinian stance contends that mental pathologies are natural entities that can be enucleated by means of a phenomenal observation (symptoms, course, etc.) and fully validated once the neurobiological factors responsible for them are discovered (e.g., etiology,

neuronal structural abnormalities or dysfunctions, other pathophysiological alterations). Contrary to the common view that the DSM-I and DSM-II were psychodynamic tools incompatible with a Kraepelinian approach, this chapter shows that this approach subtends all the DSMs, including the DSM-5. Only the DSM-I concept of "reaction of the psychobiological unit" is not clearly included in this continuity. There are reasons to believe that this Kraepelinian assumption is now in crisis (Aragona 2009b), and the most recent neurocognitive proposals revise it only in part. In fact, they maintain the general assumption that mental disorders are neurobiological diseases but revert the research direction (no more from the phenomenal picture back to the underlying dysfunctional processes, but from the neurocognitive dysfunctions ahead to the resulting symptoms).

The focus on reliability. Contrary to the received view, the unreliable use of psychiatric diagnoses was not a new problem at the time of the DSM-III. The DSM-I and DSM-II were designed to resolve the same problem. What was new in the 1970s was the acknowledgment that the credibility of psychiatry as a profession was in danger because of the poor reliability of psychiatric diagnoses. Although many other factors pertaining to the sociocultural spirit of the time had a role in this transformation, it is widely accepted that the DSM-III's claim to have improved the reliability of psychiatric diagnoses significantly contributed to its success. Although probably limited to specific trials using rigorously DSM-trained interviewers, and thus with difficult generalization in common clinical settings, early reports of improved inter-rater reliability were largely used to promote the DSM-III worldwide. Later editions, including the DSM-5, still benefit from the credibility of the DSM-III.

The neopositivist operational diagnostic criteria. Preceded by the Feighner criteria, the introduction of operational diagnostic criteria was the major innovation of the DSM-III. Although the DSM-II had already claimed to use operational definitions, the DSM-III abandoned the classical prototypical descriptions of the psychopathological syndromes in favor of distinct behavioral criteria. The shift was from general glossary definitions that could apply or not to an individual patient, depending on the clinician's judgment, to explicit criteria that *had* to be satisfied in order to make the diagnosis (Aragona 2009a). Although there are reasons to believe that the operational criteria are the major reason for the current crisis of the DSM system (Aragona 2006; 2009b), they are still the core of the structural organization of the DSM, even in its fifth edition.

The problem of validity. According to the DSM-III, reliability was the necessary precondition in order to obtain the validity of mental disorders. In this context, the kind of validity at issue was the form called *big "V"* validity by Zachar (2012). That is, mental disorders were seen as natural entities "really"

existing in the "real" world, and the validation process was directed from the reliable phenomenally based disorders to the discovery of the underlying etiology and/or pathophysiological processes responsible for them. However, as time passed by, the promised goal of reaching *diagnostic validity* proved to be elusive, and a general crisis of confidence arose. Despite its initial revolutionary proposals, the DSM-5 shares with its predecessors the same ideal of validity, so that on this issue the crisis of confidence promises to persist. However, it is noteworthy that the realistic concept of validity is just one of the possible meanings of validity.

In fact, the way validity is conceived depends on one's underlying theory of knowledge and model of scientific development. On this issue, the logical space ranges from a theory of knowledge as correspondence of our representations to external reality (*Adaequatio rei et intellectus*) on one side, and a theory of knowledge as active construction of our concepts, that do not represent the world as it is but as we see it, on the other. In the first case scientific development proceeds through successive discoveries to an increasing approximation of the truth (having the discovery of truth as the final aim), while in the second case models and observations are more or less viable and useful, depending on the internal desiderata of a given research tradition. In this second case scientific development proceeds from less to more adequate models, but the evaluation of adequacy depends on goals and aims which change with time. Moreover, these assumptions can be declined more or less radically, ranging from absolute relativism à la Feyerabend (anything goes) to neo-Kantian moderate relativisms à la Kuhn (reality directly unknowable but having a decisive role in paradigmatic shifts, because *anomalies* emerge when the predictions of the model conflict with experimental results, implicitly attesting that reality is being detected when our interpretations meet resistance).

Transposed to psychiatric nosology, realists see mental disorders as putative natural entities, and validation as the experimental proof that they really exist in nature (that researchers have been able to successfully "carve the nature at its joints"). In this context, validation is the act of showing that our diagnosis corresponds to something *external* to the diagnostic concept, such as a laboratory marker or a neurobiological feature.

On the other side, mental disorders are more or less useful concepts for practical needs, constructed in specific places and times to meet practical needs, and in need of recalibration depending on socio-cultural circumstances and scientific priorities. In this context validation consists in showing that our diagnosis is adequate, i.e., that its performance is in line with the predictions and needs of our model. It is noteworthy that because any alternative model has its own menu of validators, "a plurality of validity questions" has to be expected.

Moreover, the comparison between different models is no more absolute (the most real description, grounded on its proved link to an external reality), but relative to the list of validators that have been considered appropriate for such comparison (what Zachar (2012) calls the *small "v"* notion of comparative validity). It deserves to be stressed that to acknowledge the constructive nature of mental disorders and the fact that their validation is relative to the set of validators considered appropriate in that context does not imply that we have to abandon science. With this approach, validation is no longer absolute but relative to the diagnostic system(s) in which such validity questions make sense.

In conclusion, this brief history of modern psychiatric nosographies shows that all systems (the DSM-5 included) share the same view of validity as a matter of correspondence to an external reality (more or less explicitly declined as correspondence to neurobiological data). However, with the DSM's neopositivist and neo-Kraepelinian approach being in a state of scientific crisis, a paradigm shift may occur in the future (Aragona 2006; 2009b). A shift in the direction of the RDoC project would be revolutionary in several respects but largely continuous with the ideal of validation as grounding mental disorders in neurobiological processes (as seen, it changes the direction of the search but not the nature of the correlation). However, other models might prescribe different validity questions and hence a different list of possible validators. For this reason, a conceptual clarification and comparison between alternative views on validation is timely and useful.

Notes

1. Information variance refers to the fact that during the interview different information can be obtained based on the question asked (different interviewers may elicit different phenomena); interpretation variance arises when the same phenomenon is interpreted differently by different interviewers (e.g., raters may differ in the significance attached to what is observed, and in extreme cases they may conceive the same phenomenon in a totally different way); criterion variance occurs when raters recognize the same phenomenon but they consider it as part of different general pictures (e.g., dysphoric mood seen as a symptom of either bipolar mixed state or borderline personality disorder).
2. Some reasons for this primacy are extra-scientific (i.e., the expected future allocation of research funds). There is no space to develop this point here.

References

American Psychiatric Association (1952). *Diagnostic and Statistical Manual. Mental disorders*. Washington, DC: American Psychiatric Association.

American Psychiatric Association (1968). *Diagnostic and Statistical Manual of Mental Disorders, Second Edition*. Washington, DC: American Psychiatric Association.

American Psychiatric Association (1980). *Diagnostic and Statistical Manual of Mental Disorders, Third Edition*. Washington, DC: American Psychiatric Association.

American Psychiatric Association (1987). *Diagnostic and Statistical Manual of Mental Disorders, Third Edition, Revised*. Washington, DC: American Psychiatric Association.

American Psychiatric Association (1994). *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition*. Washington, DC: American Psychiatric Association.

American Psychiatric Association (2000). *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision*. Washington, DC: American Psychiatric Association.

American Psychiatric Association (2013). *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition*. Washington, DC: American Psychiatric Association.

Aragona, M. (2006). *Aspettando la Rivoluzione. Oltre il DSM-V: Le nuove Idee sulla Diagnosi tra Filosofia della Scienza e Psicopatologia*. Roma: Editori Riuniti.

Aragona, M. (2009a). The concept of mental disorder and the DSM-V. *Dialogues in Philosophy, Mental and Neuro Sciences*, **2**, 1–14.

Aragona, M. (2009b). About and beyond comorbidity: does the crisis of the DSM bring on a radical rethinking of descriptive psychopathology*? Philosophy, Psychiatry & Psychology*, **16**, 29–33.

Aragona, M. (2013). Neopositivism and the DSM psychiatric classification. An epistemological history. Part 1: Theoretical comparison. *History of Psychiatry*, **24**, 166–79.

Charney, D. S., **Barlow, D. H.**, **Botteron, K.**, **et al**. (2002). Neuroscience research agenda to guide development of a pathophysiologically based classification system. In D. J. Kupfer, M. B. First, and D. A. Regier (eds), *A Research Agenda for DSM-V*. Washington, DC: American Psychiatric Association, 31–83.

Compton, W. M., and Guze, S. B. (1995). The neo-Kraepelinian revolution in psychiatric diagnosis. *European Archives of Psychiatry and Clinical Neurosciences*, **245**, 196–201.

Cuthbert, B. N., and Insel, T. (2013). Toward the future of psychiatric diagnosis: the seven pillars of RDoC. *BMC Medicine*, **11**, 126.

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

First, M. B. (2010). Paradigm shifts and the development of the diagnostic and statistical manual of mental disorders: past experiences and future aspirations. *Canadian Journal of Psychiatry*, **55**, 692–700.

First, M. B. (2012a). The development of DSM-III from a historical/conceptual perspective. In K. S. Kendler and J. Parnas (eds), *Philosophical Issues in Psychiatry II: Nosology*. Oxford: Oxford University Press, 127–40.

First, M. B. (2012b). The National Institute of Mental Health Research Domain Criteria (RDoC) project: moving towards a neuroscience-based diagnostic classification in psychiatry. In K. S. Kendler and J. Parnas (eds), *Philosophical Issues in Psychiatry II: Nosology*. Oxford: Oxford University Press, 12–18.

Fischer, B. A. (2012). A review of American psychiatry through its diagnoses: the history and development of the diagnostic and statistical manual of mental disorders. *Journal of Nervous & Mental Disease*, **200**, 1022–30.

Grob, G. N. (1991). Origins of DSM-I: a study in appearance and reality. *American Journal of Psychiatry*, **148**, 421–31.

Gruenberg, E. M. (1968). Foreword. In American Psychiatric Association (ed.), *Diagnostic and Statistical Manual of Mental Disorders, Second Edition*. Washington, DC: American Psychiatric Association, vii–x.

Kawa, S., and Giordano, J. (2012). A brief historicity of the Diagnostic and Statistical Manual of Mental Disorders: Issues and implications for the future of psychiatric canon and practice. *Philosophy, Ethics, and Humanities in Medicine*, **7**, 2.

Kendell, R. E., Cooper, J. E., Gourlay, A. J., et al. (1971). Diagnostic criteria of American and British psychiatrists. *Archives of General Psychiatry*, **25**, 123–30.

Kendler, K. S. (2012). Introduction to Chapter 7. In K. S. Kendler and J. Parnas (eds), *Philosophical Issues in Psychiatry II: Nosology*. Oxford: Oxford University Press, 125–6.

Klerman, G. L. (1984). A debate on DSM-III. The advantages of DSM-III. *American Journal of Psychiatry*, **141**, 539–42.

Kraepelin, E. (1907). *Clinical psychiatry. For students and physicians*. New York: The Macmillan Company.

Kramer, M. (1968). Introduction: the historical background of the ICD-8. In American Psychiatric Association (ed.), *Diagnostic and Statistical Manual of Mental Disorders, Second Edition*. Washington, DC: American Psychiatric Association, xi–xv.

Kubota, H., and Matsuishi, T. (2003). Major revision of the Diagnostic and Statistical Manual of Mental Disorders (DSM)—Background of the change and conceptualization of mental disorders. *Journal of Disability and Medico-Pedagogy*, **7**, 11–13.

Kupfer, D. J., First, M. B., and Regier, D. A. (2002). Introduction. In D. J. Kupfer, M. B. First, and D. A. Regier (eds), *A Research Agenda for DSM-V*. Washington, DC: American Psychiatric Association, xv–xxiii.

Lidz, T. (1966). Adolf Meyer and the Development of American Psychiatry. *American Journal of Psychiatry*, **123**, 320–32.

Mayes, R., and Horwitz, A. V. (2005). DSM-III and the revolution in the classification of mental illness. *Journal of the History of the Behavioral Sciences*, **41**, 249–67.

Michels, R. (1984). A debate on DSM-III. First rebuttal. *American Journal of Psychiatry*, **141**, 548–51.

Peters, U. H. (1990). Adolf Meyer und die Beziehungen zwischen deutscher und amerikanischer Psychiatrie. *Fortschritte der Neurologie und Psychiatrie*, **58**, 332–8.

Phillips, J., Frances, A., Cerullo, M. A., et al. (2012a). The six most essential questions in psychiatric diagnosis: a pluralogue part 2: issues of conservatism and pragmatism in psychiatric diagnosis. *Philosophy, Ethics, and Humanities in Medicine*, **7**, 8.

Phillips, J., Frances, A., Cerullo, M. A., et al. (2012b). The six most essential questions in psychiatric diagnosis: a pluralogue part 3: issues of utility and alternative approaches in psychiatric diagnosis. *Philosophy, Ethics, and Humanities in Medicine*, **7**, 9.

Pincus, H. A. (2012). DSM-IV: context, concepts and controversies. In K. S. Kendler and J. Parnas (eds), *Philosophical Issues in Psychiatry II: Nosology*. Oxford: Oxford University Press, 145–60.

Raines, G. N. (1952). Foreword. In American Psychiatric Association (ed.), *Diagnostic and statistical manual. Mental disorders*. Washington, DC, American Psychiatric Association, v–xi.

Sirgiovanni, E. (2009). The mechanistic approach to psychiatric classification. *Dialogues in Philosophy, Mental and Neuro Sciences*, **2**, 45–9.

Spitzer, R. L. (1980). Introduction. In American Psychiatric Association (ed.), *Diagnostic and statistical manual of mental disorders, Third Edition*. Washington, DC: American Psychiatric Association, 1–12.

Spitzer, R. L. (1984). A debate on DSM-III. First rebuttal. *American Journal of Psychiatry*, **141**, 546–7.

Spitzer, R. L., Forman, J., and Nee, J. (1979). DSM-III field trials: I. Initial inter-rater diagnostic reliability. *American Journal of Psychiatry*, **136**, 815–17.

Spitzer, R. L., and Williams, J. B. W. (1987). Introduction. In American Psychiatric Association (ed.), *Diagnostic and statistical manual of mental disorders, Third Edition, Revised*. Washington, DC: American Psychiatric Association, xvii–xxvii.

Starks, S. L., and Braslow, J. T. (2005). The making of contemporary American psychiatry, Part 1: patients, treatments, and therapeutic rationales before and after World War II. *History of Psychology*, **8**, 176–93.

Zachar, P. (2012). Progress and the calibration of scientific constructs: the role of comparative validity. In K. S. Kendler and J. Parnas (eds), *Philosophical Issues in Psychiatry II: Nosology*. Oxford: Oxford University Press, 21–40.

Chapter 3

Reality and utility unbound: An argument for dual-track nosologic validation

Adriano C. T. Rodrigues and
Claudio E. M. Banzato

3.1 Introduction

Despite recurring claims that psychiatric diagnosis and diagnostic categories in psychiatry should be valid (Kendell 1989; Spitzer 2001; Rounsaville et al. 2002), the very notion of validation is not only multifarious but also an object of terminological and conceptual disagreement (Nelson-Gray 1991; Blashfield and Livesley 1991; Skinner 1981; First et al. 2004; Löwe et al. 2008). Indictments of psychiatric diagnostic categories for not being valid, for example, are brought up under diverse justifications, ranging from the lack of evidence for their reality to their allegedly poor theoretical grounding (Szasz 1960; Trafimow 2010; Skinner 1981).

While different criteria and procedures for the validation of psychiatric diagnostic categories have been proposed and applied (Robins and Guze 1970; Kendler 1980; Andreasen 1995; Skinner 1981; Stoyanov 2009), discussions of competing views are scarce (Zachar and Kendler 2007).

As a response to this scenario of uncertainty and ambiguity, the aim of this chapter is twofold. First, we intend to provide some organization to the zoo of validity concepts by suggesting that all validity concepts in the field of psychiatry fit into one of two domains of validation, recognized and termed by Claire Pouncey (2003), to wit, *diagnostic* and *nosologic*.

As a second task, we will consider whether the validity concepts and processes of validation in each of these two domains are distinct or similar, and whether this makes it fruitful or futile to assess each of them for every psychiatric disorder. We will suggest that, under the umbrella of *diagnostic validity*, the notions of face, content, criterion-oriented, and construct validity are highly redundant, as each of them carries little in addition to the concept of *diagnostic validity* itself, i.e., that a set of diagnostic criteria leads to proper identification

of clinical instances of a psychiatric construct. Special attention, notwithstanding, will be given to the domain of *nosologic validity*. We will argue that the main validity concepts in this domain, namely, the *pragmatic* and *realistic* conceptions of validity, not only carry relevant and specific sorts of information, but that the presence of one does not warrant that the other is also present. Accordingly, we will advocate that they should be independently assessed for each mental disorder.

3.2 Sorting Out Validation on the Basis of the Propositions at Stake

As a first step in order to provide some organization to the various conceptions of validity and processes of validation, it is worthy to point out the often overlooked fact that they do not apply to diagnostic categories but to propositions about those categories. Accordingly, just as it makes no sense to assert that a rock is valid or invalid, it is meaningless to state that a psychiatric diagnostic category is valid or invalid. On the other hand, any meaningful proposition about a rock or a psychiatric diagnostic category—for example, on the composition of the former or on the utility of the latter—is amenable to having its validity ascertained. Indeed, when some given diagnostic category is said to be valid or invalid, what is actually meant is the validity of some specific proposition about it.

Of course, not all propositions about psychiatric diagnostic categories matter to nosologists and clinicians. The proposition "schizophrenia is poorly portrayed in the movies," for example, although an amenable target to the validation process, would hardly be considered of major nosologic concern. In order to properly assess the validity of psychiatric classificatory concepts, a critical step is to have a clear idea on what propositions about these objects are relevant in our field. Indeed, having a clear idea on what propositions matter would allow for a classification of the domains on which psychiatric diagnostic categories need to be validated.

Accordingly, although the psychiatric literature alludes to various *types of validity*, it is possible to sort all of them into the two related but distinct domains insightfully distinguished by Pouncey (2003, p. 9) as the *nosologic* and the *diagnostic*.

In the *nosologic domain*, the processes of validation refer to the proposition (or hypothesis) that a given diagnostic category is reasonable. That is, there should be a good justification for including it in the system.

As regards to what makes a diagnostic category reasonable, the notion of *nosologic validity* leaves room for different views on that question. Indeed,

what Pouncey (2003) generically calls *nosologic validity* is represented—both in literature and actual programs of validation—by two different conceptions of validity that we call the *pragmatic conception* and *realistic conception* (Spitzer 2001; Kendell and Jablensky 2003; Zachar 2010; Pies 2011). At the core of the *pragmatic conception* is the assumption that a diagnostic category should be taken as a valid kind if it is useful. At the core of the *realistic conception* is the assumption that a diagnostic category is valid only if it represents a real entity.

The second domain of interest to which the notion of validity arguably applies, the *diagnostic domain*, largely overlaps with psychometrics and is usually assessed by means of procedures related to the well-known notions of face validity, content validity, concurrent validity, predictive validity, and, especially, construct validity (Spitzer and Williams 1985; Jablensky and Kendell 2002). In *diagnostic validity* what is at stake is: a) how well the criteria for a category portray the psychiatric construct; and b) how well its diagnostic criteria lead to accurate identification of clinical instances of that construct. For example, it is expected that the diagnostic category named schizophrenia, as presented in psychiatric classificatory systems, is an adequate representation of the hypothetical construct schizophrenia—but not of other psychiatric constructs—and that its diagnostic criteria actually enable us to distinguish between schizophrenia cases and non-cases. Obviously, the notion of *diagnostic validity* embeds the assumption that the construct schizophrenia and the diagnostic criteria for schizophrenia in DSM or ICD are not the same.

3.3 Narrowing the Focus to *Nosologic Validity*

We intend to argue that although interwoven, *nosologic validity* and *diagnostic validity* are not redundant, either conceptually or in practical terms. In fact, the set of diagnostic criteria that represent a category in our classificatory systems might eventually lead to an unequivocal distinction between cases and non-cases of a hypothetical construct, while that construct in itself lacks justification as a real entity. While it could be argued that a diagnostic category as such would be a nonsensical tool—after all, it identifies clinical instances of a groundless construct—, one should note that, as a tool, it would be doing precisely what it was conceived to do. In other words, a given diagnostic category may have full *diagnostic validity* (i.e., validity in psychometric terms) while lacking altogether *nosologic validity*. Conversely, it is also possible for a diagnostic category to have *nosologic validity* with poor *diagnostic validity*, as it may prove to be, for any reason, not translatable into the clinical realm. Since they can be independent, both *nosologic validity* and *diagnostic validity* should be assessed for every diagnostic category. As regards the process of assessing *diagnostic validity*, it is arguably not a mystery because the concepts

of validity it subsumes are well known and their methodologies are reasonably well described. Whereas the notions of face validity, content validity, and criterion-oriented validity address, by different means, the suitability with which instruments represent our constructs of interest and then lead to the identification of their instances, the prevailing understanding is that the notion of construct validity (as put forward by Cronbach and Meehl 1955) encompasses all their aptitudes and is the only one capable of performing those tasks thoroughly (Cronbach and Meehl 1955; Loevinger 1957; Messick 1990). Thus, construct validity should be probably taken as the representative *par excellence* of what Pouncey (2003) termed *diagnostic validity*.

Indeed, the major disagreements on whether psychiatric diagnostic categories are valid or invalid, as well as the major uncertainties on what would make them valid, are both in the domain of *nosologic validity*. Thus, it is by scrutinizing the *realistic* and the *pragmatic* conception of validity—both of which are essentially driven toward defining whether the psychiatric constructs have a legitimate place in nosology—that the following discussions might be illuminating.

3.4 Examining the Nosologic Conceptions of Validation

Propositions concerning the utility and reality of psychiatric diagnostic categories are felt to be relevant within the nosologic context and, for that reason alone, deserve to have their validity examined. But knowing whether psychiatric diagnostic categories are useful and representative of real entities is not all that matters. In fact, when classificatory meta-theory is at stake, concerns should center on the legitimacy of the criteria and approaches chosen to validate a diagnostic category. Accordingly, because utility and reality are attributes on the basis of which psychiatric constructs are most often justified—a matter of *nosologic validation*—, their very competence to do so should be carefully assessed.

3.5 The realistic Conception of Nosologic Validation

Although the particular versions of realism are manifold, the most prevailing realistic conception of validation in psychiatry is naturalistic, being characterized by the following presuppositions:

a) valid diagnostic categories are those that represent real mental disorders;
b) a real mental disorder, in addition to being accepted as a disorder, exists in its own right, in the nature, and not only by convention or human artifice.

Accordingly, the diagnostic category "schizophrenia" is valid only if it represents an actually existing disease-entity. In contrast, an example of a non-real

(or artificial) disease-entity is *calyniophenia*, here defined as the co-occurrence of the following: a) avoidance of incorporating new technologies into one's daily routine; b) aversion to sunlight which is not justified on the basis of visual discomfort, aesthetic preferences regarding skin tone, or fear of skin cancer; and c) rooting for Barcelona soccer team.

In the scientific realm, the methodologies used to confirm the existence of "natural kinds," including psychiatric nosologic entities, are characterized by the search for evidence that these are not arbitrary collections of features. Two strategies are usually employed. The first is to demonstrate that the manifestations of the disorder have distinctive features that are not included in the entity's definition. For example, one must show that individuals with a particular disorder, when compared to non-affected individuals, exhibit different biological characteristics, social and functional adjustment, life course, response to therapeutic interventions, or other nosologically relevant characteristics that are not themselves inbuilt in the description of the disorder itself. The assumption is that if a disorder such as schizophrenia exists only by convention, there would be no reason to infer that individuals thus diagnosed would systematically differ from the rest of the population in any other aspect, except for the mental and behavior features that define it (Mill 2002).

A second strategy is to assess whether the defining characteristics of the entity in question cluster together in a non-random way. The defining characteristics should aggregate in the population according to a pattern different from what should be expected to occur by chance. Taking schizophrenia as an example, evidence of its reality could be provided by the demonstration that, delusions, hallucinations, disorganized thinking, grossly disorganized behavior, and negative symptoms co-occur in the general population more often than would be expected by chance.

Both of these strategies assume a conception of reality in which the various features of the disorder take the patterns they do as a result of causal mechanisms shared between different manifestations of the disorder.

3.6 Why a Realistic Conception of Nosologic Validation?

Given the previous sketch of a realistic conception of validity, one may ask what sort of special virtues real diagnostic categories are supposed to have and what justifies expecting that psychiatric diagnostic categories with those virtues should be considered nosologically valid.

First, there is the requirement of non-triviality for psychiatric classificatory concepts. Because psychiatry is a practical field, it is only fair to expect that classificatory concepts have implications. Among other things, it is expected that the

diagnosis of a given disorder should be helpful, in a diverse degree, to establish prognosis, guide treatment, and estimate the probability of new cases among family members of the affected individual. As artificial diagnostic concepts are not expected to relate in any distinguishable way with variables external to their defining features, they are not believed to allow the inferences that real entities allow.

Among the variables whose association with a psychiatric diagnostic category would grant their non-triviality, etiopathogenic variables enjoy a special reputation, from both scientific and clinical points of view (Kendell 1989; Andreasen 1995). This is explicitly recognized, for example, in the new definition of mental disorder put forward by the DSM-5, according to which "a mental disorder is a syndrome characterized by clinically significant disturbance in an individual's cognition, emotion regulation, or behavior that reflects a dysfunction in the psychological, biological, or developmental processes underlying mental functioning" (American Psychiatric Association 2013). While correlations with other sorts of variables have broad practical implications, knowing about the causal processes underlying a mental disorder would, hypothetically, provide the best targets for intervention.

A second reason why real diagnostic categories need to be validated is of an ethical nature. The critical argument runs like this: if psychiatric disorders are not real, then psychiatric diagnoses would be little more than instruments of social control, generators of stigma and social segregation. The claim that disorders were not real was integral to the anti-psychiatry movement (Szasz 1960).

Psychiatry's response to such claims has been the attempt to validate disorders as manifestation of underlying biological mechanisms. In fact, the search for proof that psychiatric nosologic entities are real is a key feature of some of the most influential programs of validation (Gottesman and Gould 2003; Kupfer and Regier 2011).

3.7 Objections to the Realistic Conception of Validation

Several important objections to the realistic conception of validity may be presented.

The first one concerns the fact that, even if psychiatric disorders are indeed real, their very nature can make their reality impracticable to demonstrate. This is because it may not be feasible to accurately assess the required correlations with external variables. For example, the correlations of chemical elements with external variables can be tested in reasonably controlled environments, whereas mental disorders are inescapably placed in very complex scenarios—individuals' mental, biological, and social lives. By not being separable from the

complex biological and mental lives of their bearers, the correlations with other variables are influenced by an unimaginable myriad of factors, falling short of naturalistic standards required for validating real entities.

Such an effect of peripheral factors may be particularly relevant in situations where one is dealing with a network of weak causal forces or INUS conditions (Mackie 1965; Schaffner 2002)—as is likely the case with both mental disorders themselves and the variables whose correlations with mental disorders are investigated in the quest for their reality.

It could be asked then, what is a realistic conception of validity worth if their disorders, however real they may be, cannot be demonstrated to be so? Of course, one sees validation in realistic terms and questions whether it is fair to require that mental disorders have the same degree of specificity in their correlations with external variables as do chemical elements. The adoption of less strict criteria for the reality of mental disorders, however, would require nosologists to confront the uncertainties about which correlations are important and how strong those correlations need to be to refer to real entities. Would the association found between bipolar disorder and suicide (patients with bipolar disorder commit suicide at a much higher rate than the general population), for example, be a satisfactory kind of evidence? What also are we to make of the fact that an association with suicide is not unique to bipolar disorder? For instance, patients with schizophrenia have a high rate of suicide as well.

There are also caveats to be made regarding the assumption that the validation of psychiatric diagnostic categories according to a realistic perspective would be strategically advantageous in the search for etiology and pathogenic mechanisms. While supposedly the constitutive features of diagnostic categories that represent real entities are more liable to having shared causal mechanisms, it should be noticed that the role of causality in psychiatric context is largely a practical one. As perceptively observed by Kendell (1989, p. 45), there is nothing inherently important about causal elements, except for the openings for intervention they eventually provide. At the same time, etiological and pathogenic knowledge is far from being an imperative for interventions to occur appropriately.

In addition, many effective interventions on mental disorders do not depend on prior knowledge of their causes. In fact, the opposite is the rule—namely, knowledge about pathogenesis often is gained by the discovery of successful interventions.

Although validating a psychiatric diagnostic category according to the *realistic* conception may facilitate the discovery of their underlying causal mechanisms, and although this knowledge may eventually become useful, the practical purposes of those classificatory concepts can arguably be met without that aid. In fact, it is questionable whether etiologically oriented psychiatric

classifications should be pursued with such priority and such high expectations, since, as well stated by Kenneth Schaffner (2002), they are not always the most useful for clinical purposes.

The ethical recommendation that psychiatric diagnostic categories should be valid in realistic terms must also be taken with caution. It is evident that a diagnostic category should never be accepted primarily because it serves private, political, or corporative interests. However, it is disputable that the acceptance of diagnostic categories must be based on evidence that they represent real disease-entities. Particularly in psychiatry, one must treat with skepticism an assertion that a diagnostic category so closely connected to the human condition, to the experience of self, and to intersubjectivity, should have its reality ultimately dependent on its biological nature. While *reality* and *nature* are highly valued by the anti-psychiatry movement as validating criteria, this is not because of the intrinsic reach that *reality* and *nature* have as validators (e.g., nature as a neutral and fair umpire). Instead, it is a strategy to curb personal influences and class interests in the prescription of normalcy and psychopathology.

It goes without saying that, as regards human experience, other parameters may be more meaningful and more ethically oriented than *reality* and *natural status*. The personal significance of the lived experience, for example, may be one of these criteria. This includes the desire to change, to suffer less, and to acquire the kind of flourishing life one sees in others, and to share that with others. These all are relevant subjective criteria that can play a robust and unbiased role in establishing the cartography of psychopathology.

3.8 The Pragmatic Conception of Validation: Purposes, Objections, and Defenses

According to the pragmatic conception of validity—elsewhere called information-based, instrumentalist, and utilitarian conceptions of validity (Zachar and Kendler 2010; Rodrigues and Banzato 2009; Pies 2011)—nosologically valid diagnostic categories are those that are useful. It should be noted, however, that usefulness is not unique to the pragmatic conception. Realistic conceptions of validity aim at such a goal too, by searching for correlations that are supposed to make psychiatric diagnostic categories useful tools. In the pragmatic conception of validity, however, the usefulness of classificatory concepts is not coupled with any ontological claim. Usefulness is, in itself, what confers *nosologic validity* to psychiatric diagnostic categories. Nor does it matter how a diagnostic category happens to be useful, only that it be useful.

Because medicine is an eminently practical field, an important question for a pragmatic conception of validity is not *why* psychiatric classificatory concepts

should be validated on the basis of their usefulness, but why such a conception of validity leaves ontological considerations aside. This is explained for epistemological or methodological reasons. Epistemologically, once the usefulness of a diagnostic category is demonstrated, metaphysical elaboration will neither increase it nor discredit it. Methodologically, not engaging in philosophical theorization may represent a strategic move to taking as much practical and scientific advantage as possible from extant diagnostic categories, even if one hopes that this is a provisional step on the path toward a nosology that is valid in realistic terms. In any case, by not engaging in metaphysical speculation, the intrinsic value of *utility* as a criterion of validity is emphasized, even in the face of uncertainties on the links between reality and usefulness in science.

But how reasonable is it, after all, to conceive utility as a criterion of validity aside from ontological and epistemological considerations? Is it reasonable to consider *utility* an independent criterion of validity, assuming that a diagnostic category may be useful without mirroring a real entity? We will explore this question over the next two sections.

3.9 The Legitimacy of Utility as a Criterion of Validity in *Non-realistic* Scenarios

Let us consider, first, a scenario in which it is impossible to prove that psychiatric classificatory concepts represent "real" kinds or in which such a hypothesis has been refuted. Would the failure to be a real kind undermine the usefulness of such classificatory concepts and deny them any *nosologic validity*?

Some insights in this regard can be gained from considering the cases of man-made kinds such as *capitalism, poverty, democracy, law*, and *political affiliation*, all of which are pregnant with practical consequences. Whereas lacking existence aside from social interactions, perception, and human ingenuity, as these concepts get progressively enmeshed in culture and become part of the repertoire of concepts people use to deal with the world, they often end up being indispensable for the apprehension of human reality. Strictly speaking, their utility is not necessarily narrow in scope or contingent upon correlations, be they causal, transient, or spurious. In fact, their utility may come from the conceptual networks engendered, which may have a reach similar to the theoretical import of those scientific concepts thought to stand for "real" kinds.

Similarly, it is not necessary to assume that, to be useful, psychiatric diagnostic categories must represent real mental disorders. If this is so, and psychiatric diagnostic categories can fulfill the practical role that medicine asks of them irrespective of their ontological status, utility should indeed be considered a validity criterion in itself.

3.10 The Legitimacy of Utility as a Criterion of Validity within *Realistic* Scenarios

If a concept cannot have its utility dismissed simply because it does not represent a real entity, would representing a real entity ensure its usefulness? Turning the question around in this way is important in a *realistic* context because, when it is assumed that valid mental disorders must be real entities, making utility a criterion of validity for psychiatric classificatory concepts is, at best, a provisional measure and, at worst, a deviation off the right track. To review, three related features are most often attributed to real entities in the scientific realm, although variably prioritized: the existence of an underlying causal mechanism, a set of shared properties as a result of shared causes, and a predictable pattern of correlations with external variables (non-triviality). Whichever of these features is given priority, their mutual connections are such that a non-random pattern of correlations between real entities and external variables is always expected, even when not taken as a core aspect of real entities from the beginning. Of course, not all correlations between a putatively real disease entity and external variables matter in the nosologic realm, just those that have actual or potential practical bearing on what we want to use the diagnostic category for. While being associated with preference for the green color makes a diagnostic category non-trivial in some sense (maybe for fashion designers), nosologic relevance would require the category to be correlated with variables that play a role in the field. Highly valued would be correlations with a given genetic allele, or a specific biochemical cascade.

Although considered nosologically relevant, it is an open question as to whether such correlations provide clinical utility. At present, these kinds of correlations have not been proven clinically useful—for various reasons, ranging from the lack of replicability of these findings to the lack of technologies for designing interventions that link bench to bedside. That is, whereas correlations with genetic and biochemical variables (and many others) seem relevant from a theoretical and scientific standpoint, this suggests their *potential* rather than *actual* usefulness.

In sum, realistic confirmation of psychiatric classificatory concepts does not imply usefulness (though that is generally expected to be the case), and lack of reality does not imply non-utility. There is an asymmetric relation between the two. While a pragmatic utilitarian stance of validity is ontologically agnostic, the realistic one aims at being useful, even if its utility cannot ultimately be warranted. Therefore, as regards psychiatric classificatory concepts, *reality* and *utility* cannot be reduced to one or the other. As a result, the *pragmatic conception of validity* deserves to stand on its own, free of ontological and epistemological ties, in parallel to the realistic conception of validity.

3.11 Final Remarks

Both *reality* and *utility* are highly valued validators of psychiatric diagnostic categories. As psychiatry's ultimate purpose is practical (as it is for science as a whole), utility certainly deserves a special place among validators. Any psychiatric nosology ought to be considered a failure if, at the end, it has been proven to be useless for whatever purpose it has. Psychiatric nosology, however, is still in motion, and any diagnostic category taken as promising from the realistic point of view—however of little use it may provisionally be—should be taken as *nosologically valid* to a certain extent, for all the prospects entailed.

As we have shown, the attributes *reality* and *utility* are not redundant and they are not necessarily coupled with each other. Indeed, because there is no fixed standard of relationship between reality and utility, the nosologic significance they both grant to psychiatric diagnostic categories suggests that it is possible and beneficial to run simultaneously projects of validation along realistic and pragmatic lines. As much as continuous epistemic interactions between top-down and bottom-up classificatory approaches have been argued to be advantageous for the nosologic enterprise (Kendler 2009), a similar strategy could perhaps get the most from pragmatic and realist approaches to validation, by means of their mutual enhancement.

In fact, although a pragmatic approach in validation programs prioritizes the search for correlations that are promptly useful, there is no obstacle for such a program to also be considered the initial step of a realistic program. In other words, instrumental categories can be thought of as signposts on the way to a more realistic conception. Similarly, while a realistic approach in validation programs would involve searching for more extensive networks of correlations, eventually turning diagnostic categories into privileged points of theoretical intersection, it could primarily emphasize a subset of variables that fall under a more instrumentalist conception. Arguably, this would amount to a prospective program with increased likelihood of maximizing the utility of their objects of study.

Of course, triangulation is not the only strategy by means of which psychiatry could take as much advantage as possible from the virtues of pragmatic and realistic approaches. Provided that they will not overshadow each other, both programs of validation could be left free to follow their own paths in parallel, with no pre-established degree of interaction, up to the point that one of them prevails or they are shown to be inescapably complementary. Even different classificatory systems could be held at a given time, for different purposes, if the most clinically useful way to classify mental disorders does not happen to be also the most promising from a scientific point of view. There is no way to know in advance which the best path to follow is or how this story will end.

What is put forward here, evidently, is not a new or separate conception of validity. At most, it should be seen as a conceptual framework for considering the legitimacy of competing validation criteria in psychiatric nosology. The position presented here favors the combined and context-sensitive use of pragmatic and realistic approaches. Thus, the contours of the validation programs will always depend on the developments in the field and on the newly emerging interests, which we are not in a position to anticipate.

References

American Psychiatric Association (2013). *Diagnostic and Statistical Manual of Mental Disorders—Fifth edition (DSM-5)*. Washington: American Psychiatric Publishing.

Andreasen, N. C. (1995). The validation of psychiatric diagnosis: new models and approaches. *American Journal of Psychiatry*, **152**(2), 161–2.

Blashfield, R. K., and Livesley, W. J. (1991). Metaphorical analysis of psychiatric classification as a psychological test. *Journal of Abnormal Psychology*, **100**(3), 262–70.

Cronbach, L., and Meehl, P. E. (1955). Construct validity of psychological tests. *Psychological Bulletin*, **52**(4), 281–302.

First, M. B. et al. (2004). Clinical utility as a criterion for revising psychiatric diagnoses. *The American Journal of Psychiatry*, **161**(6), 946–54.

Gottesman, I. I., and Gould, T. D. (2003). The endophenotype concept in psychiatry: etymology and strategic intentions. *American Journal of Psychiatry*, **160**(4), 636–45.

Jablensky, A., and Kendell, R. (2002). Criteria for Assessing a Classification in Psychiatry. In M. Maj et al. (eds), *Psychiatric Diagnosis and Classification*. Chichester, UK: John Wiley & Sons, Ltd, 1–24.

Kendell, R. E. (1989). Clinical validity. *Psychological Medicine*, **19**(1), 44–55.

Kendell, R., and Jablensky, A. (2003). Distinguishing between the validity and utility of psychiatric diagnoses. *American Journal of Psychiatry*, **160**(1), 4–12.

Kendler, K. S. (1980). The nosologic validity of paranoia (simple delusional disorder). A review. *Archives of General Psychiatry*, **37**(6), 699–706.

Kendler, K. S. (2009). An historical framework for psychiatric nosology. *Psychological Medicine*, **39(12)**, 1935–41.

Kupfer, D. J., and Regier, D. A. (2011). Neuroscience, clinical evidence, and the future of psychiatric classification in DSM-5. *The American Journal of Psychiatry*, **168**(7), 672–4.

Loevinger, J. (1957). Objective tests as instruments of psychological theory. *Psychological Reports*, **3**(9), 635–94.

Löwe, B. et al. (2008). Validity of current somatoform disorder diagnoses: perspectives for classification in DSM-V and ICD-11. *Psychopathology*, **41**(1), 4–9.

Mackie, J. L. (1965). Causes and Conditions. *American Philosophical Quarterly*, **2**(4), 245–64.

Messick, S. (1990). *Validity of test interpretation and use*. New Jersey: Educational Testing Service, Princeton University.

Mill, J. S. (2002). *A system of logic*. Honolulu: University Press of the Pacific.

Nelson-Gray, R. O. (1991). DSM-IV: Empirical Guidelines from Psychometrics. *Journal of Abnormal Psychology*, **100**(3), 308–15.

Pies, R. (2011). Toward a Concept of Instrumental Validity: Implications for Psychiatric Diagnosis. *Dialogues in Philosophy, Mental and Neuro Sciences*, **4**(1), 18–19.

Pouncey, C. (2003). Diagnosis, classification, and validity: Reliability and sensitivity as test cases. *WPA Section on Classification, Diagnostic Assessment and Nomenclature—Newsletter*, December 9–11.

Robins, E., and Guze, S. B. (1970). Establishment of diagnostic validity in psychiatric illness: its application to schizophrenia. *American Journal of Psychiatry*, **126**(7), 983–7.

Rodrigues, A. C. T., and Banzato, C. E. M. (2009). A logical-pragmatic perspective on validity. *Dialogues in Philosophy, Mental and Neuro Sciences*, **2**(2), 40–4.

Rounsaville, B. et al. (2002). Basic nomenclature issues for DSM-V. In D. J. Kupfer, M. First, and D. Regier (eds), *A research agenda for DSM-V*. Washington: American Psychiatric Association Press, 1–30.

Schaffner, K. F. (2002). Clinical and etiological psychiatric diagnoses: Do causes count? In J. Z. Sadler (ed.), *Descriptions and Prescriptions*. Johns Hopkins University Press, 271–90.

Skinner, H. A. (1981). Toward the integration of classification theory and methods. *Journal of Abnormal Psychology*, **90**(1), 68–87.

Spitzer, R. L. (2001). Values and Assumptions in the Development of DSM-III and DSM-III-R: a Belated Response to Sadler, Hulgus, and Agich. *Journal of Nervous and Mental Disease*, **189**(6), 351–9.

Spitzer, R. L., and Williams, J. B. W. (1985). Classification in Psychiatry. In *Kaplan & Sadock's Comprehensive Textbook of Psychiatry (4th Ed)*, 591–612.

Stoyanov, D. (2009). The Cross-validation in the Dialogue of Mental and Neuroscience. *Dialogues in Philosophy, Mental and Neuro Sciences*, **2**(1), 24–8.

Szasz, T. S. (1960). The myth of mental illness. *The American Psychologist*, **15**(7), 733–56.

Trafimow, D. (2010). The Implications of Meaning for the Validity of Diagnostic Categories. *Dialogues in Philosophy, Mental and Neuro Sciences*, **3**(1), 23–4.

Zachar, P. (2010). Defending the Validity of Pragmatism in the Classification of Emotion. *Emotion Review*, **2**(2), 113–16.

Zachar, P., and Kendler, K. (2010). Philosophical Issues in the Classification of Psychopathology. In T. Millon, R. F. Kruege, and E. Simonsen (eds), *Contemporary directions in psychopathology: scientific foundations of the DSM-V and ICD-11*. New York: The Guilford Press, 127–48.

Zachar, P., and Kendler, K. S. (2007). Psychiatric disorders: A conceptual taxonomy. *American Journal of Psychiatry*, **164**(4), 557–65.

Chapter 4

Validity, realism, and normativity

Dominic Murphy

4.1 Introduction

Intuitively, validity is obvious. Whereas reliability is a gauge of agreement across measurements, validity is supposed to be about what is really there. It looks obvious that even the most expert observers could all agree but nonetheless be wrong, whereas proper validation reassures us that we are measuring something that is really there. The judgment that the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) has exactly this shortcoming—reliability but insufficient validity—has led the National Institute of Mental Health in the U.S. to encourage the use of the Research Domain Criteria (RDoC) in grant proposals (Insel et al. 2010; NIMH 2011). The originators of the RDoC acknowledge that the system entrenched by previous versions of the DSM has increased diagnostic reliability. But they worry that it is too detached from the nature of mental illness, conceived of as disorders of brain circuits. These disorders could be studied at many levels and need not be identified with simple lesions. But future models of mental illness are expected, on this vision, to draw on psychological, neurological, and genetic mechanisms, and diagnosis will have to be based on these models in order to be properly based on the underlying facts about mental illness, rather than on clinical signs and symptoms.

Clearly, in this case, validating a diagnosis is thought of as understanding its underlying causal structure: a diagnosis is valid if it rests on a biological process that can be identified by experiment and observation using the methods of the biological and cognitive sciences.

Any approach to psychiatry that looks to science to validate its categories in this way must meet at least two conceptual challenges, which I will discuss in this chapter. First, there is a metaphysical challenge, which is that a concept of validity tied to the uncovering of neurobiological processes commits *realism*, the sin of thinking that science can tell us how the world is really put together. Many philosophers think that realism adds a wholly unnecessary and unwarranted metaphysical commitment, and that all science can really tell us about

is a set of relationships among the data. These relationships let us make predictions and exert some control over nature, but do not tell us what is really out there. I shall argue that biologically based psychiatry does not make any needless metaphysical commitments, and if it is realist, it is realist in an entirely harmless way.

The second conceptual challenge to the realist interpretation of validity I will consider is normative. The challenge is that there is an important sense in which *diagnoses* cannot be validated at all, if by "validation" we mean "shown to be a real disorder." All validation can do is show that a pattern of behavior deemed to be clinically significant depends on a physical process. Whether that pattern is really pathological—rather than immoral or harmlessly odd—is another matter. The issues here are tricky, but I think this second challenge probably cannot be met. Suppose we think that judgments of pathology are like judgments of positive charge, i.e., scientifically grounded, rather than judgments of bad taste, i.e., human responses. If so, there has to be some natural fact of the matter about whether some physical system—at whatever level of description—is dysfunctional. I will review some attempts at doing this and conclude that they fail. Predictions about physical states can be validated, but disorders cannot be.

Before I discuss the metaphysical and normative questions I just raised, I will say a little more about validity in general. Then I will look at the idea of validation as the uncovering of the structure of the world. Ken Schaffner (2012) has recently argued for a pragmatic account of validity which disputes the whole notion of some phenomenon really being there. He insists that it is sufficient to think in terms of predictability and utility. I will argue that there is nothing in his overall position to worry the most uncompromising realist. Then I will try to put the normative point in the context of recent disputes about the proper analysis of the concept of mental disorder.

4.2 Concepts of Validity

As Zachar (2012) points out, the intuitive sense of validity I introduced—telling you what's really there—does not map neatly onto any of the many concepts of validity that psychiatrists argue about. There are, as he puts it, numerous small-v senses of validity that don't always fit together, nor constitute a coherent big-V concept. Zachar advocates *validity pluralism*, arguing that psychiatry can employ many distinct small-v concepts of validity. These small-v concepts enable us to answer specific questions about our constructs and their relation to sundry tests and statistical measures. But how are these concepts all related—what do they have in common, and what makes them concepts of validity rather than something else? Zachar sees validity pluralism as providing a means "to construe validity as a

matter of degree" (p. 22). That does suggest that the many concepts used in day-to-day medical and psychometric practice bear a straightforward conceptual relation to big-V validity, however modest and uncertain their clinical application. They appear to be attempts to get at whatever the big-V concept is doing: we might never get at the real metaphysical underpinnings, but we aim at approximating them by measuring whatever is accessible. The epistemic gradient provided by more or less rigorous tests gives us varying degrees of certainty that what we are measuring is what is really there. But what we get, pretty much all the time, is not a representation of underlying reality but a representation of measurable relationships of clinical interest. That is, we get a bunch of results that, we hope, will correlate a diagnosis with some test outcome, or distinguish between populations. On the basis of the results, all manner of treatments and grants are dispensed.

The big-V concept of validity, then, may not capture what the sciences of the unsound mind aim at in ordinary practice. Mostly, those sciences care about relations among measurements. The big-V concept seems more like a way of cashing out a philosophical position, to wit, that understanding the world involves grasping its causal structure. Some philosophers, as we will see in a moment, think that the big-V position carries with it objectionable philosophical commitments. They think that embracing the big-V position commits one to a kind of scientific realism that allows inferences about what is really there that the relevant science cannot legitimate.

The NIMH now seems to have thrown its considerable weight behind a particular causal-explanatory account of big-V validity. This too is a paper about big-V Validity—the idea that we can ascertain that the causes of mental illness are genuinely out there in the structure of the world, waiting to be discovered. That doesn't mean, however, that the diagnoses we currently employ will be validated. Many of our existing diagnoses may get replaced or at least reformed

Following Zachar, although he does not quite put it this way and may cavil at it, I have suggested that the small-v concepts should not be seen as aiming at something completely different from the big-V version. Scientific concepts are part of unfolding epistemic projects, and all the different concepts of validity involve attempts to understand and manipulate relationships among parts of the world. The small-v concepts track measurable relationships, but the existence of the relationships does suggest that something is going on. Even if we don't know the underlying structure, we can still obtain clues to the way it works. I think that the small-v concepts often aim to establish correlations that can be seen as points on the way to a fuller causal story. However, I do not think that the fuller causal story should be seen as making illicit metaphysical commitments, for reasons I will now outline.

Schaffner also detects a gratuitous commitment to reductionism in much recent talk of psychiatric validation. He suggests that this commitment is

biologically ill-informed. On the latter point, he is surely right. A commitment to reductionism often amounts to simply a metaphysical preference for the very small. But sometimes it involves mixing up a metaphysical position—small things good, big things bad—with an epistemic one, which is the position that satisfactory explanation involves seeing how complex phenomena arise from interactions among other (ideally, simpler) ones.

This is analysis, rather than metaphysical reduction, and it is essential to our idea of good explanation. Reductionists often couch their theses in terms of explaining higher-level phenomena in terms of lower-ones, but that is entirely optional. The point of good explanations is that they show how things come about. For that to happen, we don't need to appeal to lower-level phenomena, although we sometimes can, and scientists often strive to. What we do need is to get a grip on the processes in the world that explain the phenomena of interest. This underlies the objections by RDoC advocates to the descriptive, syndrome-driven approach to mental illness. The DSM and ICD see mental disorders as collections of signs and symptoms. The objection to this descriptive approach is that it is outmoded. In the rest of medicine it was supplanted in the nineteenth century by the concept of diseases as resting on specific pathological processes in the organism. The strong interpretation of the medical model in psychiatry holds that mental illnesses are diseases of this type. They are not just sets of co-occurring systems, but destructive processes taking place in biological systems.

It might seem that there are different conceptions of validity that are naturally congruent with these two interpretations. For instance, one might think that the strong version demands an account of validity in terms of what is really there, whereas the minimal descriptive version would be satisfied with mere predictive utility. After all, the strong interpretation is committed to the idea of specific pathologies, which involve a genuine causal story about processes unfolding within the poor individuals who share a diagnosis. But on second thoughts, this is not correct, because either the strong approach or the minimal one can rest happily with an instrumentalist or pragmatist approach. Schaffner's (2012) view is that scientific reality is a matter of predictive utility, and one can make predictions about the behavior of brain systems or ion channels just as well as anything else observable.

4.3 **Utility and Validity**

Kendell and Jablensky (2003, p. 9) clearly distinguished utility and validity, defining the former as the provision of useful information about outcomes and/or testable hypotheses about correlates of diagnoses. Validity they saw metaphysically as the existence of categorically distinct kinds of diagnostic

entity; genuine carvings at nature's joints. Schaffner (2012) argues that such a distinction is unsupportable. He makes a bet that, based on general biological considerations, we should expect to see dimensions and not categories in human populations. But Schaffner's main arguments are philosophical, based on the pragmatist idea that we cannot separate utility from a genuine representation of nature. For Schaffner, utility is constitutive of reality. Schaffner does not embrace a strong anti-realism about psychiatry and the related fields, but he does reject a thoroughgoing realism. Although I will not attempt a general defense of scientific realism, let me critically analyze some of the arguments Schaffner offers.

Philosophical disputes over realism have often centered on the notion of the unobservable. Electrons are a paradigm example, but other posits of fundamental physics play the same role. In psychiatry the situation is different; theories and constructs employ latent variables rather than reference to unobservables, as well as, just like any science, hypotheses that go beyond observed cases to cover the infinitely many more cases that have never been—could never be—observed. Science is a massive data-compression project: rather than enumerate all the individual cases, we apply a general label to them such as "schizophrenic" (or "soluble" or "haplodiploid"). This commits the scientist to a bet, which is that the behavior of phenomena that are unobserved but fall under the label will be enough like the behavior of the observed cases to justify the application of the label (i.e., labels are inference tickets that tell us what to expect from unobserved cases).

The chief anti-realist challenge concerns the unobservable. The charge with respect to the unobservable is that our techniques of observation can carry us only so far: we can perceive lots of things, but those things that cannot be observed should not be presumed to exist. This anti-realist approach originates in reflection on the physical sciences where mentioning unobservable entities might help with understanding the mathematics that expresses the main commitments of the theory. But the unobservables themselves should not be taken to exist. The point about arguing over what is really there is clear enough in these sorts of physical cases, since what is at stake is the existence or otherwise of bits of the world.

4.4 "Observation" in Psychiatry

It is not clear how to translate this dispute into the context of psychiatry. What are the unobservable things whose existence we are contesting? The answer could be either all of psychiatry, or none of it. In the first case, one could argue that signs and symptoms are conceptual inferences. What is actually observed

are bodily movements and vocalizations. The notions of sign and symptom go beyond these observations and amount to theoretical posits.

Objecting that these posits are illicit is absurdly strong, for it would also rule out every other possible psychological state. We have only movements and noises to go on when we see someone as sad or flirtatious, or as believing one thing and desiring another. Seeing someone's behavior as clinically significant obviously requires training in a special class of concepts, but so does mastering our everyday folk psychology. In short, the mere presence of a special set of concepts does not license anti-realism, otherwise we would have to be anti-realists about everything that we attribute to human beings on the basis of their behavior, including "this person is dancing" and "this person is kicking a football." Concepts do not just describe the behavior, they interpret it.

Obviously nobody is arguing for anti-realism about human behavior in general (although I am not arguing for an uncritical realism about all our psychological concepts either). But anti-realism does have some force when we start thinking about an empirical approach to psychiatry. For, as Schaffner notes (2012, p. 176), the constructs that we typically seek to validate must be validated indirectly. So we are looking for correlates of constructs, and the constructs put an interpretation on the observed behavior.

Schaffner (2012, p. 177) adopts the view that reality is constituted by scientific utility. He calls this *conditionalized realism*, as an acknowledgment that our acceptance of any scientific claim is conditional upon 1) not just the evidence, but the acceptance of auxiliary hypotheses (about, for example, the reliability of our instruments) and 2) the absence of plausible alternative hypotheses.

I agree with Schaffner on both of these points, but I dispute what he sees as their significance. Neither point causes any trouble for the scientific realism that Schaffner regards as "too strong." Schaffner insists (2012, p. 178) that we do not have any "direct intuitive experience of the certitude of scientific hypotheses or theories." This is true, but whoever said realism requires some direct intuitive certitude, as if doing science were a matter of revelation? What a realist might be inclined to insist on (as Kitcher 2013 does) is that perception provides unmediated access to the world. Our perceptual encounters with the world require us to be in certain mental states, but we do not perceive those states. We just perceive the world.

Certainly to perceive what is there one needs to possess concepts. Most people will not recognize a googly, a sestina, or a nudibranch when they see one, because they don't have the relevant concepts. But dependence of recognition on concepts does not mean that what is perceived doesn't exist, even if it requires a background of concepts. (Of course in saying this I am disregarding global skepticism about perception, but that is everybody's problem, and not a

difference between this view and a pragmatist one.) The realist point is that our perception of the world is a matter of causal contact with it. We have to *be in* a certain psychological state—concept possession—to recognize a nudibranch or a panic attack. But we do not *perceive* mediating psychological states or entities. This departs from the "way of ideas" of early moderns like Hume and Descartes, which assumed that we perceived the world via perception of mediating mental entities. This direct causal contact with the world, however, is not some strange philosophical power of "intuition" that puts us in touch with the constitution of reality. It is ordinary perception.

Schaffner argues that the most one can hope for is that a scientific claim has relatively direct evidence in its favor. This is all theory or construct can hope for because of its inevitable latent aspects. Like Kitcher (2013) I propose that we see scientific access to theoretical constructs as akin to epistemic access to ordinary folk "constructs" like flirtatiousness or cynicism. Ordinary perception, like science, provides relatively direct access to the world assuming that we accept auxiliary hypotheses (the light is good, I'm wearing my glasses) and can discount plausible alternative hypotheses. The same considerations that support truth claims in ordinary life support truth claims in science, or at least in psychiatry. Schaffner does not actually dispute this, but he regards it as a strike against realism. In contrast, I think it is decisive evidence in favor of realism, because I reject the idea that realism requires some direct intuitive access to the world.

4.5 **Observation and Causality**

There is another issue, though, concerning the relations between truth and utility, where Schaffner and the realist may part company. Schaffner thinks that the point of a psychiatric or psychological construct is essentially practical: to gather useful information about a population through tests that are as rigorous as we can devise. Again, I agree, but would emphasize something different, which is that these aims are more likely to be fulfilled the more we know about the underlying structure of the categories involved.

The realist claim would be that schizophrenia, for example, is not just a label that gives us a convenient way of grouping people who seem worth grouping together for predictive and other purposes. We want to think that there is a genuine natural phenomenon that they all share, as there might be if we grouped organisms together on the grounds that they all share the same viral infection. The realist bet is that coming up with the construct is the first step toward further investigation. To begin with we want to find biological markers that correlate with schizophrenia. Much of biological psychiatry's commitment to big-V validity comes in here as we look for the biologically relevant stuff going on

inside people. Schaffner's point appears to be that these further questions can be asked, but that they should not be seen as leading us toward the truth about how the world really is; they will just uncover more facts about the relations between measurements and outcomes. It is correlations all the way down, and the predictive power and utility gathered in this way constitute reality.

But the schizophrenia construct, unlike electrons, is not a further type of stuff that lies behind the appearances and explains the appearances: it is constituted by visible phenomena. We are not looking for an unobservable, but asking which observable phenomena explain the symptoms. Latent constructs and unobservability are not the same, and when we push further after the sorts of markers and causal processes that the RDoC envisages, we are uncovering causal structures and generating useful predictions. Of course we may refine our categories in such a way as to decide that the original construct is of limited use, and schizophrenia may disappear in favor of one or more alternatives, but the point remains: the generation of new constructs using biological, psychological, or genetic markers is a step toward both causal knowledge and utility. The purpose of a psychiatric construct might be utility, but the investigation of it can uncover the causal structure of the world. This is just part of our normal drive to find out why things happen.

We often explain an entity's behavior in terms of the kind of object it is, as when we say that Miffy is afraid of dogs because she is a rabbit. Cooper (2007) calls this *natural history explanation*. We might prefer to think of a natural history explanation as a placeholder for a more complete explanation, but even in the absence of a causal account of why something behaves as it does, we may obtain useful information just by noting the characteristic relations it enters into. Different types of plant may need to be put in the ground at different times, or in different seasons, in order to maximize crop yield, for instance, and different patients may respond to different drugs even if the causal basis of these differences remains unknown. And although we might be ignorant about whatever it is that explains those relations, the natural inference to make is that there is something about the world that explains why the relations hold reliably. Miffy might do all sorts of things occasionally, but she is reliably and predictably afraid of dogs, and everyone will conclude there is something in Miffy's nature that makes her behave like that. Predictable and repeatable phenomena get measured and serve as the basis for further inquiry.

Zachar's idea that small-v validity is on a continuum with the big-V concepts expresses this thought: understanding reliable and predictable relations among measurements is the first step toward understanding the hidden structure that accounts for surface phenomena. Small-v validity lets us make use of descriptive and statistical reasoning and offers the hope of accurate prediction and

effective control. To know more about why Miffy is so craven we look inside her and her conspecifics to find the perceptual, endocrinological, and physiological mechanisms that relate, in her brain, the representation of a canid to a particular suite of evasive behaviors. It is also a good bet that closely related species will share such systems. And we can then go on to build a picture of the evolution of those systems by seeing how they differ in less related species. It is useful to know that x system often produces y outcome in z conditions, so that we learn about new interventions and points of manipulation. But such knowledge also allows us to dig deeper, and look for the underlying causal picture (and then the causal picture that underlies that, and so on). All this uncovering of further connections gives us both useful predictive knowledge and knowledge of causal structure.

To reiterate: my talk of underlying systems should not be taken as a commitment to reductionism. In my sense, long-term unemployment or family environment can underlie something just as much as stretches of DNA can. The point is that looking for useful, predictive knowledge is a way station on the road to figuring out how things really work. There is little reason to suppose that psychiatry has got very far along that road, but the objection to big-V validity is not a metaphysical or epistemic objection in principle to the project. Big-V validity requires no commitment to direct intuition of the world, nor is it at loggerheads with a desire for utility. It builds on utility and it uses the methods that we use to establish useful predictive knowledge.

So I think that big-V validation of causal stories about mental illness is possible. The question I turn to now is whether that also amounts to validation of diagnoses, where validation is understood as uncovering the causal structure of the world.

4.6 **Normativity**

The second topic that the big-V conception of validity leads me to is normativity. Suppose we find something that is really there, perhaps a distinctive pattern of activity in a brain circuit that correlates reliably with thought disorder and makes sense relative to our background knowledge of cognitive neuropsychology. Are we showing that a diagnosis corresponds to an objective fact about human ill-being, and hence supports the idea that psychiatric illnesses are not social constructions in some pejorative sense but rather objective pathological conditions? No, we are not. We are showing that there is some objective fact that explains why one group of humans is different in some respect to some ideal type of normal behavior, but we are not showing that the difference is pathological.

For this to amount to big-V validation—showing that something is really part of the world—biological *dysfunction* must be objectively established, not just biological markers. According to the RDoC advocates, however, the goal is to discover the underlying dysfunctions that give rise to psychiatric symptoms. I propose that we are entitled only to a weaker goal, the discovery of biological difference, not objective dysfunction. We judge certain ways of life to be pathological. And we can discover objective scientific correlates of those ways of life. This discovery of why people act in ways we judge to be pathological is what the anti-psychiatric movement bet against. It should not have made that bet. On the other hand, the discovery of objective biological difference does not mean that we have found a dysfunction. The attribution of dysfunction requires assumptions that are themselves normative.

4.7 **The Two-Stage View**

The yoking of biological and normative considerations in the validation of mental disorder (or any medical condition) is the hallmark of what I call the two-stage view. It is the most popular account of psychiatric disorder among theorists who deny that ascriptions of mental illness are entirely normative. It was introduced by Wakefield (1992), who adapted earlier ideas of Boorse (1975, 1976). Two-stage theorists hold that there are two individually necessary and jointly sufficient conditions for the attribution of a disorder. First, there is a biological dysfunction. Wakefield's innovation saw dysfunction as a failure by a bodily system to perform the naturally selected function that explained the system's replication in past generations, whereas Boorse saw dysfunction in terms of a system's failure to contribute to the overall systemic capacities of the organism.

Second, the dysfunction must result in harm to the individual concerned, as judged by prevailing social norms. "Harm" is a normative notion. So psychiatric dysfunction is assumed to be a matter for medicine to establish, just as it would establish that an esophagus has become dysfunctional. But, according to this view, whether somebody is harmed is a matter of prevailing social judgments. Establishing whether a specific state of affairs constitutes harm for a person is often going to be controversial—although consensus on many harms is easy. The issue of what counts as harm is not one science can settle, though empirical findings can provide evidence that supports the judgment of harm.

The two-stage view, then, is designed to give both science and social context their due. It aims for a middle ground between (i) a scientism that says psychiatry has no role for values at all and (ii) a constructivist claim that our judgments that a person is disordered depend entirely on their having violated some

norm. The view supposedly respects both the role of science in psychiatry and that of social norms. However, the two-stage view faces two sets of conceptual problems. First, there are the difficulties involved in justifying the intuition that science plays a role in the discovery of objective facts about dysfunction. In the rest of this chapter I will discuss what that role is.[1]

Second, we have an intuition that norms have a role to play in whether an individual is harmed by his or her dysfunction. This intuition is thought to have normative implications with respect to rights and duties to treatment. Now, unpacking the notion of harm is at least as problematic as unpacking the notions of function and dysfunction (e.g., see De Block 2008). But the relevant concept of harm involves judgments about the quality of a life. These judgments need to be sensitive to both the individual's own needs and goals, and the ideas about well-being that feature in the wider society.

4.8 **Function and Dysfunction**

There are two broad concepts of function in biology (Godfrey-Smith 1993). We can see the function of a biological system as Wakefield does, as that effect of the mechanism which contributed to the success of the ancestral population and thereby the replication of the system (Millikan 1984). I call this the selectionist view. But we can also understand a system's function as the contribution it makes to a broader biological system of which it is a part (Cummins 1975). I call this the systemic view.

The life sciences encompass many projects, and trim their accounts of function to suit them. Cummins argued that the basic explanatory use of function talk in the life sciences derives from a particular analytic strategy in which the biologically significant capacities of a whole organism are explained by breaking down the organism's biology into a number of "systems"—the circulatory system, the digestive system, the nervous system, and so on—each of which has its characteristic capacities. These capacities are in turn analyzed into the capacities of their component organs and structures. We can reiterate this systemic concept of functions through levels of physiology, explaining the workings of the circulatory system, the heart, certain kinds of tissue, certain kinds of cell, and so on. Much mechanistic research in biology exemplifies this approach.

As well as questions of survival value, we can ask questions that simply aim to find out how a system does what it does in the context of the superordinate system. There is an idea almost as old as natural selection that may help to answer these questions. It is Claude Bernard's (1927 [1865]) suggestion that major systems in the human body seek to maintain stable internal homeostatic states. Bernard argued that organisms can only explore and transform the external environment if they have sufficient internal stability. In this view the answer to

the question "What is the function of the major physiological systems?" is "To keep the internal environment stable." Homeostasis, not survival value, is what guides physiological answers to questions about causal explanations of biological systems in this tradition.

It has become clear that this cannot be quite right, since much behavior (reproductive behavior, for example) seems to disrupt homeostasis and organisms endure lengthy periods of stress in which the system is dysregulated. This has led some theorists to embrace the supplementary idea of *allostasis*, "to take account of the physiology of change and adaptation to diverse circumstances, and to the behavioural and physiological anticipation of future events" (Schulkin 2011, p. 5). Organisms achieve internal viability by adapting as circumstances change over time. Regulation involves response to, and anticipation of future, social and environmental needs. Physiology and medicine appear to be guided by a homeostatic–allostatic concept of function. They employ the systemic view, and not the selectionist view.

4.9 **Medicine, Function, and Normativity**

The idea behind the two-stage view is that science can tell us that the disorder is real not just by finding markers or causal processes, but by finding dysfunctions, thereby answering the skeptic who thinks of diagnosis as just labeling.

The two-stage view says that scientific facts play a significant role in determining whether or not a condition is a disorder, by ascertaining that something is a dysfunction. If there is a dysfunction that explains the symptoms, then we have a clear link between the biology of the organism and the behavior that attracts the diagnosis (and more broadly, the attention of one's fellows, which is what leads to the search for the underlying dysfunction).

The dysfunction criterion was initially introduced to help us determine which individuals are in fact disordered, in a way that avoids subjective, mind-dependent, or culturally relative judgments. The scientific aspect of the two-stage view thus has the job of rebutting the skeptical claim that disorders are only violations of prevailing social norms. To rebut such skepticism using the two-stage view, psychiatry (or the basic sciences it draws on) needs to be able to see processes as objectively dysfunctional, not just as causes of ways of being. The skeptic asks why we should see a pattern of brain activity as a correlate of disorder rather than a correlate of eccentricity or immorality or something else that is none of psychiatry's business. The two-stage view is supposed to give us a decisive answer: that brain activity is not just different, but disordered, and we can show it scientifically.

An alternative view is that disease concepts have a different structure altogether. Cooper (2007) and Murphy (2006) have drawn an analogy between the

concept of mental disorder and that of weeds. Weeds are not a scientifically relevant category of entities. We can perhaps say that a weed is a fast-growing species that negatively impacts on economically valuable crops, usually through competition for nutrients, sunlight, and space. What fixes the extension of "weed" (and similar concepts like "vermin" or "precious metal") is a set of contingent human interests that can change over time.

There is nothing inherently weedish about a species; weeds are just species that we don't like because of certain interests that we have. Suppose that determining that a condition is a disorder is like determining that a plant is a weed. The judgment is determined by normative considerations that we have already made. But nonetheless there is real, explanatory mind-independent knowledge to be had about each sort of "weed." Or take "precious metals": these are just metals that are valuable. They are valuable because demand far outstrips supply, but the demand is based on their aesthetic qualities and the way they let you show off, not on their chemical properties or mere rarity. Aluminum used to be precious but now it is not. Other metals, like Bismuth, are rare but not precious. Being a precious metal is a matter of quite complex human considerations, but there is still perfectly good scientific knowledge of each one of them to be had.

It might be, then, that "mental disorder" or "disease" more generally, works like "weed" or "precious metal," in that it is a concept that is driven by human concerns. We respond to people in a way that makes us judge them to be pathological. We can uncover facts about them, but the facts do not explain why they are judged to be pathological; they rather supply the causal story about the behaviors that lead us to make the judgment in the first place, and that judgment would be in place even if the facts were different.

I'll call this the norm-first view. Unlike pure constructivist views it acknowledges the role of science in establishing genuine knowledge of the physiology or psychology underlying a diagnosis and in providing opportunities for remedy and manipulation. The norm-first view contrasts with the two-stage view in holding that science does not uncover dysfunction in a way that is independent of our value judgments; science is directed by those value judgments. We first ascertain that someone is dysfunctional based on our socially generated expectations about what people ought to be like. Science can then investigate those people, but it does not determine whether they are pathological. The order of discovery here is often the same as that which the two-stage view would lead us to expect. But the order of nature is the other way round.

Theorists have often thought that the presence of normativity in psychiatry would undermine psychiatry's status as a branch of medicine, but if diseases are like weeds, then all of medicine has the same normative status as psychiatry. Consider cancer as an example. It is an uncontroversial instance of a biomedical disorder.

If we want to understand cancerous cell development we have two ways of proceeding. One way is to initially proceed by building an idealized model of cell development in general. We can then model cancerous cell development by explaining what "breaks down" during development in order to explain cancer as a "biological dysfunction" of the normal cell development. An alternative would model cancerous cell development on its own terms. Groups of scientists could proceed differently: one group would understand cancer as a "biological dysfunction" whereas the second group would understand it as a particular natural kind of process. Both models seem capable of capturing precisely the same information with respect to understanding biological change and providing different points at which we can intervene to alter the process we have modeled. We can disrupt the course of cell development and we can disrupt the course of cancerous cell development.

But cancer cannot be both a biological dysfunction and a natural kind of biological development at the same time. The two-stage view bets that there are further scientifically discoverable facts that tell us definitively that cancer is really a dysfunction. But what fact is there that science can discover what discriminates between cancer as a dysfunction and cancer as an unusual developmental pathway? If there is no such fact, then we must reconsider whether science can play a foundational role in determining that conditions are disorders, as the two-stage view says. The skeptic's alternative is that science discovers important biological facts guided by prior normative judgments that something is a disorder.

At this point in the argument, a proponent of the two-stage view has another option to rebut the skeptic who embraces the norm-first view. This is to ask about the role of the system in the overall economy of the organism. The two-stage theorist who adopts a systemic view of function can say: look at what the system you are studying does for the organism. The reason cancer is a dysfunction is that it drives the organism out of equilibrium and into a new state in which other systems stop being able to act as we usually explain them. This approach also requires a way of differentiating normal from abnormal development; it defines normal development as the set of pathways that lead to the final, functional, adult form.

The systemic theorist, then, can use the idea of a natural hierarchy in the organism to defend the claim that disease perverts the functioning that is normal for an organism. However, this does not solve the problem of finding natural, mind-independent dysfunctions, for the proponent of the norm-first view can now ask what justifies our idealized or assumed "normal" systems? Variation in biological traits is ubiquitous. Therefore, establishing whether or not a mechanism is functioning normally depends on whether an overall picture of normality for the organism can be adumbrated in a way that

doesn't depend on our prior values. The proponent of a norm-first view doubts that can be done.

These arguments might occur anywhere in medicine. But this sort of skepticism that motivates a norm-first view does seem to be especially telling in psychiatry, in so far as it is concerned with how people live, think, and feel. Humans are a set of systems, but what is the overall function of the organism they all comprise? In other words, how should people live? If the two-stage view is to work we must be able to objectively determine when people are living dysfunctionally, because only then can we say that there is a failure of the overall system, and thus establish that a subsystem is dysfunctional in the sense that it is not making the correct contribution to overall functioning. At this point, then, the next move in the dialectic should be for me to tell you how we can find out what the good life for humankind is. I hope you see the problem. The norm-first view says that science cannot answer that question. However, there might doubtless be scientifically relevant findings, such as those that suggest that you might not want to consume three packets of cigarettes and a liter of vodka a day if you plan to thrive in the long term.

Nobody doubts that people often suffer horrible mental pain or become detached from reality. There is nothing wrong with responding to these plights in the hope of making things better. And in so far as big-V validation can understand the basis of these plights in our biological nature we should pursue it. But that is not the same as validating a claim about pathology. It is uncovering the causal basis of what we respond to when we respond to the mentally ill. The two-stage view thinks that we can establish a diagnosis with scientifically objective credentials. I have not refuted this claim, but I have suggested that to do so requires a scientific solution to the puzzle of what human beings ought to be like, and I do not like the odds of doing that.

4.10 **Conclusion**

The big-V idea of something really being there brings up two philosophical issues within psychiatry with special resonance: first, a metaphysical one about the relation of validity to scientific realism of some sort; second, a normative one about whether we can validate a diagnosis, or merely validate a scientific conjecture. Roughly, the first issue raises the question whether we can know if something is there, and the second raises the question whether, if we find something, we can call it a disorder. I have discussed these two issues in turn. "Validation" sounds like a justificatory concept, as though we were not simply confirming a hypothesis, but also attesting to the correctness of a normative claim. Underlying accounts of validity is the idea that we do not just find a physical malfunction or imperfection, but something more, a genuine disorder built

into the structure of the world. I don't think we can find that. Whatever biological or psychological phenomena we uncover will still leave the normative issues open. In that sense, we can't validate a diagnosis. We can just correlate it with part of the world's structure.

Note

1. This discussion draws heavily on Roe and Murphy 2011.

References

Bernard, C. (1927/1865). *Introduction to the Study of Experimental Medicine.* New York: Macmillan.

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 for the Theory of Social Behavior*, **6**, 61–84.

Cooper, R. (2007). *Psychiatry and the Philosophy of Science*. Stocksfield, UK: Acumen.

Cummins, R. (1975). Functional analysis. *Journal of Philosophy*, **72**, 741–64.

De Block, A. (2008). Why mental disorders are mental dysfunctions (and nothing more). Some Darwinian arguments. *Studies in the History and Philosophy of Science (Part C)*, **39**, 338–46.

Godfrey-Smith, P. (1993). Functions: consensus without unity. *Pacific Philosophical Quarterly*, **74**, 196–208.

Insel, T. R., Cuthbert, B., Garvey, M., et al. (2010). Towards a new classification framework for research on mental disorders. *American Journal of Psychiatry*, **167**, 748–51.

Kendell, R., and **Jablensky, A.** (2003). Distinguishing between the validity and utility of psychiatric diagnoses. *American Journal of Psychiatry*, **160**, 4–12.

Kitcher, P. (2013). Real realism: The Galilean strategy. In *Preludes to Pragmatism: Towards a Reconstruction of Philosophy*. Oxford, UK: Oxford University Press.

Millikan, R. (1984). *Language, Thought and other Biological Categories*. Cambridge, MA: MIT Press.

Murphy, D. (2006). *Psychiatry in the Scientific Image*. Cambridge, MA: MIT Press.

NIMH (National Institutes of Mental Health) 2011: NIMH Research Domain Criteria, Draft 3.1. Available at: <http://www.nimh.nih.gov/research-priorities/rdoc/nimh-research-domain-criteria-rdoc.shtml> (accessed October 2013).

Roe, K., and Murphy, D. (2011). Function, dysfunction, adaptation? In P. Adriaens and A. Block (eds), *Maladapted Minds* (pp. 216–37). Oxford, UK: Oxford University Press.

Schaffner, K. (2012). A philosophical overview of the problems of validity for psychiatric disorders. In K. Kendler and J. Parnas (eds), *Philosophical Issues in Psychiatry II: Nosology* (pp. 169–89). Oxford, UK: Oxford University Press.

Schulkin, J. (2011). *Adaptation and Well-Being*. Cambridge, UK: Cambridge University Press.

Wakefield, J. C. (1992). The concept of mental disorder: On the boundary between biological facts and social values. *American Psychologist*, **47**, 373–88.

Zachar, P. (2012). Progress and the calibration of scientific constructs: the role of comparative validity. In K. Kendler and J. Parnas (eds), *Philosophical Issues in Psychiatry II: Nosology* (pp. 21–34). Oxford, UK: Oxford University Press.

Chapter 5

Natural and para-natural kinds in psychiatry

Nigel Sabbarton-Leary, Lisa Bortolotti, and Matthew R. Broome

5.1 Introduction

The aim of this chapter is to investigate how we should understand kinds in psychiatry. That psychiatry has kinds of some sort is uncontentious; the ever-growing list of mental disorders is testament to that. The problem with the classificatory categories set out in manuals like the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) is to articulate precisely what mental disorders amount to. The question that arises is: Are the classificatory categories of psychiatry natural kinds?

Psychiatrists concerned with classification are interested in a number of interconnected issues: 1) whether, or not, a particular mental disorder is *real* or *valid*—that is, whether it is a *bona fide* piece of the furniture of reality; 2) whether the disorder has inductive potential—that is, whether the symptoms cluster uniformly across all instances of the disorder and the category supports inferences about treatment; 3) what the causal history of the disorder is; and 4) just how informative the classificatory category is.

Philosophers discussing whether kinds are natural kinds are interested in the same set of issues (see Beebee and Sabbarton-Leary 2010 for a discussion of contemporary issues). On most standard definitions a natural kind is a discrete, mind-independent entity marking a real division in nature. These natural divisions constitute the classificatory units of the sciences (e.g., physics, chemistry, biology, and so on). The periodic table is the exemplar *par excellence* of science's endeavor to discover and demarcate nature's joints. Furthermore, the discovery of such natural divisions by a particular branch of empirical enquiry seemingly validates that branch of enquiry, elevating it to the status of a science.

The classification of the objects of experience—the things we see, hear, smell, feel, and taste—into natural kinds also brings with it a number of fringe (epistemic) benefits. Knowing that the animal one happens across whilst on an Asian safari is a member of the species *Panthera tigris*, for instance, allows us to

make useful and potentially life-saving inductions about the likely behavior of the said animal: When it issues a low and throaty growl as it stalks us we know it is time to make a hasty retreat. Similarly, since kind concepts are projectible,[1] knowing that the substance in front of us is water allows us to induce it is (likely) potable, since typical members of the kind *Water* are potable.

Of course, not all objects of experience can be classified into natural kinds. Many of the items found in a kitchen, for instance, form kinds of some sort—pots, pans, utensils, ingredients, etc.—but these kinds are not natural. Rather, they are artificial kinds. The contrast is fairly straightforward, at least at the level of intuition. Consider the subatomic particle *electron* on the one hand and the kitchen utensil *spatula* on the other. Electrons are negatively charged particles that are responsible for the substance-forming bonds between atoms of different elements. Covalent bonds between atoms of chlorine, for instance, are created when a pair of atoms "share" a pair of electrons—each atom contributing one electron—giving each atom eight electrons on its outer electron shell. Electrons have definite characteristics; their mass, charge, velocity, and so on are uniform between each member of the kind. Put more generally, they have key determinate properties that all and only members of the electron-kind possess; they are natural kinds.

Spatulas, in contrast, are not uniform. They do not have a determinate material or dimension. They are characterized by their function: Roughly, for flipping, turning, stirring, and scraping foodstuffs. But many objects can be employed to fulfill the function of a spatula. A ruler, for instance, could be used to flip a frying pancake, stir a cooking stew, or turn a poaching fish. This interchangeability of function between items of disparate design typifies the intuitive natural/artificial distinction. We take that distinction to boil down to something like the following: There are many reasonable ways to group and divide the objects of our experience. Some groupings and divisions latch onto real distinctions in nature. Their investigation is subject to the highest degree of methodological scrutiny, that is, scientific investigation, and they survive such scrutiny. They are natural kinds. Other groupings and divisions do not survive such scrutiny. They do not latch onto real distinctions in nature but capture different functions the objects of experience might have.

Within the philosophical literature one of the perceived challenges for psychiatry is to justify that its kinds are natural. The critique of the 1960s and 1970s has left psychiatry with vestigial questions concerning its scientific credentials (Murphy 2006, 2009; Cooper 2007, 2009; Pickard 2009). The anti-psychiatry movement went even further and questioned whether mental illness represents the medicalization of socially deviant behavior, and the pathologizing of normal problems of living (Bolton 2008: 163; see also Bortolotti 2013). The thought was that the label of mental illness not only fails to denote a natural

kind, but does not denote anything at all. Szasz argued, for instance, that mental illness is just a "convenient myth" (Szasz 1961: 113). If entities classified as mental disorders could be shown to be natural kinds, then progress could be made with many of the controversies surrounding the very notion of mental illness.

5.2 Clarifying the Challenges for Psychiatry

There are two challenges faced by psychiatry. The first root pressure is metaphysical: Are mental disorders natural kinds? The second pressure is normative: What behaviors should receive medical treatment?

The first challenge has significant contemporary bite when we consider the ever-increasing list of so-called mental disorders. The list of mental disorders stood at 374 in DSM-IV-TR compared to 106 in DSM-I (Grob 1991: 421–31). Since the DSM has only been running since 1952, this trend indicates a staggering increase in i) our ability to recognize and diagnose mental disorders, ii) our propensity to classify behaviors as mental disorders, or iii) the scientific, clinical, or political need for a more fine-grained taxonomy of disorders, with previous disorders being split into multiple disorders.

The second challenge faced by psychiatry is to some extent a more general problem in medicine.

> Psychiatry's crisis revolves around the question of whether the categories of human distress with which it is concerned are properly considered "disease" as currently conceptualized and whether exercise of the traditional authority of the physician is appropriate for their help functions. Medicine's crisis stems from the logical inference that since "disease" is defined in terms of somatic parameters, physicians need not be concerned with psychosocial issues which lie outside medicine's responsibility and authority. (Engel 1977: 129).

Engel's characterization is useful since he identifies important issues that are interconnected, from a practitioner's point of view, with the justification of psychiatric illnesses as natural kinds. How far does the "traditional authority" of the physician extend? Should all behaviors associated with mental disorder be medically treated? If so, how far along the continuum of human behavior should this treatment extend? In the context of mental disorders, it is also important to ask what "medical treatment" amounts to. Medical treatment is not synonymous with pharmacological treatment. For instance, it has been argued recently in the context of personality disorders that it is appropriate for medical treatment to take the form of a "retraining of habits," which can be achieved in a community environment with the assistance of different therapies (Pickard 2009). In addition, as science advances, the scope of what can be

treated also expands, and what may have been considered an "enhancement" by one generation could be considered a therapy by the next.

There is a range of human behavior that can, more or less, be quantified across a range of variables. For instance, human responses to aggression could, in principle, be statistically mapped. Those responses might include behaviors including: a) answering aggression with aggression; b) answering aggression with violence; c) answering aggression with passivity; or d) answering aggression with panic. Now, imagine that a particular individual, call her Hannah, reacted to aggression in a harmful fashion—say, physical violence, impairing her ability to form healthy, meaningful social relationships, and to function effectively within typical social environments. Does Hannah's behavior qualify as medically treatable? If so, why is her particular behavior medically treatable as opposed to, say, Peter's response to aggression—say, total passivity, that also impairs his formation of healthy and meaningful social relationships? What should Hannah's medical treatment amount to? Does society's view of Hannah's response—as one which is inherently problematic—make it more likely that Hannah, rather than Peter, will receive treatment?

Here, the concern is that, if mental disorders are not genuine disorders, and the boundaries of mental illness are drawn arbitrarily, then it may not be justified to treat people diagnosed with those disorders medically. Engel's characterization of the crisis in medicine now comes into play. On Engel's account, a wholly somatic characterization of medicine rules out some psychosocial harms that impinge on people's quality of life. Thus, these impingements either go untreated, leaving those individuals to suffer, or are re-conceptualized as criminal acts and punished. Such considerations do not necessarily set psychiatry apart from the rest of medicine. Physical health has arbitrary boundaries, too. How many bacteria need to be found in a lung for the case to be a case of pneumonia? Chronic illness, earlier detection of illness, and the expanding boundaries of our conception of "ill health" all lead to a need for doctors to manage impaired glucose tolerance as well as diabetes, polyps, and tumors. There is a sense in which the whole of medicine, including psychiatry, deals with fuzzy boundaries. Medicine needs to manage less severe illnesses, more illnesses, and more chronic illnesses than in the past due to our longer lifespan.

One strategy for accounting for kinds in psychiatry we would like to explore in this chapter is to argue that the category "mental disorder" should be restricted to all and only those disorders that have biological causes. This strategy comes with the following caveat: There are mental *harms* that should be treated medically, if we assume a broad conception of "medical treatment," but do not count as mental disorders. We shall examine this proposal in some detail, and highlight its benefits and limitations.

5.3 The Metaphysics of Medicine

There have been different strategies to resist the challenge that mental disorders are not natural kinds. They include: i) justifying the category of mental illness as a category of natural kinds; ii) sketching out the different philosophical notions of natural kind, and illustrating how mental illness falls within the scope of one such notion; and iii) presenting an alternative concept to that of a natural kind, such as a practical kind, that is scientifically respectable and accounts for mental illness.

> In response to the general challenge formulated in the 1960s, reinvigorated by debate in the 1970s, the task was clearly set to define mental disorders, to make clear the basis of why conditions were in the manuals or excluded, and clear that this basis was medical—scientific and objective—not a matter of social rules of normal behaviour. (Bolton 2008: 164)

The view we want to explore is broadly naturalist. Hence our investigation of the metaphysical challenge begins with a brief discussion of medical nosology, since the concepts of mental illness, disease, disorder, and impairment are clearly intended to be analogous to established physical counterparts. At this stage it is worth noting that nothing in particular hangs on the distinction between mental illness, disease, disorder, or impairment as we employ those terms. Toward the end of the chapter, we shall draw a terminological distinction between disorders and harms, but until then we will use the current terminology of psychiatry somewhat interchangeably.

What are diseases according to medicine? The Hippocratic tradition construes diseases as entities. The presence of a disease-entity within a host is indicated by a defined cluster of signs or symptoms. Consider for example *Transmissive Spongiform Encephalopathy* (or TSE). The presence of TSE in a host is indicated by two physical changes: The degeneration of the brain and the spinal column, and the formation of amyloid plaques in the extracellular matrix. There are also behavioral symptoms. Hosts exhibit a general deterioration in behavior, including rapidly progressive dementia and psychotic symptoms, ataxia (an unsteady gait), and myoclonus (sudden jerky movements). The cause of TSE is attributed to a protein called a *prion*. The prion protein can be found within the brain and spinal column. During standard cell replication the prion protein, specifically in the brain and spinal tissues of the host, misfolds. The misfolding converts the normal prion protein (or PrPc) into a toxic counterpart (PrP^{Sc}), which is ultimately responsible for the physical and behavioral symptoms we observe.

TSE conforms neatly with the disease-as-entity model espoused by modern medicine. The toxic prion protein PrP^{Sc} is a natural kind; it is a mind-independent, objective ontological entity. Moreover, it is causally responsible for a particular set of symptoms used to diagnose TSE in a host subject.

Moreover, the identification of PrP^{Sc} allows the cause of TSE to be empirically investigated, analyzed, and eventually treated.

The construal of diseases-as-entities appears to be a metaphysical condition of medical nosology. It is not accompanied by an epistemic counterpart: Diseases are classified despite the entity responsible for the symptoms being unknown. For instance, Caffey disease (or *Infantile Cortical Hyperostosis*) is a disease of unknown cause. Affected host symptoms include bone lesions, soft-tissue swelling, hyperesthesia, tenderness, and irritability. In extreme cases paired bones (e.g., tibia and fibula) can become fused. Recent research claims both that "an underlying viral aetiology has been implicated" (Hall 2005: 2). A genetic mutation within a collagen-encoding gene (*COL1A1*) has been identified, during a genome-wide screen of affected individuals (Gensure et al. 2005: 1250). Despite this, the existence of the disease is (fallibly) indicated by the existence and stable clustering of the symptoms in repeated cases. The category *Caffey disease* has the hallmarks of a natural kind: It is mind-independent, apparently causally stable, projectible, and epistemically useful. This, it seems, is sufficient to give medical science confidence that there is a cause of Caffey disease, consequently construing it as a natural kind.

This does not imply that there are no extant questions concerning the kind category. For example, it may be that there are two forms of Caffey disease that do not share a common causal basis. A pre-natal form of the disease has been identified, occurring before week 35 of the gestation cycle. The contraction of pre-natal Caffey disease results, typically, in perinatal death. However, the mutated *COL1A1* gene is absent in pre-natal Caffey disease. Thus the relationship between pre- and post-natal versions of the disease is unclear. But this need not threaten the inclusion of Caffey disease in medical ontology—the disease has been successfully identified. Rather, investigation into the precise mechanisms that produce the indicative symptoms continues.

Medical taxonomy's outstanding challenge is to determine whether pre-natal Caffey disease—which is symptomatically identical but etiologically distinct (with no *COL1A1* gene mutation)—is a *bona fide* instance of the disease. There are two options: i) expand the category of Caffey disease to include both pre- and post-natal variants, and expand our conception beyond the mutation of *COL1A1*; or ii) maintain that Caffey disease *is* the mutation of *COL1A1* and carve out a new category of disease corresponding to the pre-natal form, given its distinct cause.

The example elucidates philosophically interesting features of physical disease classification. Most pertinent is the appeal to a "disease-as-entity model" that underlies medical nosology (Caplan et al. 2004: Part 1). The example demonstrates that the metaphysics of medical nosology is one of kinds that aspire

to be natural kinds. Diseases are predominantly classified as discrete units of our *ontology*; they are independently existing, objective entities. As members of the natural kind club they enjoy membership perks: They are law-like in their interactions with other kinds, they are projectible (in the inductive sense), and their readily observable features are a product of a unified internal nature or structure. As Georges Canguilhem notes, in his discussion of Louis Pasteur's germ theory of contagion, what makes the position philosophically attractive is that "it embodies an ontological representation of sickness . . . a germ can be seen" (Canguilhem 2004: 41).

Philosophically, this is an enticing prospect for psychiatry. If a disease or illness is a *bona fide* ontological object, then it can be reliably investigated without (ontological) reservation. Moreover, even if our understanding of it is incomplete, the existence of the illness is not at issue. The symptoms of *Infantile Cortical Hyperostosis*, for instance, are observable and empirically investigable. The bone lesions, soft-tissue swelling, and irritability of the host cluster together reliably. Granted, the cause is currently unclear, but the evidence suggests that the category is a natural kind.

However, mistakes may occur. We may be mistaken, for instance, about some of the symptoms of TSE. It could be that the aggressiveness of many humans is, in actual fact, caused by some other disease that tends to accompany, but is not actually a feature of, TSE. But mistakes do not undermine the utility of the thesis. Rather, they are a reminder of the requirement for methodological rigor—hypotheses are subject to revision in light of confounding evidence.

5.4 The Metaphysics of Mental Disorder

The ontological attraction of the disease-as-entity model is obvious. The challenge for psychiatry is to justify its categories as ontologically respectable by showing why mental disorders should be construed as a *bona fide* feature of the furniture of reality, defined by their objective features and subject to the highest standards of scientific scrutiny. *Naturalism* attempts to answer this challenge by situating psychiatric illnesses firmly within biology. What follows is a brief sketch of two versions of naturalism to illustrate the sort of position that would offer a viable account of mental disorders as natural kinds.

The central tenet of naturalism in psychiatry is that mental disorders can be identified with natural facts. Perhaps the two most famous views are those developed by Christopher Boorse (1975) and Jerome Wakefield (1992). According to Boorse, the human body is made up of sub-systems that have natural functions. The function of the amygdala sub-system, for instance, is to process memories and emotions. It is also thought to be responsible for the

regulation of memories associated with both negative and appetitive conditioning (see for instance Killcross et al. 1997: 377–80). The idea is that sometimes systems become dysfunctional. Liver dysfunction, for example, can lead to a variety of illnesses and diseases. When the liver becomes infected or damaged by, say, alcohol, it no longer effectively removes bilirubin from the blood. The resultant increase in bilirubin levels in the blood results in jaundice.

Analogous examples can be found within sub-systems in the brain. For instance, the International Classification of Diseases (ICD) entry F07.0 *Organic Personality Disorder* captures so-called Klüver–Bucy syndrome, where an affected individual may suffer from various symptoms including polyphagia—excessive hunger. The cause of this behavior is attributed to lesions of the anterior temporal lobe (Klüver and Bucy 1939: 979–1000). These lesions are causally responsible for a general dysfunction of the amygdala sub-system, resulting in Kluver–Bucy syndrome. Central to Boorse's account is the notion of "function." For Boorse the functionality of a system is determined via its normal role within an organism, where that role contributes to biological success (i.e., survival and reproduction). As such, "dysfunction" can be construed as a disruption to the normal role of a system hampering biological success. Wakefield's naturalism similarly appeals to the notion of function. On Wakefield's view there are various mental mechanisms at play in human cognition. When the normal function of a mental mechanism is disrupted, becoming dysfunctional, we have a mental disorder. The key difference between Boorse's account and Wakefield's is that for Wakefield the notion of a normal function is determined in relation to the evolutionary design of the mechanism. Wakefield is clear that what he has in mind when he talks of normal function is *natural* function.

> A disorder is different from a failure to function in a socially preferred manner precisely because a dysfunction exists only when an organ cannot perform as it is naturally (i.e., independently of human interactions) supposed to perform. Presumably, the functions that are relevant are natural functions. (Wakefield 1992: 381)

The general feature of Boorse's and Wakefield's respective account that is relevant to our discussion of the medical model is that both appeal to biological features of organisms to provide an account of mental dysfunction. Appealing to the natural functions of the organism to explain mental disorders coheres well with the medical model. Illnesses are etiologically connected to empirically investigable, mind-independent physical entities. In other words, they are natural kinds.

There have been various objections to both Boorse's and Wakefield's respective versions of naturalism. We do not discuss those objections here, but instead direct readers to Rachel Cooper (2007) for a useful survey of these positions. Our discussion is intended to be higher-level in so far as we are not proposing a

particular account of naturalism *per se*, but endorsing a naturalist approach to the classification of mental disorders.

5.4.1 Mental Disorders in the Medical Model

How does the disease-as-entity model apply to a paradigmatic psychiatric disorder? To illustrate the sort of position we have in mind let us consider a mental disorder that has been revised for the most recent iteration of the DSM, namely *Autism*.

In DSM-5 the various species of autism have been unified with the introduction of a single disorder *Autism Spectrum Disorder* (ASD). ASD merges four discrete disorders (which were categorized separately in DSM-IV): 1) *Autistic Disorder*; 2) *Asperger's Disorder*; 3) *Childhood Disintegrative Disorder*; and 4) *Pervasive Developmental Disorder*. Like the ICD, the DSM sketches out the symptoms affected individuals will likely exhibit. According to DSM-5, people with ASD typically exhibit a communication deficit. What this amounts to is a general propensity to misread non-verbal interactions and an inability to build age-appropriate friendships. Individuals can be highly routine-dependent, and extremely sensitive to change. They may also be overly attached to inappropriate items.

In the context of the current debate concerning the classification of mental disorder, and the methodology of medical nosology, we might first ask: What is, or what causes, ASD? Underlying that question is a philosophical one: Is there some ontologically respectable entity responsible for the symptoms exhibited (on a spectrum) by subjects? Unfortunately, the causes of ASD remain largely unknown, although there are four causal categories that are typically mentioned as possible candidates. These are: 1) genetic factors; 2) environmental factors; 3) psychological factors; and 4) neurological factors.

Each category postulates its own cause of ASD, sometimes in combination with factors from other categories. For instance, genetic factors are thought to be responsible for an individual's susceptibility to ASD, due to the correlation between the development of ASD in identical twins. According to the National Health Service (NHS), where one twin has ASD there is a 60 percent chance that the other twin will also develop ASD. In addition, it is also thought that to develop ASD an individual requires an environmental trigger.

The psychological factor thought to be causally responsible for ASD is that the subject lacks a comprehensive theory of mind. The idea, roughly, is that individuals with ASD do not possess an adequate theory of mind, thereby failing to attribute mental states to other individuals. This theoretical gap, or so the hypothesis goes, accounts for their inability to deal adequately with social interactions and to form appropriate relationships.

Finally, neurologists cite two possible explanations of ASD. The first is the connection between the amygdala, the limbic system, and the cerebral cortex. According to this hypothesis, in individuals with ASD the connections between these systems have somehow become jumbled. The empirical basis for the hypothesis comes from neural imaging studies that appear to show signaling differences between individuals with and without ASD. This neural cross-wiring is thought to be responsible for the abnormal responses ASD sufferers exhibit toward trivial events. The second attributes ASD to a difference in the function of mirror neurons found in an individual's brain. Mirror neurons, which allow us to mimic the behavior(s) of others, are thought to function differently in people with ASD. Since mirror neurons are responsible for language acquisition and recognition of emotion, and furnish us with an ability to learn from others, the deficits ASD sufferers experience in these areas may be attributable to dysfunctional mirror neurons.

The genetic, environmental, psychological, and neurological causes of ASD cited in the literature are not necessarily distinct. For example, neurological changes could underpin psychological and cognitive changes, which may have been caused by environmental or genetic effects. To complicate things further, the propensities of such an organism may further lead to that individual being exposed to other risks, such as bullying or social isolation, that may compound the issue, leading to further genetic and neurological changes.

The ASD example is interesting for a number of reasons. There are different but potentially complementary causal stories we can tell (more than one type of factor may contribute to a given case of ASD and different cases of ASD may have different causal etiological structures). These alternative causal explanations reveal our classificatory intentions, but need not undermine our confidence in the conceptualization of ASD as a natural kind—remember the Caffey disease example. However, not all the causal accounts of ASD are as ontologically respectable. Compare and contrast the ontological attraction of a broadly neurological account, citing genetic and environmental triggers, with purely psychological accounts—the former, but not the latter, has clear parallels with the disease-as-entity model that dominates medical nosology. Tracking ASD back to either a particular way that electric and chemical signals are transmitted between the amygdala, limbic system, and cerebral cortex, or to dysfunctional mirror neurons, has clear parallels with Louis Pasteur's germ theory of contagion, and coheres well with the disease-as-entity biomedical model. The potential of such an account is illustrated by one of the inaugural stories of biological psychiatry—namely, that the myriad psychiatric manifestations of syphilis, including general paralysis of the insane, has been causally traced back to an infection by *Treponema pallidum*, and the discovery of spirochetes in the brain of those affected (Pearce 2012).

Issues surrounding the etiology of ASD (and other mental disorders) need to be considered next to analogous cases in medical nosology such as Caffey disease, where the cause is uncertain, but the stable cluster of symptoms is well established, and the classificatory category appears to have significant empirical utility.

5.4.2 A Theory of Psychiatric Kinds as Natural Kinds

The position we want to describe and test is that there are objective, mind-independent facts that are the causes of mental disorders. Once these causes have been identified an additional piece of furniture is added to our theory of the natural kind structure of the world. The precise shape of that piece of furniture may take some time to become fully defined, but this is an epistemic rather than a metaphysical problem, affecting all branches of classification across the sciences. The state of our knowledge may be such that we are unable to determine precisely what the cause of such-and-such a mental disorder is.

One unique problem psychiatric classification faces is that the main symptom of a disorder is often a behavior or a first-person account of a particular behavioral episode. Because there are numerous causes of behavior, the accurate diagnosis of a particular set as disorderly is particularly challenging. The key conceptual point is that we should restrict the category of mental disorders to those with biological causes, aligning the main conceptual category of psychiatry to the prevailing strong version of the medical model. The advantage of the naturalist position is that it is less metaphysically controversial and more empirically respectable. Moreover, the construal of mental disorders as natural kinds brings with it a host of fringe benefits. The move alleviates pressure from the (in)famous critique of the 1960s and 1970s; the condition on the classification of mental disorders—that they must be natural kinds—along with convincing examples like ASD reduces the critique to battles over the reality of *particular* illnesses, not the category "disorder" *simpliciter*. And this is a benefit, since individual battles over particular disorders will assist psychiatric classification by promoting methodological rigor.

The difficulty is that psychiatry often starts from a psychological phenomenon deemed "disordered" on the basis of whether psychological characteristics fit normative frameworks of good functioning, rationality, social acceptability, etc. When it is possible to identify causes of the disordered phenomenon, these may not be entire pathological mechanisms but deviations resulting in slight qualitative changes to normal function. As such, not all the kinds classified in DSM-5 will qualify as natural kinds, and hence as disorders, within the framework. To put it another way, some conditions that are presently treated by

psychiatrists do not qualify as mental disorders. To account for these kinds that are not disorders we introduce the notion of a para-natural kind.

5.5 Para-Natural Kinds and Mental Harms

Roy Sorensen has recently introduced a novel notion that may assist current efforts to conceptualize psychiatric kinds, namely the notion of a para-natural kind. According to Sorensen, a para-natural kind is parasitic upon some natural kind or other; it is "an absence defined by a natural kind" (2011: 113). Sorensen explicates his idea with two intuitive examples. Consider the natural kind *heat* and its counterpart *cold*. Where heat is defined (very) roughly as molecular motion, cold is simply the absence of heat (i.e., the absence of molecular motion). Hence cold is parasitic upon heat, and a para-natural kind. Similarly one can distinguish the contrasting pair shadow and light. Where light is a natural kind (i.e., the presence of photons), shadow can be defined as the absence of light and thus, again, a para-natural kind.

So, parasitic kinds are *not* natural kinds. They are *absences of actual natural kinds*. However, they do appear to have some of the characteristics of natural kinds in so far as they "inherit the lawfulness and projectibility of the natural kinds that shape them" (Sorensen 2011). To illustrate, consider, for instance, the electron hole postulated by Paul Dirac to explain the "intense chemical activity of chlorine" (Sorensen 2011: 119). The idea is that since the outer shell of a chlorine atom has seven electrons, but space for eight electrons, there is a kind of energy hole, a gap in the electron shell that has the opposite charge to an electron (i.e., it has a positive charge). This electron-gap attracts atoms of other substances which, in turn, can result in intense chemical activity.

Moreover, these interactions are law-like and projectible. We can and do make reliable inductions about chlorine's chemical activity on the basis of the electron-gap, and knowing that other atoms have similar electron gaps helps us to induce information about them. Ontologically speaking, since we have a prior commitment to the existence of electrons—they are *bona fide* natural kinds, and part of the furniture of reality—the explanatory power of the electron-gap, which is to say the *absence* of an electron, comes with no ontological costs. We do not have to postulate some further mind-independent entity to explain an observable phenomenon, but we nevertheless are able to provide an accurate and true explanation, which is consistent with our current theory.

How might this idea extend to psychiatry? Well, first we need to identify a natural kind to work with, and be ontologically committed to, around which we can then note the absence(s). One starting point would be to construe human cognition as such a kind, albeit of a more general nature. Nancy Andreasen

argues that schizophrenia can be conceived of as a deficit in certain information processing systems (see for example Andreasen 2000). Inspired by her account, we could say that, when the brain as a complex set of information processing systems and sub-systems is operating sub-optimally, what causes the sub-optimal functioning can be divided into two broad categories: Natural kinds and para-natural kinds.

The natural kinds will be those sorts of causes that have a biological basis, which are independent items of our ontology. The etiology of these sub-optimal states will be pathophysiological, and the disorder should be identified with that cause. For instance, brain lesions are known to cause behavioral changes, mental confusion, and loss of memory, amongst other things. As such, any behavioral changes that symptomatically match a mental disorder, which can be causally attributed to a lesion in a particular region of the brain, genetic mutations, or infective agents would conform to this sort of model. On this view, sub-optimal states with the correct type of etiology are natural kinds.

The para-natural kind category will capture the other causes of sub-optimally functioning processes that are not attributable to an independent natural kind, but rather to the absence of the fully functional information-processing system.[2] The para-psychiatric kinds will be those mental harms that are not grounded in specific biological causes *per se* (although the causes may well have a pathophysiological realizer, in the sense that all behaviors must be physically realized), but are nevertheless sub-optimal states of the information-processing system. Consider, for instance, depression, whose changed diagnostic criteria in DSM-5 has caused a stir. Imagine an agent that qualifies for a diagnosis of a major depressive episode under the auspices of DSM-IV-TR (satisfying five of nine qualifying criteria over a two-week period). Now, imagine that the cause of that agent's depression is bereavement, and that the qualifying factor leading to the diagnosis—the straw that broke the camel's back, so to speak—stems from the fact that they now fall outside the arbitrary grief time-limit DSM-IV places upon patients. Whilst the symptoms the agent exhibits will be physically realized if depression is construed as a para-natural kind, there need be no biological etiology for the episode.

According to the proposal, only natural kinds are appropriately labeled "disorders," whilst para-natural kinds are not. However, the scope of behavior that can be treated by psychiatrists is broader than the category of mental disorders, and can extend to para-natural kinds provided treatment decisions are subject to robust scrutiny.

5.5.1 Challenges for Para-Natural Kinds

Consider major depressive episodes, which appear to lack a biological etiology. On our account they are a species of para-natural kind. This, we claim, coheres

well with recent work by Horwitz and Wakefield (2007), discussed by Dominic Murphy (2009: 110), who identify a challenge for the contemporary category "depression." They claim that it is applied without common sense, using symptoms that capture far too much normal human behavior, and that as such there is a "needless alarmism about an epidemic of depression" based upon erroneous diagnosis (Murphy 2009: 111). The root of the challenge can be illustrated via an appeal to one of philosophy's oldest puzzles concerning vagueness, the *Sorites paradox*.

Consider a heap of sand consisting of 10,000 grains. Does the heap remain a heap if we remove one grain of sand? Since removing one grain of sand does not make much difference to the heap, and since 10,000 grains of sand constitute a heap, so too should 9,999 grains. However, repeating this process eventually leads to one grain of sand, which definitely is not a heap. Where, then, is the boundary between heaps and non-heaps? The point is that at each step of the process (of removing grains of sand) we are inclined to say that no significant change has occurred. Hence the boundary is a vague one. Consider a man, Andrew, who has lost his wife, and now experiences symptoms associated with a major depressive episode, including: A depressive mood, diminished interest in life (e.g., work, his house, and so forth), diminished appetite leading to weight loss, insomnia, and fatigue. For the sake of argument let us imagine that Andrew experiences these symptoms for 30 days. On day 1 Andrew is grieving within normal human limits. The loss of a loved one is a significant negative event that can have a serious impact upon cognitive equilibrium. Since there is no significant difference between day 1 and day 2, on day 2 Andrew is grieving within normal human limits, and so on until we reach day 30. However, according to DSM-IV-TR, if the symptoms continue beyond 14 days then the agent in question is experiencing a major depressive episode. So, on day 15 Andrew's grief has developed into a major depressive episode. But if there is no cognitive change in Andrew between days 14 and 15, and the only difference is the length of time he has experienced these symptoms, then the diagnosis appears as arbitrary as, say, stipulating that anything less than 5,000 grains of sand is *not* a heap, whilst anything more than 5,000 *is* a heap.[3]

The problem is that arbitrarily stipulated boundaries for mental disorders seem to undermine a) the disorder's scientific respectability, and b) *a fortiori* the scientific credentials of psychiatry. The purported "disorder" cannot satisfy some of the more conservative ontological credentials of natural kind-hood, namely an ontologically discrete entity with a decisive boundary separating it from all other kinds of entity. In the proposal we are examining, such kinds should be construed as candidate para-natural kinds. Horwitz and Wakefield, we claim, are correct about the erroneous diagnosis of major depressive episodes

based upon the way we have conceptualized it—as a natural kind. The arbitrarily drawn boundaries of a major depressive episode, in conjunction with the lack of a unified causal etiology, disqualify such episodes from being natural kinds. But are such episodes para-natural kinds?

Since a major depressive episode is an absence of an optimally functioning information-processing state, from day 1 it is an absence, as we would expect from a para-natural kind. Similarly, major depressive episodes are, by definition, uniform; to qualify as an episode the majority of the nine available symptoms listed in DSM-IV-TR must be present. The arbitrariness of stipulating that there is only a major depressive episode after 14 days—given that Andrew feels precisely the same on day 1 as he does on day 15—still presents a residual challenge. But importantly, that challenge is *not* to the category of mental disorders (via implication), since a major depressive episode is *not* a mental disorder.

The proposed distinction, then, is as follows: On the one hand, there are natural kinds, which are those illnesses of the mind with a biological etiology, firmly rooted within the ontology of the medical model—that is, *bona fide* entities that are categorically distinct and properly called mental disorders. They result in a sub-optimally functioning information-processing system, and qualify for medical treatment of a pharmacological nature, since it is the biological cause of the symptoms that is the target of the treatment. On the other hand, there are the para-natural kinds of psychiatry, which are those cognitive states that are harmful to the agent without a clear and distinct biological etiology (although they are, of course, biologically realized). They are characterized as absences of optimally functioning information-processing states, with various causes. They are harmful mental states in which an agent may find herself, where she requires access to medical treatment broadly construed. The harms are biologically realized, in so far as they occur (in some sense) within human minds and brains. However, although pharmacological treatment can impact the acuteness of the symptoms—since symptoms are biologically realized—such treatment may be inappropriate, since there is no biological cause to target and alter.

Exploring the extent and prevalence of para-natural kinds amongst the taxonomy of psychiatry within the latest editions of the DSM is a larger project than we can investigate here. But the benefits of para-natural kinds for psychiatry will include a fairly open set of potential causes of sub-optimal information-processing states, including psychosocial causes, environmental causes, and so on. Para-natural kinds are still a proper feature of the taxonomy of psychiatry (along with natural kinds), but they are *not* mental disorders.

Para-natural kinds, then, are accompanied by their own unique problem of demarcation, when distinguishing between health and illness. Consider the nosological status of psychotic experiences, such as hearing voices and having

unusual beliefs, in young people. When does prodromal psychosis become a first episode? Here, the experiences could be within the range of normal experience and development, but conversely, may also signify the prodromal phase of schizophrenia or even a frank first episode of a psychotic illness. What does seem to be the case is that the nature of the experiences themselves may not help us in demarcating pathology from non-pathology. It is other factors, such as intensity, duration, distress, or additional dysfunctions such as cognitive impairment or depression (Murray and Jones 2012; Broome and Fusar-Poli 2012; Broome et al. 2013) that may help determine whether the prognosis of such experiences is clinically of interest and where intervention may prevent future harms.

None of this implies that the investigation of para-natural kinds is not an appropriate practice for medical science; quite the contrary. The point, rather, is that the current list of mental disorders are not all natural kinds, and that the effective conceptualization of psychiatric kinds is critical to our understanding of both mental harms and mental disorders, and critical to the appropriate treatment(s) available to the agents affected by them.

5.6 Conclusions and Implications

The natural kind account of mental disorders we have considered in this chapter aims to align the nosology of mental disorder with that of contemporary medicine. The benefit of the account is that it avoids some of the vestigial pressures of the anti-psychiatry movement, being realist, naturalist, and broadly empiricist. The inclusion of the notion of a para-natural kind helps account for those mental harms that are *not* natural kinds but are, nevertheless, worthy of the attention of clinicians and medical professionals. Both conceptions we have discussed rely upon an underlying notion of a general natural kind of effectively/appropriately functioning human cognition. We have adopted the notion of an optimally functioning information-processing system, but other accounts may be available. An underlying notion of a natural kind of human cognition is vital to the account, to distinguish disorders and harms from orderly mental processes—after all, orderly mental processes such as happiness are certainly physically realized in precisely the way we have envisaged mental disorders to be.

How we conceptualize psychiatric kinds is fundamental to how we understand and treat mental disorders and mental harms. In this chapter we have attempted to demonstrate that psychiatry has natural kinds, called "mental disorders," but that the various iterations of the DSM have *not* restricted their nosology to them. The list of mental kinds of psychiatric interest has increased with each issue of the DSM, and includes both natural and para-natural kinds, that is both mental disorders and mental harms. The recognition of two

distinct categories of mental kind within the DSM will, we hope, lead to a more fine-grained approach to the way we think of mental disorders.

Finally, the recognition of natural kinds in psychiatry provides the discipline with the sort of theoretical basis enjoyed by other branches of empirical medicine and science more generally. Mental disorders are no more mythical than misfolded prion proteins or mutated collagen-encoding genes. Nevertheless, the precise extent of psychiatry's natural kinds is something in need of more study and discussion. Our hope is that the framework we have outlined here will contribute to that discussion.

Notes

1. The notion of projectibility within philosophy was introduced by Nelson Goodman (1984) and is connected to the notion of induction and inductive inferences. The idea is that a concept is projectible when it supports an inference from premise to conclusion that is ampliative. Or, to put it another way, when the concept within the premise of an argument supports an inference to a conclusion that contains information which is not contained within the premise. Mental disorder concepts would be projectible if they supported inferences about, say, future unobserved instances.
2. Note that one must be committed to "normal mental function" being a natural kind for the account to work. However, the details of what precisely "normal mental function" amounts to can be specified in different ways, provided the cluster of behaviors associated with the category cluster together reliably.
3. The illustration involves a simplification. We are aware that duration of a depressive episode is also used as a treatment filter—those that remit spontaneously are screened out of treatment. The DSM wants to pick out more chronic conditions that are worth treating.

References

Andreasen, N. (2000). Schizophrenia: the fundamental questions. *Brain Research Reviews*, **31**, 106–12.

Beebee, H., and Sabbarton-Leary, N. (eds) (2010). *The Semantics and Metaphysics of Natural Kinds*. London, UK: Routledge.

Bolton, D. (2008). *What is Mental Disorder: an essay in philosophy, science, and values*. Oxford, UK: Oxford University Press.

Boorse, C. (1975). On the distinction between disease and illness. *Philosophy and Public Affairs*, **5**, 49–68.

Bortolotti, L. (2013). Rationality and Sanity: The role of rationality judgements in understanding psychiatric disorders. In K. W. M. Fulford, M. Davies, G. Graham, et al. (eds), *Oxford Handbook of Philosophy and Psychiatry*. Oxford, UK: Oxford University Press.

Broome, M. R., and Fusar-Poli, P. (2012). Philosophical Issues in the Prodromal Phase of Psychosis. *Current Pharmaceutical Design*, **18**(4), 596–605.

Broome, M. R., Wuyts, P., and Fusar Poli, P. (2013). Philosophical and Ethical Issues in the Prodromal Phase of Psychosis. In K. W. M. Fulford, M. Davies, G. Graham, J. Sadler, G. Stanghellini, and T. Thornton (eds), *Oxford Handbook of Philosophy and Psychiatry*. Oxford UK: Oxford University Press.

Canguilhem, G. (2004). The Normal and the Pathological—Introduction to the Problem. In A. L. Caplan, J. McCartney, and D. A. Sisti (eds), *Health, Disease and Illness: Concepts in Medicine*. Georgetown University Press.

Caplan, A. L., McCartney, J., and Sisti, D. A. (eds) (2004). *Health, Disease and Illness: Concepts in Medicine*. Georgetown University Press.

Cooper, R. (2007). *Psychiatry and Philosophy of Science.* Stocksfield, UK: Acumen.

Cooper, R. (2009). Is psychiatric research scientific? In M. R. Broome and L. Bortolotti (eds), *Psychiatry as Cognitive Neuroscience*. Oxford, UK: Oxford University Press.

Engel, G. L. (1997). The need for a new medical model: a challenge for biomedicine. In *Science*, Vol. **196**, No. 4286. (Apr. 8, 1977), pp. 129-136.

Gensure, R. C., Mäkitie, O., Barclay, C., et al. (2005). A novel *COL1A1* mutation in infantile cortical hyperostosis (Caffey disease) expands the spectrum of collagen-related disorder. *The Journal of Clinical Investigation*, **115**(5), 1250–7.

Goodman, N. (1984). *Of Mind and Other Matters*. Cambridge, MA: Harvard University Press.

Grob, G. (1991). Origins of DSM-I: A study in appearance and reality. *American Journal for Psychiatry*, **148(4)**, 421–31.

Hall, C. (2005). Caffey Disease. Available at: Orphanet—<https://www.orpha.net/data/patho/Pro/en/Caffey-FRenPro431.pdf> (accessed 01/06/2013).

Horwitz, A., and Wakefield, J. (2007). *The Loss of Sadness*. New York, NY: Oxford University Press.

Killcross, S., Robbins, T. W., and Everitt, B. J. (1997). Different types of fear-conditioned behaviour mediated by separate nuclei within amygdala. *Nature*, **388**(6640), 377–80.

Klüver, H., and Bucy, P. (1939). Preliminary analysis of function of the temporal lobe in monkeys. *Archives of Neurology*, **42**, 979–1000.

Murphy, D. (2006). *Psychiatry in the Scientific Image*. Cambridge, MA: MIT Press.

Murphy, D. (2009). Psychiatry and the concept of disease as pathology. In M. R. Broome and L. Bortolotti (eds), *Psychiatry as Cognitive Neuroscience*. Oxford, UK: Oxford University Press.

Murray, G. K., and Jones, P. B. (2012). Psychotic symptoms in young people without psychotic illness: mechanisms and meaning. *British Journal of Psychiatry*, **201**, 4–6.

Pearce, J. M. S. (2012). Brain Disease Leading to Mental Illness: A Concept Initiated by the Discovery of General Paralysis of the Insane. *European Neurology*, Vol. **67**, No. 5, 272-278.

Pickard, H. (2009). Mental illness is indeed a myth. In M. R. Broome and L. Bortolotti (eds), *Psychiatry as Cognitive Neuroscience*. Oxford, UK: Oxford University Press.

Sorensen, R. (2011). Para-natural Kinds. In J. K. Campbell, M. O'Rourke, and M. H. Slater (eds), *Carving Nature at Its Joints: Natural Kinds in Metaphysics and Science*. Cambridge, MA: MIT Press.

Szasz, T. (1961). The myth of mental illness. *American Psychologist*, **15**, 113–18.

Wakefield, J. (1992). The concept of mental disorder: on the boundary between biological facts and social values. *American Psychologist*, **47**, 373–88.

Chapter 6

The background assumptions of measurement practices in psychological assessment and psychiatric diagnosis

Jared W. Keeley

6.1 Introduction

The validity of mental health diagnoses has been questioned long before the systems were formalized into official documents like the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) of the American Psychiatric Association or the *International Classification of Diseases* (ICD) of the World Health Organization. For example, Hippocrates' humoral account of mental illness was contested by the contemporaneous Cnidian school, which argued mental illness came from external disease agents rather than a constitutional imbalance (Weckowicz and Liebel-Weckowicz 1990). The debate arose from a difference in the two schools' fundamental ontological assumptions, which translated into disagreements about appropriate medical diagnosis and intervention. Even in its relatively short 200-year history (Lewis 1941; Menninger et al. 1963), psychiatry has gone through multiple periods of severe criticism of the validity of its diagnostic concepts, each roughly coinciding with a major shift in the process and procedure of diagnosis (Hempel 1964; Houts 2000; Menninger et al. 1963; Szasz 1961). The ontological assumptions inherent in the procedures of diagnosis can have a profound impact upon the validation of those diagnoses.

With the advent of new editions of both DSM and ICD, the time is ripe to consider the implications of the validity of the concepts included in these manuals. While other chapters in this volume address the finer points of changing models of validation in the context of psychiatric diagnosis, this chapter will focus on some of the background epistemic values and ontological assumptions present in psychological assessment and the process of diagnosing. Assessment is critical to understanding *diagnostic validity*, because it is through the measurement process, both in research and in clinical work, that information comes to bear for providing evidence for the validation of a diagnostic concept or the basis of a diagnostic

decision. However, the assumptions of the measurement process can—in no small part—reciprocally shape the structure of the diagnostic entity. Certain assumptions restrict (or at least make more likely) certain diagnostic structures. Further, these measurement assumptions do not exist in a vacuum. Rather, they are partially determined by the decisions made by human beings, either acting as clinicians or researchers. Thus, some of the factors of human cognition undergird the measurement assumptions and exert an additional indirect influence upon the validity of diagnostic constructs. Diagnosis is a practical endeavor. The clinicians that use these diagnostic constructs are subject to quirks and biases of human cognition that limit the validity of different kinds of diagnostic constructs.

This chapter will begin by outlining some of the influences human cognition might exert upon measurement concepts in the process of diagnosis. These influences form an ontological loop that reifies some of the assumptions made by the developers of psychological assessment methods and those clinicians that use them. The chapter will then progress to discuss some of the key assumptions made in diagnostic assessment practices and how those impact diagnostic validation.

6.2 The Interaction of Diagnostic Process and Measurement

Diagnostic validity involves the process of diagnosis as much as it does the ontological assumptions surrounding the underlying concepts. Both the process and ontology include a variety of epistemological assumptions about how we know anything and the optimal way to come to know anything. Ideally, a thorough deconstruction and examination of those processes could help elucidate problems or missteps in diagnostic validation as those constructs and measurements are initially developed. If simply understanding the assumptions were enough to develop and maintain *diagnostic validity*, then the problems of the field would have been solved long ago. There are additional processes that influence clinicians and researchers regarding diagnostic validation. Specifically, the cognitive processes involved in making a diagnosis (in a practical, day-to-day manner) also impact assumptions made about the underlying measurement properties.

Even for clinicians who eschew formal diagnosis on theoretical grounds, some level of diagnostic process is fundamental to all clinical acts. Without understanding the nature of the person's presenting complaint (i.e., an assessment), it is impossible to make determinations about a course of clinical action (unless one literally assumes all people and all problems are the same and provides a "one size fits all" approach to treatment). Thus, the way in which a clinician conceptualizes the information provided by an assessment has implications for

later clinical decisions. Just as there are a number of ontological and epistemological assumptions underlying the measurement process, similar assumptions affect the clinician's decision-making process.

For example, a clinician's assumptions about the ontology of mental disorders will shape what information he or she gathers in an assessment. If a clinician believes that psychiatric symptoms are fundamentally different than normal processes, he or she will look for discrete or categorical demarcations between them. In contrast, a clinician who believes psychiatric symptoms vary continuously in the population will ascribe to a dimensional model of psychopathology. These two clinicians are more likely to adopt assessment strategies that are compatible with their underlying assumptions and conceptualizations. Thus, the first clinician may be more comfortable with a checklist of the presence or absence of symptoms. The second clinician might be more likely to include self-report inventories that provide dimensional scores referenced against a normative population. The irony here is that the choice of assessment measure provides information that reinforces the clinician's initial assumptions. For example, by constantly seeing the dimensionality of a client's information, the second clinician is further convinced that his or her assumptions have been validated. However, both categorical and dimensional assumptions can be applied to every client. Of course, the process being described here is *reification*, which has long been recognized in the mental health literature as a problem for clinical science and practice (Hyman 2010).

Mental health diagnosis is not unique; reification purveys all of human experience. The process of a clinician (or scientist) reifying a diagnostic concept has its roots in cognitive-perceptual processes. Experience shapes perception in a top-down fashion, whereby cognitive knowledge structures and expectations influence the way in which information is received and processed (Kveraga et al. 2007).

What aspects of clinicians' experience are most relevant to shaping how they perceive disorders? I will divide these influences into two domains: cognitive processes common to all human beings, and processes specific to the education of mental health professionals. I hold no illusion that these are the only two influences, or that they are mutually exclusive in any meaningful way. I use the distinction merely as a contrivance for elucidating their effects.

6.2.1 Common Features of Human Cognition

There are some inherent communalities to the way human beings operate generally that are relevant to the process of diagnosis. First, human beings tend to organize their world in categorical, hierarchical ways. A variety of work from anthropology (e.g., Atran 1990; Berlin 1992) and cognitive psychology (Coley et al. 2004; Deneault et al. 2005; Johnson and Mervis 1997; Lopez et al. 1997;

Medin et al. 1997; Shafto and Coley 2003) has found that human beings organize the living world in very particular ways. Specifically, they organize living things into groups (categories) that reflect meaningful perceptual or pragmatic properties of the group. These groups are organized in a hierarchical fashion, such that smaller groups (e.g., dogs) are placed within larger groups (e.g., mammals).

Interestingly, categorizing things into groups seems to be the way children learn these concepts, and their capacity for understanding the world in categorical terms mimics the inherent way in which human beings assimilate language in those early years. Thus, it is not surprising that children grow up to organize other aspects of their experience in similar ways (e.g., where things are in the grocery store or mental disorders). These sorts of categorical hierarchies seem to serve the function of making access to information easier and faster by virtue of simplifying the wide array of information present in the world (Biederman et al. 1999; Rosch et al. 1976). Flanagan and Blashfield (2007) have applied this notion of "folk taxonomies" to how mental health professionals organize mental disorders, and indeed, clinicians follow many of the same "natural" patterns that arise from common human mechanisms for organizing objects and constructs in the world.

If there is something "natural" or inherent to utilizing categorical organizations (regardless of whether it is genetically or culturally driven), then it stands to reason that clinicians—being human beings—will default to organizing mental disorders in categories. Thus, an intuitive understanding of non-categorical models of mental disorder (regardless of their scientific validity) may be more difficult to acquire. Indeed, a preponderance of evidence suggests that many (if not most) mental disorder concepts should reasonably be considered dimensional at a latent level (Eaton et al. 2011; Haslam et al. 2012; Wright et al. 2013). The point, rather, is that using dimensional organizational structures for mental disorders, even if they better reflect the scientific validity of the concept, may be an uphill battle for human cognition. Preserving all the additional information of a dimension may overload clinicians' cognitive capacities, such that they will implicitly (or even explicitly) try to simplify the amount of information for easier storage and use.

A second factor affecting the diagnostic process for clinicians is *overconfidence bias*. Again, the overconfidence bias is not something unique to mental health; rather, it is common to human cognition in nearly all settings (West and Stanovich 1997). In essence, after people make a judgment, they tend to be more confident in that judgment than their accuracy would justify. In other words, people are often wrong, but they nearly always believe they are right once they have committed to a decision. Clinicians are the same after making diagnoses. Reliability statistics show that clinicians, in real world settings, can have

relatively low agreement on their diagnoses (especially for common conditions like Major Depressive Disorder; Regier et al. 2013). However, individual clinicians continue to be relatively confident in their diagnoses (Smith and Dumont 2002). Thus, their overconfidence in the correctness of their diagnosis may lead clinicians to ignore disconfirming evidence, further reifying their understanding of the diagnostic construct and how they assess it.

6.2.2 Education and Reification

It is reasonable to assume that clinicians' extensive training has an impact on the way they think. Indeed, a common definition of learning is an enduring change in cognition or behavior (Barker 2001). The educational process for mental health professionals has many goals, including gaining an understanding of the variety and forms of psychopathology, developing skills in case conceptualization, and learning how to formulate a treatment plan (e.g., see the "cube model" for a representation of educational goals for psychologists; Fouad et al. 2009; Rodolfa et al. 2005). Presumably, the educational process results in greater knowledge about these topics, although the various mental health disciplines are relatively unsystematic in demonstrating that they have met these goals. Nonetheless, if a clinician has not been exposed to a way of thinking about diagnosis (e.g., never been exposed to dimensional models of psychopathology), it stands to reason that it will be less likely the clinician would implement such a model in his or her practice. Thus, the educational system might place limits or boundaries upon the range of responses a clinician can utilize. If they are to expand upon those boundaries, it will have to occur after completing a degree through continuing education or self-study.

Most educational systems provide some level of exposure to formal medical nosologies such as the DSM or ICD. One purpose of formalizing a classification system is to standardize the language used by professionals (Keeley et al. in press). Thus, indoctrination into the field requires some exposure to the common language of that field, including diagnostic terms like "Major Depressive Disorder" and "Schizophrenia." The structure of diagnoses in these manuals tends to follow a set of underlying ontological assumptions (Sadler 2005). First, the DSM and ICD operate from the medical model, where the disorder is an imperfect pattern of common signs and symptoms that represents a (sometimes presumed) pathological process. The idea of a syndrome becomes inherent to some degree for individuals' representations of disorders when trained with these manuals. They then organize their understanding of psychopathology around these patterns of behavior in the overall landscape of all mental health symptoms.

Exposure to this model begins the process of reification. If nothing else, the DSM and ICD serve as a baseline against which alternative models of psychopathology are compared. Clinicians likely use DSM and ICD to structure

their understanding of psychopathology when they start to see actual patients. Thus, rather than forming a bottom-up understanding of symptoms and their arrangement, students start with top-down concepts that they "discover" in their patients. The educational process, by virtue of coming first, inherently places some arrangement on clinicians' perceptions. To be clear, I am not stating that the alternative of having each clinician construct a bottom-up classificatory system for psychopathology based upon individual experience is desirable. Such a state of affairs would create mass confusion in the field, as evidenced by the state of mental health services prior to DSM-I (see Grob 1991 and Houts 2000 for commentary). Rather, I am claiming that there will be some *a priori* biases to clinicians' views by virtue of having been trained to be a clinician.

Nevertheless, clinicians' experience beyond their education seems to have an impact on their understanding of diagnoses. For example, a series of studies examining how mental health professionals would organize a classification of mental disorders have shown that clinicians do reproduce some familiar aspects of the DSM or ICD. However, they also show unique (and consistent) features that are not present in the DSM or ICD (Flanagan et al. 2008; Reed et al. 2013; Roberts et al. 2012). For example, clinicians do not preserve personality disorders as a coherent group. Rather, they tend to spread those disorders throughout the classification based upon the disorder's phenomenology (e.g., Avoidant Personality Disorder is placed with anxiety disorders, Schizoid Personality Disorder is placed with psychotic disorders, Borderline Personality Disorder is placed with mood disorders; Flanagan and Blashfield 2006).

6.3 Education in Measurement and Assessment Basics

Clinicians who undergo psychological or academic psychiatric training are exposed to a variety of information about measurement. These experiences also shape their decisions about diagnosis and assessment. This information will form a necessary backbone for investigating the underlying ontological assumptions and epistemic values that are part of *diagnostic validity*. From a practical standpoint, the process of diagnosis requires an initial period of information-gathering (a.k.a., an assessment). That assessment, whether explicitly called such or not, is inherently a measurement process. Information is gathered, and that information has particular properties.

Common practices for diagnostic assessment vary widely across mental health disciplines and professionals. Some ways of gathering information include diagnostic interviews, clinician-administered instruments, self-report questionnaires, neuropsychological tests (often performance based), projective tests, and many others. Other tests, although not commonly used, might have potential as diagnostic

information, including neuroimaging scans, genetic testing, observation of the person's social interactions or occupational performance, etc. Depending upon the presenting complaint of the person, a mental health professional might use any or all of these sources of information. Regardless of the source, the information could be classified in two kinds: qualitative versus quantitative information.

Qualitative information retains its initial value only (usually verbal language, although it could be in another form, like a drawn picture or a facial expression) and—unlike quantitative information—is not assigned a numeric value. There is nothing inherent about the response that requires it to be qualitative or quantitative. Assessment responses traditionally treated as qualitative could be assigned a value—usually that process is just considered too difficult or burdensome to be useful.

Quantitative information, on the other hand, is codified and assigned a numerical value. Quantitative responses may take on any of a set of values, and thus are appropriately termed "variables." Variables may take one of four measurement scales: nominal, ordinal, interval, or ratio. Each measurement scale represents a different level of assumption about the kind of information portrayed by the numerical value. Nominal values provide no other information than group membership, such as 1 = Canadian, 2 = American, and 3 = Mexican. In a true nominal scale, there are no justifiable values in between the discrete groups. An ordinal scale adds a comparative value to the groups and ranks them. However, with an ordinal scale, the relative distance between the groups is unknown. The distance between 1 and 2 might or might not be the same as the distance between 2 and 3. Interval and ratio scales preserve the spacing between units, such that the movement from 1 to 2 to 3 on the scale represents equal amounts of difference. The difference between the two is that ratio scales have a true zero point, meaning that meaningful ratios can be computed from the numbers.

Another important distinction that will be necessary for some of the discussion to follow is the difference between an indicator of a diagnostic concept and the concept itself. Under the DSM and ICD systems, individual symptoms and signs are taken as imperfect indicators of a disorder. The "reality" of that disorder may take on more or less meaning depending upon one's ontological stance about measurement. The range of stances goes all the way from a belief that diagnoses correspond to real entities that are discoverable and even locatable, to a belief that they are instruments that serve a variety of practical purposes (Zachar and Kendler 2007). Regardless of the level of meaning one places on the disorder, the syndrome represents a conglomeration of symptoms and signs that seem to go together. It is important not to conflate any individual indicator (e.g., depressed mood) with the diagnostic construct (e.g., Major Depressive Disorder).

Further, the structure of the disorder is—in part—assumed, but it can also be subjected to empirical investigation, assuming at least two competing models

can be identified. The most common competing models (although not the only two) are categorical and dimensional approaches (Widiger and Samuel 2005). Categorical models assume that the diagnostic construct refers to a distinct group in a population, whereas dimensional models assume the disorder maintains continuous covariation among all individuals in the population. There are a number of models that exist between these two extremes (see Haslam 2002). A variety of data analytic strategies including taxometrics (Meehl 1995; Ruscio et al. 2006) and facture mixture analysis (Muthen 2006; Muthen and Muthen 2010) have been developed to test these alternative models.

6.4 Scientists are People, Too

All the factors discussed thus far regarding how clinicians' perceptual and cognitive processes shape their diagnostic decisions also apply to the researchers who undergird the mental health field. The scientists who develop assessment measures or who investigate psychopathological constructs are subject to the same biases. As such, these individuals' assumptions about the structure, nature, and cause of mental disorders may affect the measures they create and the constructs they investigate. If a scientist follows a belief in the inherent categorical nature of mental disorders, then his or her measurement choices in constructing an assessment tool could reflect that ontological assumption. The scientific investigations that studied the ontological structure of the construct would be all but forced to find support for its categorical nature if they used that measure.

Similarly, researchers may adopt a measurement technology that presumes a specific ontology (like a yes or no question, discussed further in section 6.5). In that case, they may unintentionally adopt an ontological stance that shapes the diagnostic construct. Whether the researcher intentionally adopts a certain kind of measurement due to his ontological beliefs or simply selects a technology that does so for him, the measurement influences upon *diagnostic validity* form an ontological loop with those that create them, perpetuating ontological and epistemological assumptions about mental disorder. The process of diagnosis is reciprocally influenced by (and influences) the validation of the concept it is designed to assess. To borrow a quote from Wittgenstein, "Show me how you are searching and I will tell you what you are looking for" (1975, p. 67).

6.5 Assessment Options and Their Measurement Characteristics

The following sections will address two of the more common assessment styles and the ontological assumptions of each. The measurement choices employed in an assessment strategy simultaneously structure what sort of information is available and define what kind of diagnostic constructs they can detect.

6.5.1 Diagnostic Interviews

A common method of gathering diagnostic information is a clinical interview. The nature of that interview can vary from an unstructured format where the clinician chooses the topics and questions based upon the quality of the interaction, to structured interviews where the wording, order, and scope of the questions are set. Arguments for the use of unstructured interviews tend to highlight the importance of qualitative information not otherwise gathered in structured interviews (Segal et al. 2012), particularly for the purpose of treatment planning or rapport building (Segal et al. 2008). These arguments state that the process of quantifying the interaction in an interview inherently loses information. Thus, they ontologically privilege the quality of information that is not typically preserved in quantitative codes (such as emotional tone, or the interviewers' emotional reaction; Churchill 2006). However, an unstructured interview does not presume any particular ontological structures for the diagnostic constructs it investigates.

One criticism of unstructured interviews is that individual clinicians might inadvertently miss a topic area due to theoretical dispositions, overshadowing by more florid symptoms, or other factors (Segal et al. 2012). Similarly, the way in which a question is phrased might pull for particular responses from the interviewee (Rogers 2001). For example, asking, "How bad is your depression?" might pull for a more negative and thereby pathological response from an interviewee than, "How is your depression?" Therefore, by setting the wording, ordering, and number of questions, structured interviews purportedly create a more reliable diagnostic outcome.

Structured interviews also contain a number of ontological assumptions. The first regards its domain of content. In being designed to ensure complete coverage of a topic area, the authors of the interview assume that their definition of the boundary (and thereby also the components) of the domain is complete. For example, the *Anxiety Disorders Interview Schedule for DSM-IV* (ADIS-IV; Brown et al. 1994) is designed to be a comprehensive examination of DSM-IV anxiety disorders. By measuring DSM-IV disorders, the validity of their interview is inherently limited by the validity of the diagnostic choices made by those individuals who developed the DSM-IV. Whether an interview is based upon the DSM or some other scheme, the validity of the measurement is inherently based upon the choices the researcher made when defining those boundaries. The biases and assumptions guiding the individuals who define the diagnostic construct are perpetuated by a researcher adopting that construct for the domain of an assessment instrument.

A second ontologically loaded assumption of structured interviews is contained in the nominal or ordinal coding of the questions. Often, the response

to an inquiry about a particular symptom is "yes, it is present" or "no, it is not." This sort of information is consistent with a nominal, categorical definition of the symptom (although not necessarily with a categorical definition of the diagnostic construct, although the equivocation is often made). The consequence of using the yes/no response alternatives is that there are no (or negligible) options in between the two poles. The symptom is present or it is not, much in the same way a medical patient either has an abscess or does not. A partial abscess is an abscess.

Some psychiatric symptoms are readily amenable to that sort of measurement scheme. For example, a person either does or does not have hallucinations. "Partial hallucinations" are not given that name; rather they are labeled "unusual perceptual experiences" or "illusions," because the experience is considered qualitatively different. There might be additional, dimensional information about the severity or frequency of the hallucinations, but their presence (which is the information relevant for current diagnostic definitions) is categorical.

Some diagnostic interviews utilize in-between response options to address symptoms that are not as clear-cut. For example, the *Structured Clinical Interview for DSM-IV Axis I Disorders* (SCID-I; First et al. 2002) includes coding options for absent, below threshold, or present. Including a "below threshold" option allows for the measurement to capture in-between states where some of the symptom is present, but it has not yet reached diagnostic levels. For example, many individuals might experience depressed mood, but that mood might not be present "most of the day, more days than not," which is required for a Major Depressive Episode (APA 2013). Thus, the interview could capture the information that some level of the symptom was present, but differentiate it from a higher level required for diagnosis.

When an in-between coding option is available to interviewers, there is an implicit ontological implication that the symptom exists at varying levels. The three-point coding scale preserves the ordinal nature of distinctions (absent < sub-threshold < present); however, it loses information about the distance between the three points. Is a sub-threshold symptom closer to absent or to present? Just how frequent is the individual's depressed mood? In the case of these sorts of symptoms, a categorical/discrete measurement scale is less justifiable than cases where there is clear separability to the state.

In instances where there is not a clear demarcation in the nature of the symptom, a measurement scheme that divides the symptom must be based upon some convention. In other words, where on the continuous distribution of depressed mood does the interviewer decide that the level of mood is indicative of disorder? Often, these operational definitions are made explicit

as part of developing the interview schedule. Other times, the threshold is more implicit, or developed during standardization training. These sorts of decisions may be entirely justifiable on the basis of some value-statement, such as increased risk of harm (to self or others), impairment in important daily tasks or functions, or distress to the person. However, the category created by the cut-point is not natural, in that the measurement itself did not suggest where the line should be drawn. Rather, the cut-point is practical. Other authors (Haslam 2002; Zachar 2000) have termed this sort of category a "practical kind."

Structured interviews are not required to use nominal or ordinal measurement schemes; their inclusion is a reflection of the ontological assumptions of the authors creating them or the technology available to them. For example, an interviewer could rate the client's depression on a scale from 1 to 10—a dimensional measurement—instead of an ordinal set of thresholds. However, the symptoms, in-and-of themselves, rarely demonstrate a clear structure that obviously lends itself to one sort of measurement versus another. The choice of measurement probably reflects as much about the interview developer's ontological stance and purpose in creating the assessment as it does about the nature of the symptom. As discussed previously, some reciprocal process of reification probably plays a role in the author's assumptions about the nature of the symptoms and choice of how to measure them. The zeitgeist of the field also plays a role, in that cutting-edge assessment technology (like Item Response Theory) probably influences individuals' selection of measurement options. New technologies are developed to replace the problems of the old, and thus are preferred. Nevertheless, each measurement technology has its own implied ontology. Often the developer of an assessment method may not be aware of the implications of that method, and thus adopts unintended ontological boundaries.

Further, the best structure of the symptom (again, be it in dimensional or categorical terms) need not correspond to the structure of the diagnostic concept. Categorical measurement of symptoms could support a dimensional diagnostic concept, and dimensional assessment of symptoms could correspond to a diagnostic category. For example, if ten symptoms of a disorder were determined categorically (present/absent), the description of the disorder overall could still be dimensional (a score ranging from 0 to 10), with more symptoms indicating more severe cases. Vice versa, dimensionally assessed symptoms could be used to produce a categorical diagnosis, such as a taxon defined by individuals scoring at the high end of two or more dimensions. The best measurement scheme for the indicators of a disorder may be the same as or different than the assumed structure of the diagnostic construct. However, those choices are

often conflated, leading researchers and clinicians to assume the structure of a diagnostic construct given the measurement of its components.

6.5.2 Self-Report Questionnaires

Perhaps the second most common assessment method is a self-report questionnaire. There are two major types of self-report measures: (a) large inventories (e.g., Minnesota Multiphasic Personality Inventory 2 [MMPI-2]; Butcher et al. 1989) that assess a variety of constructs simultaneously, and (b) small scales designed to assess a single construct (e.g., Beck Depression Inventory-II [BDI-II]; Beck et al. 1996) or perhaps a few aspects of similarly related constructs. Both rest on some similar measurement assumptions, but they also incorporate some subtle differences.

In self-report questionnaires, each item is considered an imperfect indicator of the target construct (Embretson and Reise 2000). The combination of the items (because each represents an aspect of the construct) then creates a representation of the construct. Because each item is quantitatively measured, the sum or average of the numerical values of the items creates a continuous measurement scale (a.k.a., a dimension). The appropriate scaling (e.g., ordinal or interval) of that variable rests upon what ontological assumptions are made about the items. For example, if each item is considered to be a roughly equal indicator of the construct, then summing the items creates a scale with equal units. Classical test theory tends to assume items are equal indicators (Nunnally and Bernstein 1994), but they need not be. Item response theory is more flexible by allowing items to be weighted. However, allowing the indicators of the construct to be unequal requires additional assumptions before the scale can be considered interval (Embretson and Reise 2000). Averaging items for a total scale score necessitates at least interval data. Thus, any scale that uses an averaged score assumes interval properties for its items—that assumption may be justified or unjustified. If the items are treated as unequal, and their aggregate as an ordinal scale, then the only mathematically justifiable combination of those items is a sum. However, the danger of a summed score is to assume it is interval, because there is a wide range of possible values.

Patterning the previous discussion of interviews, the individual items of the scale may or may not have the same measurement scale as the overall diagnostic concept. Some inventories use dichotomous response options, like true/false or present/absent. Perhaps the best-known user of this response style is the Minnesota Multiphasic Personality Inventory (MMPI). Each item is presented in a true/false format, where the individual must make a judgment about whether that characteristic is true of him- or herself. Thus, each indicator is

assumed to follow a categorical present/absent structure. This ontological assumption may not be representative of the nature of the symptom (i.e., there is true variance in the severity between presence and absence) and the nature of the respondent's decision-making process (Morey 1996). For this reason, many self-report questionnaires, like the Personality Assessment Inventory (PAI; Morey 1996), include a Likert-style range of responses in which indicators are scored as 0, 1, 2, or 3.

For the MMPI, on some questions people earn one point by agreeing with the item and on others by disagreeing with the item. All the item scores are summed to create a dimensional scale of the variable in question. For example, Scale 2 (Depression) ranges from 0 to 57 in its raw responses, which are then converted into standardized, norm-referenced scores. The fact that these scores are converted to a standardized distribution with a known mean and standard deviation implies interval assumptions about the scale. Scores form a continuous (if not entirely normal) distribution across the population. This conceptualization of depression then assumes a dimensional structure to the construct, and further assumes that each item is an equal indicator of the construct (which is likely not true; see Aggen et al. 2005).

Even though the scales of the MMPI assume dimensional structures, they also employ cut-points, making them practical kinds as described earlier. Based upon the normative likelihood of individuals endorsing a similar number of items, the authors of the test assign a cut-point. Individuals above the cut-point are interpreted to have problems significant enough to warrant intervention, where those below do not. In other words, the dimensional scheme is converted into a category for practical purposes like the decision to initiate treatment. That said, it is a practical decision, and the line could easily be drawn in another place with similar results. Interestingly, the scales are not always used in a purely categorical way by clinicians. If an individual's score falls close to the cut-off, the clinician reserves the right of "clinical judgment" to interpret the meaning of that score in its context, and may consider it strong enough to indicate treatment. Similarly, the dimensional nature of the scale continues to be interpreted as an indicator of severity. In other words, individuals with scores over the cut-point are sorted based upon the relative degree of how high their scores are, and that relative information may be used to inform the clinical picture and intensity of intervention.

Finally, there is an important ontological distinction to be made between multidimensional scales and unidimensional scales. Multidimensional scales assume that constructs have multiple components (e.g., like verbal and non-verbal intelligence for IQ). Unidimensional scales assume constructs are homogeneous. Multidimensional scales have an extra hurdle in establishing their validity for a purpose (like diagnosis) because measurement error can

be attributed to multiple sources. If the author of the scale assumes the ontological structure of the construct is multifaceted, and thereby includes multiple components in the scale, the total scale which combines those elements may be flawed if any one of those components is not functioning adequately. Take, for example, a measure of depression which includes cognitive, affective, and physiological components in its definition. If the assessment of the physiological components of the construct is unreliable or poorly measured, then the total scale is similarly compromised, even though it might be masked by the adequate functioning of the other two areas.

However, a unidimensional scale, which assumes a unitary structure to its construct, can be judged by its merits as if all measurement error pertains to the same construct. This sort of reasoning has led some measurement theorists to insist that it is only justifiable to construct measurements of unitary constructs (Smith and Zapolski 2009). In other words, we can only validate the meaning of one construct at a time. To validate a multidimensional construct, one must break it down into each of its components, to ensure that each component is functioning appropriately—hence, creating a series of unidimensional scales, in essence. This viewpoint reflects an eliminativist scientific realism whereby the constructs do not correspond to reality, but their components do.

However, this reductionistic argument could be taken a step further. Unidimensional scales are determined to be unidimensional based upon factor analysis, i.e., all of the individual items on the scale are correlated to each other. However, it is never the case that each item is equally correlated to the assumed latent construct. Some items are better indicators than others. If the reductionist argument is taken to its extreme, we must validate individual items. Indeed, that is the approach taken within Item Response Theory (Embretson and Reise 2000), where each item is evaluated based upon its measurement characteristics. However, an instrument could go through a process of validation and yet not be valid because of its ontological assumptions. This reductionistic sort of argument contains an ontological assumption that the components of psychopathology can be broken into meaningful, separable elements, and the combination of those elements in an additive fashion creates the total meaning of the diagnostic concept. That sort of measurement strategy removes any interactive or causal effects among the elements of a diagnostic construct, such that its whole might be more than the sum of its parts (Keeley et al. 2013; Kim and Ahn 2002). By way of analogy, a reductionist argument would say the concept of clinical depression is no more than separable components of cognition, affect, and physiology. However, most theorists would easily agree that cognition effects affect, affect effects physiology, and so on, such that the interaction of the components is an important part of understanding the concept. Unidimensional

measurement strategies fail to capture those sorts of ontological assumptions. Thus, multidimensional measurement approaches have been developed and are beginning to be applied to this problem (Mok and Xu 2013; Wang et al. 2004).

6.6 Conclusion

The validity of diagnostic concepts cannot be disentangled from the way in which they are conceptualized and measured, i.e., the process of validation. There will never be a single or all-purpose "objective" truth to mental disorder concepts, because there are legitimate reasons to use them for different purposes. Sometimes more information is necessary, like using a continuous variable in research to predict a low base rate outcome like suicide, justifying a dimensional measurement of a construct. Other times, all that information is unnecessary, because a clinical decision is dichotomous (Do I hospitalize this patient or not?). In those situations, gathering extra-dimensional information would waste time and resources; hence a categorical measurement process is preferred. The same clinical phenomenon might be legitimately conceptualized differently in different contexts for different purposes. Thus, the validity of a diagnostic construct is pluralistic, and impossible to disentangle from the measurement and pragmatic contexts in which it is used.

The validity of diagnostic constructs is a reciprocal process involving many ontological, epistemological, and measurement-based assumptions. This chapter argues that the assumptions made in the measurement process, both in terms of constructing measures and using them practically in diagnosis, influence the assumptions made about the ontology of the diagnostic construct, and vice versa. The result is a situation where there will never be a single estimation of the validity of a diagnostic concept; rather, there will be many based upon the intended use and desired properties of the concept.

References

Aggen, S. H., Neale, M. C., and Kendler, K. S. (2005). DSM criteria for major depression: Evaluating symptom patterns using latent-trait item response models. *Psychological Medicine*, **35**, 475–87.

American Psychiatric Association (2013). *Diagnostic and statistical manual of mental disorders*, Fifth edition. Washington, DC: American Psychiatric Association.

Atran, S. (1990). *Cognitive foundations of natural history: Towards an anthropology of science*. Cambridge, England: Cambridge University Press.

Barker, L. (2001). *Learning and behavior: Biological, psychological, and sociocultural perspectives* (3rd edn). Upper Saddle River, NJ: Prentice-Hall.

Beck, A. T., Steer, R. A., and Brown, G. K. (1996). *Manual for the Beck Depression Inventory II*. San Antonio, TX: Psychological Corporation.

Berlin, B. (1992). *Ethnobiological classification*. Princeton, New Jersey: Princeton University Press.

Biederman, I., Subramaniam, S., Bar, M., et al. (1999). Subordinate-level object classification reexamined. *Psychological Research*, **62**, 131–53.

Brown, T. A., DiNardo, P. A., and Barlow, D. H. (1994). *Anxiety Disorders Interview Schedule for DSM-IV* (ADIS-IV). Albany, NY: Graywind Publications.

Butcher, J. N., Dahlstrom, W. G., Graham, J. R., et al. (1989). *The Minnesota Multiphasic Personality Inventory-2 (MMPI-2): Manual for administration and scoring*. Minneapolis, MN: University of Minnesota Press.

Churchill, S. (2006). Phenomenological analysis: Impression formation during a clinical assessment interview. In C. Fischer (ed.), *Qualitative research methods for psychologists: Introduction through empirical studies* (pp. 79–110). San Diego, CA: Elsevier Academic Press.

Coley, J., Hayes, B., Lawson, C., et al. (2004). Knowledge, expectations, and inductive reasoning within conceptual hierarchies. *Cognition*, **90**, 217–53.

Deneault, J., Ricard, M., and Rimouski, P. (2005). The effect of hierarchical levels of categories on children's deductive inferences about inclusion. *International Journal of Psychology*, **40**, 65–79.

Eaton, N. R., Krueger, R. F., South, S. C., et al. (2011). Contrasting prototypes and dimensions in the classification of personality pathology: Evidence that dimensions, but not prototypes, are robust. *Psychological Medicine*, **41**, 1151–63.

Embretson, S. E., and Reise, S. P. (2000). *Item response theory for psychologists*. Mahwah, NJ: Lawrence Erlbaum Associates.

First, M. B., Spitzer, R. L., Gibbon, M., et al. (2002). *Structured clinical interview for DSM-IV-TR Axis I Disorders, research version, patient edition*. New York: Biometrics Research, New York State Psychiatric Institute.

Flanagan, E. H., and 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., and Blashfield, R. K. (2007). Clinicians' folk taxonomies of mental disorders. *Philosophy, Psychiatry, and Psychology*, **14**, 249–69.

Flanagan, E. H., Keeley, J. W., and Blashfield, R. K. (2008). An alternative hierarchical organization of the mental disorders of DSM-IV. *Journal of Abnormal Psychology*, **117**, 693–8.

Fouad, N. A., Grus, C. L., Hatcher, R. L., et al. (2009). Competency benchmarks: A model for understanding and measuring competence in professional psychology across training levels. *Training and Education in Professional Psychology*, **3**, S5–S26.

Grob, G. (1991). Origins of DSM-I: A study in appearance and reality. *American Journal of Psychiatry*, **148**, 421–31.

Haslam, N. (2002). Kinds of kinds: A conceptual taxonomy of psychiatric categories. *Philosophy, Psychiatry, and Psychology*, **9**, 203–17.

Haslam, N., Holland, E., and Kuppens, P. (2012). Categories versus dimensions in personality and psychopathology: A quantitative review of taxometric research. *Psychological Medicine*, **42**, 903–20.

Hempel, C. (1964). Fundamentals of taxonomy. In *Aspects of scientific explanation and other essays in the philosophy of science*. New York: The Free Press.

Houts, A. (2000). Fifty years of psychiatric nomenclature: Reflections on the 1943 War Department Technical Bulletin, Medical 203. *Journal of Clinical Psychology*, **56**, 935–67.

Hyman, S. E. (2010). The diagnosis of mental disorders: The problem of reification. *Annual Review of Clinical Psychology*, **6**, 155–79.

Johnson, K., and Mervis, C. (1997). Effects of varying levels of expertise on the basic level of categorization. *Journal of Experimental Psychology: General*, **126**, 248–77.

Keeley, J., DeLao, C. S., and Kirk, C. L. (2013). The commutative property in comorbid diagnosis: Does A+B = B+A? *Clinical Psychological Science*, **1**, 16–29.

Keeley, J., Morton, H., and Blashfield, R. K. (in press). Classification. In P. Blaney, R. Krueger, and T. Millon (eds), *Oxford textbook of psychopathology* (3rd edn). New York: Oxford University Press.

Kim, N. S., and Ahn, W. (2002). Clinical psychologists' theory-based representations of mental disorders predict their diagnostic reasoning and memory. *Journal of Experimental Psychology: General*, **131**, 451–76.

Kveraga, K., Ghuman, A. S., and Bar, M. (2007). Top-down predictions in the cognitive brain. *Brain and Cognition*, **65**, 145–68.

Lewis, N. (1941). *A short history of psychiatric achievement with a forecast for the future*. New York: W. W. Norton & Company, Inc.

Lopez, A., Atran, S., Coley, J., et al. (1997). The tree of life: Universal and cultural features of folk-biological taxonomies and inductions. *Cognitive Psychology*, **32**, 251–95.

Medin, D., Lynch, E., Coley, J., et al. (1997). Categorization and reasoning among tree experts: Do all roads lead to Rome? *Cognitive Psychology*, **32**, 49–96.

Meehl, P. E. (1995). Bootstraps taxometrics: Solving the classification problem in psychopathology. *American Psychologist*, **50**, 266–75.

Menninger, K., Mayman, M., and Pruyser, P. (1963). *The vital balance: The life process in mental health and illness*. New York: The Viking Press.

Mok, M., and Xu, K. (2013). Using multidimensional Rasch to enhance measurement precision: Initial results from simulation and empirical studies. *Journal of Applied Measurement*, **14**, 27–43.

Morey, L. (1996). *An interpretive guide to the Personality Assessment Inventory (PAI)*. Lutz, Florida: Psychological Assessment Resources, Inc.

Muthen, B. (2006). Should substance use disorders be considered as categorical or dimensional? *Addiction*, **101**, 6–16.

Muthen, L. K., and Muthen, B. O. (2010). *Mplus User's Guide*, Sixth edition. Los Angeles, CA: Muthen & Muthen.

Nunnally, J. C., and Bernstein, I. H. (1994). *Psychometric theory*, Third edition. New York: McGraw-Hill.

Reed, G., Roberts, M., Keeley, J., et al. (2013). Mental health professionals' natural taxonomies of mental disorders: Implications for clinical utility of ICD-11 and DSM-5. *Journal of Clinical Psychology*, **69**, 1191–212.

Regier, D. A., Narrow, W. E., Clarke, D. E., et al. (2013). DSM-5 field trials in the United States and Canada, part II: Test–retest reliability of selected categorical diagnoses. *American Journal of Psychiatry*, **170**, 59–70.

Roberts, M., Reed, G., Medina-Mora, M., et al. (2012). A global clinicians' map of mental disorders to improve ICD-11. *International Review of Psychiatry*, **24**, 578–90.

Rodolfa, E. R., Bent, R. J., Eisman, E., et al. (2005). A cube model for competency development: Implications for psychology educators and regulators. *Professional Psychology: Research and Practice*, **36**, 347–54.

Rogers, R. (2001). *Handbook of diagnostic and structured interviewing*. New York: Guilford.

Rosch, E., Mervis, C., Gray, W., et al. (1976). Basic objects in natural categories. *Cognitive Psychology*, **8**, 382–439.

Ruscio, J., Haslam, N., and Ruscio, A. M. (2006). *Introduction to the taxometric method*. Mahwah, NJ: Erlbaum.

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

Segal, D. L., Maxfield, M., and Coolidge, F. L. (2008). Diagnostic interviewing. In M. Hersen and A. M. Gross (eds), *Handbook of clinical psychology, Volume 1: Adults* (pp. 371–94). Hoboken, NJ: John Wiley & Sons.

Segal, D. L., Mueller, A. E., and Coolidge, F. L. (2012). Structured and semistructured interviews for differential diagnosis: Fundamentals, applications, and essential features. In M. Hersen and D. C. Beidel (eds), *Adult Psychopathology and Diagnosis*, Sixth edition. Hoboken, New Jersey: John Wiley & Sons.

Shafto, P., and Coley, J. (2003). Development of categorization and reasoning in the natural world: Novices to experts, naïve similarity to ecological knowledge. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, **29**, 641–9.

Smith, G. T., and Zapolski, T. C. (2009). Construct validation of personality measures. In J. Butcher (ed.), *Oxford handbook of personality assessment* (pp. 81–98). New York: Oxford University Press.

Smith, J. D., and Dumont, F. (2002). Confidence in psychodiagnosis: What makes us so sure? *Clinical Psychology and Psychotherapy*, **9**, 292–8.

Szasz, T. (1961). *The myth of mental illness: Foundations of a theory of personal conduct*. New York: Hoeber-Harper.

Wang, W., Chen, P., and Cheng, Y. (2004). Improving measurement precision of test batteries using multidimensional item response models. *Psychological Methods*, **9**, 116–36.

Weckowicz, T., and Liebel-Weckowicz, H. (1990). *A history of great ideas in abnormal psychology*. Amsterdam, Holland: North-Holland.

West, R. F., and Stanovich, K. E. (1997). The domain specificity and generality of overconfidence: Individual differences in performance estimation bias. *Psychonomic Bulletin & Review*, **4**, 387–92.

Widiger, T. A., and Samuel, D. B. (2005). Diagnostic categories or dimensions: A question for DSM-V. *Journal of Abnormal Psychology*, **114**, 494–504.

Wittgenstein L. (1975). *Philosophical Investigations*. Oxford: Blackwell Publishing Ltd.

Wright, A. G., Krueger, R. F., Hobbs, M. J., et al. (2013). The structure of psychopathology: Toward an expanded quantitative empirical model. *Journal of Abnormal Psychology*, **122**, 281–94.

Zachar, P. (2000). Psychiatric disorders are not natural kinds. *Philosophy, Psychiatry, and Psychology*, **7**, 167–82.

Zachar, P., and Kendler, K. (2007). Psychiatric disorders: A conceptual taxonomy. *American Journal of Psychiatry*, **164**, 557–65.

Chapter 7

Neuroimaging in psychiatry: Epistemological considerations

Ivana S. Marková and German E. Berrios

7.1 Introduction

There is little doubt that the development of neuroimaging technologies has transformed understanding, research, and practice in medicine. The resolution power of MRI or fMRI enables the visualization of tissues, organs, and metabolic activity in fine detail and has resulted in clarification of anatomical structures and the biochemistry of physiological processes. Brain structure has been a particular focus of such research and, in the last decades, this has been extended to brain function. This, however, has raised new epistemological challenges. Whilst brain structure, whether at the level of organ, tissue, cell, or other, lends itself well to instrumental visualization, the capture of brain "function" demands additional conceptual justification. And, when it comes to "higher brain functions" or cognitive functions (such as working memory, problem solving, perception, etc.), neuroimaging techniques are beset by even more complex conceptual problems (Faux 2002; Uttal 2001, 2004). This notwithstanding, the claim by many that it is possible to capture or "localize" cognitive functions (e.g., Jezzard and Buxton 2006) has encouraged the application of these techniques to abstract concepts such as self-reflection (van der Meer et al. 2010), vocal deception (Spence et al. 2008a), self-knowledge (Ochsner et al. 2005), guilt/innocence (Spence et al. 2008b), morality (Decety and Cacioppo 2012), etc. and also to psychiatric symptoms and psychiatric disorders.

In recent years, therefore, there has been much research effort dedicated to the localization of psychiatric symptoms and disorders to brain structures and neuronal circuitry (e.g., Allen et al. 2008; Alvarenga et al. 2012; Brambilla et al. 2009; Drevets 2001; Hulshoff Pol et al. 2004; Shad et al. 2012; Tanskanen et al. 2010). Remarkably, however, relatively little interest has been directed at questions concerning the validity of such research enterprises, that is, at questions about whether and/or to what extent it makes sense to try to capture psychiatric objects (mental symptoms and mental disorders) using techniques designed to capture physical structure and physiological processes (e.g. Honey et al. 2002).

This is even more surprising given major disagreements about the definition, identification, and classification of mental symptoms and uncertainties about the "nature" of mental disorders and their neurobiological correlates. Indeed, the enthusiasm and rapidity with which the new technologies have been applied to psychiatric objects suggests that much has been invested in the hope that the high-level resolution power of these technologies will silence conceptual misgiving and provide answers to the problems of mental disorders and symptoms which so far have eluded the psychiatric discipline.

Setting aside for the moment the conceptual problems underlying such enthusiastic research drive, it is important to highlight at the outset a worrying consequence of it, namely, the desire to bestow on neuroimaging data a sort of wholesale validity that threatens to undermine the very ontology of the original "psychiatric" data. In other words, according to the "neurorealist" view (Racine et al. 2005), the very existence and legitimacy of mental symptoms and disorders is being made to depend upon their capture by neuroimaging. Taken to its extreme, it would follow that if patients' complaints do not correlate with the requisite brain circuitry, then such complaints would be deemed irrelevant or non-existent. The paragon of "valid" mental phenomena would thus be determined by what is captured by neuroimaging. In this new world, subjective experience and subjectivity in general would be relegated to being a trivial non-scientific discourse. A similar point has been made in relation to the neuroimaging of pain (see Hardcastle and Stewart 2009). The newly proposed Research Domain Criteria for the classification of psychiatric disorders (RDoC) exemplifies well this way of thinking. Taking a "precision medicine" approach, this project by the National Institute of Mental Health (NIMH) sets out a new method of classifying mental disorders based on: "levels of analysis progressing in one of two directions: upwards from measures of circuitry function to clinically relevant variation, or downwards to the genetic and molecular/cellular factors that ultimately influence such function" (Insel et al. 2010: 749). In other words, and in direct reversal of the conventional approach, the starting point in the exploration of mental disorders and symptoms becomes neurobiology or neural circuitry (Cuthbert and Insel 2013). In order to achieve a more "scientific" and objective classification of mental disorders, the authors suggest a transfer of the gold standard of validity from the psychiatric complaint to the "data captured by neuroimaging" and other "neuronal dysfunction" measures. Thus, data whose only claim to validity is that they correlate with real-life complaints are made to become now the gold "standard" against which the reality of complaints (by future patients) should be assessed and judged. It remains an important question as to what might be the epistemological arguments to warrant such devaluation of the patient's subjectivity.

The issue here seems to be that against a background of perceived lack of "scientific" progress in the understanding of psychiatric disorders, the advent of neuroimaging technologies has seemingly provided an alibi to transform a hypothesis into a law of nature. In other words, the desideratum that psychiatric disorders may only consist in disruptions of neurobiological systems (Cuthbert and Insel 2013) is suddenly converted into a foundational claim. This carries implications not only for decisions concerning what constitutes mental symptoms and mental disorders, and the ways in which these should be researched, but also for the very language with which these are expressed and understood.

It is not the aim of this chapter, however, to focus on the consequences of this particular approach to the exploration of mental symptoms and disorders. Ultimately this should be determined by its predictive validity and therapeutic usefulness for patients. Instead, this chapter wishes to address the question of the epistemological quality of the neuroimaging of psychiatric objects (mental symptoms and disorders), or to put it another way, to what extent psychiatric objects lend themselves to valid neuroimaging. This question can be examined from various standpoints, but here it will be restricted to one level of analysis, namely, to an exploration of the sorts of objects or structures psychiatric objects are and, in the light of this, whether it is possible for them to be mapped onto specific brain systems. The issue alluded to earlier, as to whether it makes *ethical* sense to convert the meanings that human beings and societies have developed to understand and communicate about themselves and the world into the language of neurobiology, is an important one, but demands a different level of analysis and will not be covered here.

Why an epistemological perspective? An epistemological approach examines the grounds on which knowledge in relation to things can be developed and understood. It lays out the presuppositions on which the knowledge is based and the conceptual framework within which it operates. This becomes particularly important in relation to psychiatric objects which are not easily locatable in standard classifications that include organic material (e.g., brain tissue, cells, etc.), functional processes (e.g., blood flow, glucose metabolism), functional events (e.g., sneezes), and abstract entities (e.g., virtues, selves). Yet, if any sense is to be made of research that attempts to correlate psychiatric objects with neurobiological objects, then it is imperative that the structure of psychiatric objects can be understood if not to the same level of sophistication, then at least to a clearer conception of their constitution (Marková and Berrios 2009, 2012). Only then can the validity of neuroimaging research in psychiatry be delineated. As indicated earlier, there has been relatively little interest taken in this question and most work questioning the use of neuroimaging in psychiatry has

tended to focus predominantly on methodological problems relating to the use of techniques such as scanning the environment, selecting appropriate cognitive tasks, and achieving statistical power (e.g., Kanaan and McGuire 2011).

The chapter considers the question of whether and/or to what extent psychiatric objects are structurally suited to the project of localization and neuroimaging. Focusing thus solely on the nature of psychiatric objects (mental symptoms), there is no space given here to the distorting effects generated by their transformation into proxy variables for mapping purposes. In other words, the whole issue of proxyhood, that is, how symptoms are actually represented for mapping purposes (e.g., scores on questionnaires), although important, will not be covered here. It goes without saying that issues concerning proxyhood play a significant part in epistemological considerations of neuroimaging research, since the representational quality of all the proxy variables entering into both sides of the correlation (complaint and brain) will be a determining factor of the validity of the results obtained. However, in order to highlight the importance of knowing something about the psychiatric objects entering into such correlations we will concentrate only on the role their *structures* play in trying to map them to brain processes.

Lastly, there will be no discussion of problems associated with the interpretation of neuroimaging data. This is a major area in itself and has been covered in detail elsewhere (e.g., Heeger and Ress 2002; Logothetis 2008; Price and Friston 2002; Sutton et al. 2009). The chapter is divided into two parts. The first examines the nature of psychiatric objects. For reasons of space, it will concentrate only on mental symptoms and will argue that these should be viewed as hybrid objects. The second will deal with the implications that the hybrid nature of psychiatric objects has on their putative brain inscription.

7.2 Mental Symptoms as Objects

7.2.1 The Structure of Mental Symptoms

The question as to what kind of objects are mental symptoms can be looked at from different perspectives. One can, for example, focus on whether mental symptoms represent changes (impairments or exaggerations) in ordinary mental functions or whether in fact they represent something quite different. Since the late nineteenth century this has been a debate that remains to be resolved (Berrios 1996), though much of neuroimaging research is based on the assumption that mental symptoms are expressions of ordinary mental functions that have become pathological (e.g., Halligan and David 2001). The focus here, however, will be on trying to determine what kind of structures mental symptoms might be, how they compare with other structures, and, in particular, what are

the features of structures that make them amenable to capture by instruments such as neuroimaging machines.

In general terms mental symptoms as currently listed are divided into subjective mental complaints, that is, those that are expressed by patients (e.g., low mood, interference with thinking), and "objective" signs/behaviors, that is, those that are observed by clinicians (e.g., psychomotor retardation, flight of ideas). For reasons of space, only subjective mental symptoms will be examined here.

Subjective mental complaints are those that are expressed by the patient, whether volunteered or elicited through questioning. They include, for example, depressed mood, anxiety, hearing voices, feelings of unreality/strangeness, experiencing thoughts being put inside one's head, feelings of anger, feeling that familiar people are strangers, being unable to move or talk, fatigue, experiencing the world as strange and people as persecutors, and many more. It is evident that these "symptoms" are heterogeneous, differing in many ways. Thus, some refer to familiar mental states, ones that most people can relate to (e.g., worries, irritability, low mood), whilst others refer to strange, alien experiences (e.g., feeling that messages are coming into one's stomach from a particular agency). Some relate to everyday events whilst others incorporate fantastical contents. Some are expressed directly as the "symptom" (e.g., anxiety, depression) whilst others are labeled as symptoms by the clinician on the basis of how they are expressed (e.g., delusions). Some seem to be feelings, others seem to be beliefs or perceptions, and others seem to be mixtures of many or all. Some might be volunteered freely whilst others are elicited with difficulty. Some are uttered easily and others might be expressed with hesitancy and uncertainty, and so on. Whilst they all fall within the current grouping of subjective mental symptoms, clearly they are very heterogeneous phenomena (Marková and Berrios 1995).

The question here is what sort of "objects" are they? What are they made up of? How can this be determined? One useful way of thinking about this is considering how they might arise. By definition, subjective mental states (whether "symptoms" or ordinary mental states) must refer to states about which people are aware. In other words, when people complain of low mood or a sense of unreality or hearing voices, then this is something that they are saying on the basis of their interpretation of some internal experience. The cause of this experiential change makes little difference at this point. It could be the result of an acute stressor or trauma, or ongoing pressures in one's life, or some form of brain condition, or indeed combinations of such factors in the context of genetic vulnerability and so on. The point is, however, that there must be some awareness of experiential change which at its earliest can be envisaged as an inchoate, pre-linguistic, pre-conceptual state (Berrios and Marková 2006).

How, then, does such an experiential change become converted into a subjective mental symptom?

It would seem likely that in order to make sense of this change and to articulate this, individuals will have to draw on a variety of sources. First of all there will be sources that relate to the individual and his/her sociocultural background. Here, factors such as past experiences, personality traits, personal biases and outlooks, levels of education, media influences, peer pressures, social contexts, language skills, and many more will all be important in shaping the experiential change into an articulated "symptom." For example, a history of past similar experiences or knowledge of others with what seem like similar experiences might facilitate interpretation of some states such as depressed mood or anxiety. A tendency to introspection might generate more detailed and colorful expressions of some experiences. The level of education or interest in reading might determine the range of vocabulary an individual has to describe what he/she is experiencing. The family, society, and culture in which the individual is brought up will help to structure and color the interpretation he/she makes of the internal state. Thus, in a society where it is frowned upon to express feelings explicitly, it might be more likely that an emotional experience is understood and described in cognitive terms. Or, a culture lacking in obvious ways of articulating emotional distress might encourage descriptions of specific experiences in somatic terms, such as fatigue, pain, etc. In other words, in the same way that individuals will report on an external event in different ways, individuals will likewise interpret and make sense of changes in their conscious states according to their personality and sociocultural background.

Second, factors around the development of the experiential change will play a part. Here, in the first place, the *rate* at which this change in conscious state occurs must be important. A change in an internal state that builds up slowly might draw on more sources such as memory, emotion, knowledge, etc. to make sense of this than an internal state that changes very rapidly. In the second place, the particular *context* in which it happens may also influence the way in which an individual will interpret this and understand it as an experience. In the third place, the *quality* of the change in conscious state will also play a part in how the internal state is interpreted. Something that is experienced as familiar might be more easily interpreted than something that is novel or alien, which might require effort to make sense of and need additional sources (e.g., cultural factors, imagination, etc.) to construct.

Third, in addition to these factors, there will be interactional forces that are also likely to be important in making sense of a particular internal state. Here, for example, the dialogical encounter may be vital in contributing to the shaping and articulation of the mental phenomenon. Thus, whether in communication with a clinician

or with someone else, a nebulous, initially strange experience that the patient may have difficulty in capturing might, through the encounter itself, become crystallized into a specific "symptom," as the mutual exchange may offer descriptions or meanings which resonate with the patient. Likewise, in some cases it might be that noticing a particular response in the interlocutor (e.g., the clinician may appear more interested or understanding in relation to certain terms) might encourage the use of a specific description by a patient which subsequently becomes fixed as a symptom. Similarly, in the interaction with the environment and context, sense may be "constructed" of a particular internal experience. Furthermore, whilst here, for the sake of analysis, examination is focused on how single symptoms might arise, in reality symptoms do not occur in isolation but alongside multiple other "symptoms" and variable mental state changes. Interaction, with whatever else is being experienced at the time, must also be important in shaping the description of the final symptom.

The question, then, is what does this all mean for understanding the structure of subjective mental symptoms? So far it would seem that there are many different kinds of factors or forces which will affect the way in which individuals will make sense of and articulate changes in their internal states. However, from this we can identify two basic elements. First, there has to be some core of neurobiology or brain signaling, since it goes without saying that all mental activity is underpinned by brain activity. Second, there is the "meaning" element, the product of all the different factors involved in shaping and formulating an experience. Thus we have a "biological" element and, what for the sake of brevity, can be called a "semantic" element, on the understanding that this refers to meaning in the broadest sense of the term as determined by the aforementioned forces relating to individual, sociocultural, and interactional factors. This can be represented by the following diagram (see Figure 7.1):

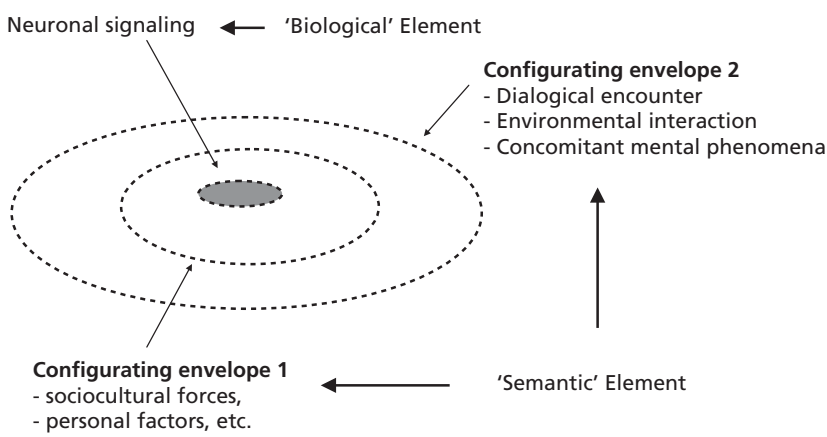

Fig. 7.1 Diagrammatic representation of structure of subjective mental symptoms

The figure is a schematic representation of the structure of a subjective mental symptom as constituted by a "biological" element and the "semantic" element. The latter is depicted in the form of two constructive "envelopes." The first one represents the configuration that occurs as a result of the individual and sociocultural forces (i.e., factors relating to personality, past experiences, education, personal biases, etc.). The second one represents the configuration that occurs as a result of interactional forces (i.e., through interaction with people, with the particular environment and/or context, and with concomitant mental experiences). What is evident from the process, though difficult to illustrate diagrammatically, is that the structure of the final subjective symptom must represent a product of the interaction between biological processes and the meaning that is configured on the basis of multitudinous factors.

7.2.2 Mental Symptoms as Hybrid Objects

The world is populated by all sorts of objects which, at different times, might become the subject of scientific enquiry. In general, objects may exist in time, in space, and in combinations of these. The question of what constitutes an "object" and how objects are classified has itself been an area of much debate (see Ferrater Mora 1991). For the purposes here, "object" simply refers to "a thing or being of which one thinks or has cognition, as correlative to the thinking or knowing subject; something external or regarded as external to the mind" (Oxford English Dictionary 1989). (It has to be understood that whilst subjective mental symptoms are by definition "mental," that is, within the mind so to speak, nevertheless, as "objects of enquiry" (e.g., as correlational variables of neuroimaging techniques) they become objects in a putative "external" space.) Defined in this way, objects have been classified into (i) physical or natural and (ii) abstract or ideal. Physical or natural objects refer to objects which exist in time and space such as houses, trees, clouds, brains, cells, atoms, etc. As such, providing there are the technologies available, they can be visualized at some level—whether this is by eye, by ruler, by microscope, or by any other instrument—and hence captured and measured accordingly.

Abstract or ideal objects refer to objects such as virtue, numbers, morality, beauty, etc. Unlike physical objects, these do not exist in space. They are constructs, that is, objects created by society and culture in order to help describe or explain aspects of life and the world. As they do not exist in space, such objects cannot be instrumentally visualized and captured. One cannot take a picture of e.g. morality or examine virtue with a microscope or X-ray tube. Such objects cannot be defined or measured in such physical ways. Individuals can relate to

the concept of beauty, but will find different things beautiful and/or beautiful to differing extents.

What about subjective mental symptoms, then? How do subjective mental symptoms compare with other "objects"? Unless one takes a position of extreme reductionism, materialism, or physicalism, then they don't seem to belong to the physical kinds of objects such as trees or atoms. Neither, however, can they be viewed as entirely abstract or ideal objects. Instead, as complexes of the biological (physical) and the "semantic" (constructed meanings), they seem to share features of both. In other words, they would appear to be *hybrid* objects. Hybrid refers to "anything derived from heterogeneous sources, or composed of different or incongruous elements" (OED 1989). Since subjective mental symptoms appear to be constituted by biological elements on the one hand, and "semantic" elements on the other, thus seemingly derived from heterogeneous sources and incorporating incongruous elements, then they must be considered as hybrid. The fundamental question that naturally arises from this conception of mental symptoms as hybrid is one that concerns the relationship between these two incongruous elements. How do the biological and "semantic" elements relate to each other?

7.2.3 Relationship Between the Biological and "semantic" in Symptom Structure

Vital for any brain localization project has to be some understanding of the relationship between the biological and the "semantic" elements constituting the mental symptom. Where there is a relatively direct relationship between the biological and "semantic," that is, where specific neuronal signaling is consistently correlated with a specific "meaning," then clearly this would give localization efforts some justification. However, the problem emerging from examining how certain mental symptoms might arise is that it cannot be assumed that the relationship between the biological and "semantic" is always that direct or that consistent.

We have seen that many different kinds of factors are likely to influence the interpretation of a particular internal state, and whilst such factors (sociocultural, individual, interactional, etc.) will themselves be underpinned by neurobiological activities, such configuration does preclude a specific and direct relationship between a "final mental symptom" and a particular neurobiological signal. Questions pertaining to the directedness and specificity of the "biological–'semantic'" relationship need dealing with because their answers will help with understanding: 1) the vexed problem of brain localization, 2) the structure of hybrid objects, and 3) the role that components play within each symptom, thereby determining the *sense* of the symptom as a whole.

The term "sense" is used here in this specific way in order to differentiate between the different aspects of meaning that are being referred to in this chapter. To restate, the term "semantic" refers to the meaning derived from one of the structural *constituents* of the mental symptom. Since, as has been shown, this is configured by diverse factors spanning individual, sociocultural, and interactional sources, the meaning contained in "semantic" is wide. In contrast, the term "sense" refers to the meaning of the symptom as a *whole* and specifically how, as a "research variable," the symptom can be understood and handled.

Given the heterogeneity of mental symptoms, it would seem likely that the nature (directedness) of the relationship between their biological and "semantic" components, and hence the role of each, will also be complex and variable. Some symptoms may have a relatively direct relationship between a specific biological signal and a specific associated meaning. In this case, the biological element may have the greater role and will carry the *sense* of the symptom. In other symptoms, this relationship is not direct on account of the influence of the configuring factors. Here the "semantic" element will have the greater role and will carry the *sense* of the symptom. It is the *sense* of the symptom that should determine the type of research approach taken to its study.

Elsewhere, it has been proposed that from a structural point of view, mental symptoms might usefully be divided into those which have primary brain inscriptions and those which have secondary brain inscriptions according to the directedness of the "biological–'semantic'" relationship (Berrios and Marková in press) (see Figure 7.2).

Mental symptoms with primary brain inscriptions are those which correspond in time and space with the brain activity that gives rise to them. Here there would appear to be a direct and specific relationship between the biological and the "semantic." For example, an ictal focus or brain lesion in a particular area might directly trigger organic hallucinations. Or, perhaps some

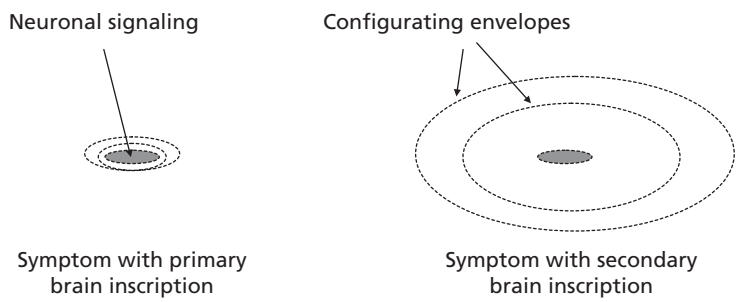

Fig. 7.2 Schematic representation of mental symptoms with primary and with secondary brain inscriptions

of those symptoms that are directly "observed" or captured by the clinician, such as flight of ideas or psychomotor retardation might more likely be associated with specific reproducible brain activity—as such symptoms will not have been subject to the sort of interpretative and constructive forces within the individual. (There would of course be other factors to take into account in these situations, such as the interpretative factors on the part of the clinician which may also serve to "distort" the ensuing symptom (Berrios and Marková 2002)—but this will not be examined here.) The *sense* of the symptom in these cases would be carried predominantly by the "biological" element and thus have less in the way of a meaningful connection for the individual. In other words, irrespective of how the symptom is expressed, it can be viewed as more stereotypical and relatively empty from personal significance.

On the other hand, mental symptoms with secondary brain inscriptions are those symptoms where the relationship between the biological and "semantic" is not direct. Here, the brain representations can be viewed as simply the concomitant neurobiology substratum. In other words, the biological is the non-specific brain activity that accompanies mental activity. In this case, the *sense* of the symptom is carried predominantly by its "semantic" element. Here, the meaning is important from the point of view of understanding its connection to the individual. This may or may not carry personal significance. For example, an individual in the context of depression and on the basis of a particular internal state might articulate a symptom of "guilt." Various sources will have been important in configuring it as such, including perhaps past experiences, personality factors, recent events, peer pressure, etc. Such factors within the individual and his/her sociocultural background will, by constructing the experience, serve to connect the individual with the symptom in a deep sense. When, in addition, the symptom carries a personal value, e.g., if the guilt refers to perceived failures in life as opposed to a non-specific feeling, then the connection becomes personally significant. The important issue, however, is that the connection to the individual may lead to the clarification of the symptom in terms of its "semantic" roots.

7.3 Implications for Neuroimaging of Subjective Mental Symptoms

7.3.1 Localization of Hybrid Objects

If subjective mental symptoms are considered hybrid objects, what, then, are the implications of this fact for their localization in the brain and hence for neuroimaging? We have shown that as hybrid objects, subjective mental symptoms consist of biological and "semantic" elements. We have also suggested that the

nature of the relationship between the biological and "semantic" determines the *sense* of the symptom, that is, the particular locus of the symptom that carries its meaning. Based on this, it was proposed that mental symptoms might usefully be divided into those with primary brain inscriptions and those with secondary brain inscriptions—on the understanding that mental symptoms are likely to fall within a range between these "prototypes." Brain localization involves the mapping of, in this case, a subjective mental symptom onto a specific neuronal structure or circuitry. The manner in which the biological and the "semantic" are related within each particular mental symptom thus becomes crucial to making possible and to understanding the brain localization endeavor.

In the case of mental symptoms with primary brain inscriptions, here it was argued that the relationship between the biological and "semantic" elements of the symptom was relatively direct and specific. Thus, picking up neurobiological activity can be said, relatively speaking, to be tantamount to capturing the *sense* of the symptom. In other words, from a theoretical perspective and without considering the other factors creating noise in this sort of correlational exploration, it could be argued that these types of mental symptoms might be amenable to brain localization projects.

In the case of mental symptoms with secondary brain inscriptions, here the *sense* of the symptom as a whole is carried by the "semantic" element of the symptom structure. The neurobiology, whilst present, does not have the direct and specific relationship with the "semantic" aspect of the symptom. It follows that the "same" neurobiological signal may be associated with a number of different symptoms—as individuals will configure their internal states differently according to the influences of the sorts of factors mentioned earlier, such as past personal experiences, sociocultural backgrounds, interactional effects, and so on. Thus, one patient might complain of low mood whilst another one talks of pain or anxiety or depersonalization, etc. Conversely, "different" neurobiological signals may be associated with the "same" mental symptom. The issue is that trying to map such mental symptoms onto specific neurobiology is fraught with problems. Sprevak (2011) argues for the importance of interactional effects between cognition and the contextual environment, neatly showing how neurobiology is not sufficient to explain specific cognitive processes. In relation to more complex mental phenomena, such interactions will be magnified in scope. Capturing the neurobiology, no matter how sophisticated the technology, may have little bearing on the meaning of the symptom itself. Since the "semantic" element of such symptoms carries the greater role, the *sense* of the symptom lies in the constructive forces that have played a part in its constitution. Research directed at understanding such symptoms should be aimed instead at developing new hermeneutical approaches which could seek

to disentangle such constructive forces. Determining any neurobiological correlates of such symptoms with neuroimaging technologies is unlikely to add to the understanding of these symptoms and could only have limited validity in terms of mapping the structures involved.

7.3.2 Neuroimaging of Mental Symptoms

Research aimed at the neuroimaging of mental symptoms must therefore consider very carefully the specific mental structures under enquiry. As was shown, it may be that those symptoms whose *sense* is carried by the biological element might more readily lend themselves to neuroimaging projects. Thus, in terms of such research, careful selection of symptoms would be imperative. In turn, identifying these will necessitate further research, both conceptual and empirical.

The recognition that from a structural perspective not all mental symptoms lend themselves to brain localization, and hence to neuroimaging research, carries wider implications. In the first place, it opens up new directions for the exploration and understanding of mental symptoms. As mentioned previously, consideration of the sorts of factors contributing to the "semantic" constituent of particular mental symptoms raises possibilities of developing different hermeneutical approaches for clarification of their nature as symptoms. In the second place, there are therapeutic implications in that the management of those symptoms whose *sense* is carried by the "semantic" element may need more than "biological" treatments to alleviate them fully. Thus research aimed at disentangling the meaningful constituents of such symptoms may lead to new ways of their management. In the third place, and from a wider perspective still, understanding symptoms as hybrid and their consequent relationship with neurobiological circuitry obviates the need for the assumption that all mental disorders are neurobiological disorders. This assumption, which is the driving force behind much of the neuroimaging work, is a major assumption and carries the potential of steering research in psychiatry into blind alleys (Berrios and Marková 2002). By contrast, highlighting the differential roles of the biological and the "semantic" in conveying the *sense* of mental symptoms not only draws attention to the different relationships between biological processes and meanings, but opens up the possibilities of examining these relationships from any causative direction (particularly relevant in relation to the question of psychogenesis).

7.4 Conclusions

Much research effort and funding is directed at neuroimaging in psychiatry, driven amongst other things by the successes obtained by such technologies in medicine as well as by the belief that mental disorders are neurobiological

disorders. It has been argued here that consideration must be given to both these claims to determine the validity of such research. First, psychiatry is a hybrid discipline, in a stronger and deeper sense than medicine. Its objects (mental symptoms and mental disorders) are likewise hybrid in structure, that is, they are made up of incongruous elements. Focusing here on subjective mental symptoms, such elements were identified as biological on the one hand and "semantic" on the other, the latter referring to the complex meaning that is constructed from the interaction of multitudinous factors including individual, sociocultural, and interactional. On account of this structure, mapping to brain structure and function becomes problematic and raises questions about the meaning of the results obtained. The validity of localization projects and thus neuroimaging research must depend on careful selection of "appropriate" mental symptoms.

Second, it has to be understood that the claim that mental disorders are neurobiological disorders is simply an assumption. It can only be considered trivially true in the sense that all mental states are underpinned by neurobiology. An epistemological clarification of how mental symptoms are structured and formed is crucial for determining valid approaches to both empirical research and clinical management.

References

Allen, P., Larøi, F., McGuire, P. K., et al. (2008). The hallucinating brain: a review of structural and functional neuroimaging studies of hallucinations. *Neuroscience and Biobehavioral Reviews*, **32**, 175–91.

Alvarenga, P. G., do Rosário, M. C., Batistuzzo, M. C., et al. (2012). Obsessive-compulsive symptom dimensions correlate to specific gray matter volumes in treatment-naïve patients. *Journal of Psychiatric Research*, **46**, 1635–42.

Berrios, G. E. (1996). *The History of Mental Symptoms*. Cambridge, UK: Cambridge University Press.

Berrios, G. E., and Marková, I. S. (2002). Conceptual issues. In H. D'haenen, J. A. den Boer, and P. Willner (eds), *Biological Psychiatry* (pp. 3–24). Chichester, UK: John Wiley & Sons, Ltd.

Berrios, G. E., and Marková, I. S. (2006). Symptoms—Historical Perspective and Effect on Diagnosis. In M. Blumenfield and J. J. Strain (eds), *Psychosomatic Medicine* (pp. 27–38). Philadelphia: Lippincott Williams & Wilkins.

Berrios, G. E., and Marková, I. S. (2014). Towards a New Epistemology of Psychiatry. In L. J. Kirmayer, R. Lemelson, and C. Cummings (eds), *Revisioning Psychiatry. Cultural phenomenology, Critical Neuroscience and Global Mental Health*. Cambridge, UK: Cambridge University Press.

Brambilla, P., Bellani, M., Yeh, P. H., et al. (2009). White matter connectivity in bipolar disorder. *International Review of Psychiatry*, **21**, 380–6.

Cuthbert, B. N., and Insel, T. R. (2013). Toward the future of psychiatric diagnosis: the seven pillars of RDoC. *BMC Medicine*, **11**, 126.

Decety, J., and Cacioppo, S. (2012). The speed of morality: a high-density electrical neuroimaging study. *Journal of Neurophysiology*, **108**, 3068–72.

Drevets, W. C. (2001). Neuroimaging and neuropathological studies of depression: implications for the cognitive-emotional features of mood disorders. *Current Opinion in Neurobiology*, **11**, 240–9.

Faux, S. F. (2002). Cognitive neuroscience from a behavioural perspective: a critique of chasing ghosts with Geiger counters. *The Behavior Analyst*, **25**, 161–73.

Ferrater Mora, J. (1991). *Diccionario de Filosofía. Círculo de* Lectores, Barcelona.

Halligan, P. W., and David, A. S. (2001). Cognitive neuropsychiatry: towards a scientific psychopathology. *Nature Reviews Neuroscience*, **2**, 209–14.

Hardcastle, V. G., and Stewart, C. M. (2009). fMRI: a modern cerebroscope? The case of pain. In J. Bickle (ed.), *The Oxford Handbook of Philosophy and Neuroscience* (pp. 179–99). Oxford, UK: Oxford University Press.

Heeger, D. J., and Ress, D. (2002). What does fMRI tell us about neuronal activity? Nature *Reviews Neuroscience*, **3**, 142–51.

Honey, G. D., Fletcher, P. C., and Bullmore, E. T. (2002). Functional brain mapping of psychopathology. *Journal of Neurology, Neurosurgery & Psychiatry*, **72**, 432–9.

Hulshoff Pol, H. E., Schnack, H. G., Mandl, R. C., et al. (2004). Focal white matter changes in schizophrenia: reduced interhemispheric connectivity. *Neuroimage*, **21**, 27–35.

Insel, T., Cuthbert, B., Garvey, M., et al. (2010). Research domain criteria (RDoC): toward a new classification framework for research on mental disorders. *American Journal of Psychiatry*, **167**, 748–51.

Jezzard, P., and Buxton, R. B. (2006). The clinical potential of functional magnetic resonance imaging. *Journal of Magnetic Resonance Imaging*, **23**, 787–93.

Kanaan, R. A. A., and McGuire, P. K. (2011). Conceptual challenges in the neuroimaging of psychiatric disorders. *Philosophy, Psychiatry & Psychology*, **18**, 323–32.

Logothetis, N. K. (2008). What we can do and what we cannot do with fMRI. *Nature*, **453**, 869–78.

Marková, I. S., and Berrios, G. E. (1995). Mental symptoms: are they similar phenomena? The problem of symptom heterogeneity. *Psychopathology*, **28**, 147–57.

Marková, I. S., and Berrios, G. E. (2009). Epistemology of mental symptoms. *Psychopathology*, **42**, 343–9.

Marková, I. S., and Berrios, G. E. (2012). Epistemology of psychiatry. *Psychopathology*, **45**, 220–7.

Ochsner, K. N., Beer, J. S., Robertson, E. R., Cooper, J. C., Gabrieli, J. D. E., Kihlstrom, J. F., and D'Esposito, M. (2005). The neural correlates of direct and reflected self-knowledge. *NeuroImage*, **28**, 797–814.

Oxford English Dictionary, Second edition (1989). Oxford UK: Oxford University Press.

Price, C. J., and Friston, K. J. (2002). Functional neuroimaging studies of neuropsychological patients: applications and limitations. *Neurocase*, **8**, 345–54.

Racine, E., Bar-Ilan, O., and Illes, J. (2005). fMRI in the public eye. *Nature Reviews Neuroscience*, **6**, 159–64.

Shad, M. U., Keshavan, M. S., Steinberg, J. L., et al. (2012). Neurobiology of self-awareness in schizophrenia: an fMRI study. *Schizophrenia Research*, **138**, 113–19.

Spence, S. A., Kaylor-Hughes, C., Farrow, T. F. D., et al. (2008a). Speaking of secrets and lies: the contribution of ventrolateral prefrontal cortex to vocal deception. *NeuroImage*, **40**, 1411–18.

Spence, S. A., Kaylor-Hughes, C. J., Brook, M. L., et al. (2008b). 'Munchausen's syndrome by proxy' or a 'miscarriage of justice'? An initial application of functional neuroimaging to the question of guilt versus innocence. *European Psychiatry*, **23**, 309–14.

Sprevak, M. (2011). Neural sufficiency, reductionism and cognitive neuropsychiatry. *Philosophy, Psychiatry & Psychology*, **18**, 339–44.

Sutton, B. P., Ouyang, C., Karampinos, D. C., et al. (2009). Current trends and challenges in MRI acquisitions to investigate brain function. *International Journal of Psychophysiology*, **73**, 33–42.

Tanskanen, P., Ridler, K., Murray, G. K., et al. (2010). Morphometric brain abnormalities in schizophrenia in a population-based sample: relationship to duration of illness. *Schizophrenia Bulletin*, **36**, 766–77.

Uttal, W. R. (2001). *The new phrenology*. Cambridge, MA: MIT Press.

Uttal, W. R. (2004). Hypothetical high-level cognitive functions cannot be localized in the brain: another argument for revitalized behaviorism. *The Behavior Analyst*, **27**, 1–6.

van der Meer, L., Costafreda, S., Aleman, A., et al. (2010). Self-reflection and the brain: a theoretical review and meta-analysis of neuroimaging studies with implications for schizophrenia. *Neuroscience and Biobehavioral Reviews*, **34**, 935–46.

Chapter 8

Translational validity across neuroscience and psychiatry

Drozdstoj St. Stoyanov, Stefan J. Borgwardt, and Somogy Varga

8.1 Introduction: Validity, Realism, and Instrumentalism

In all fields of inquiry, be it in the humanities or the sciences, an important goal is to establish the *validity* of theories, methods, and lastly knowledge about the world. In this context, validity (derived from the Latin *validus*) means "well-grounded" and "sound." Nonetheless, the criteria by which we judge whether knowledge is valid differ between the fields. For instance, philosophy, and particularly the tradition of hermeneutics, addresses the conditions under which valid understanding and valid interpretation can proceed. Hermeneutics focuses on meanings rather than facts. In hermeneutics, we can say that we are dealing with valid knowledge if we are able to place the phenomenon under investigation in that "space of reasons," to use a term coined by the philosopher John McDowell.

The picture is different with scientific knowledge. Scientific knowledge aims to provide us with plausible explanations and trustworthy predictions of phenomena in the world. While different scientific disciplines share many features, such as the generation of explanatory theories, reliance on observable evidence, and testing of hypotheses by experimental studies, they fundamentally differ in their methods of validating theoretical constructs. This may partly be why the meaning of validity tends to remain ambiguous and it tends to elude a neat cross-disciplinary definition.

In addition, the answer to the question of validity in the sciences also hinges on whether one adopts an *instrumentalist* or *realist* attitude toward the methods and results of scientific inquiry. While proponents of both attitudes agree that science advances by trial and error and generates genuine knowledge, the question that divides them is the nature of the knowledge thus generated. On an instrumentalist conception, the knowledge that scientific investigations give us is understood as trustworthy quantitative predictions of phenomena. In this

case, scientific knowledge is *instrumental*: it provides us with suitable information about some limited domain of phenomena, and it explains and solves problems associated with that domain.

On a realist conception, there is a general agreement that a valid theory is not merely explanatorily powerful, but in an important sense captures the nature of a mind-independent world and thus "cuts nature at its joints." In other words, the knowledge that scientific investigations offer us should be comprehended as true discovery and accurate description of the world "as it really is," independently of human perceptions, theories, and methods of measurements.

It is quite clear that these positions are committed to very different underlying metaphysical pictures. Realists claim that the world is independent of particular theories and that scientific statements are truth-evaluable, i.e., they can be confirmed or rejected by empirical research. On the instrumentalist view, scientific statements are not truth evaluable, and should therefore not be evaluated based on how accurately they describe objective reality; rather, they are assessable by their usefulness, thus by how successfully they explain and predict particular phenomena.[1]

8.2 The Question of Validity in Psychiatry

In psychiatry, the question of validity and validation is particularly important and complex. Since its birth in the nineteenth century, psychiatry has occupied a unique position within the science of medicine (Gadamer 1996: 163). In spite of continued efforts to bring psychiatry back within the boundaries of neurology, it remains an amalgam discipline, located on the border between science and the humanities. After much work on improving the reliability of diagnostic criteria, the question of the validity in psychiatry is becoming especially prominent.

One of the challenges that makes the issue of validity in psychiatry extremely complex is the so-called brain–mind problem, which is a modern version of the well-known mind–body problem. Some argue that mental phenomena should be reduced to underlying brain processes, while others maintain that intentionality, or the subjective perspective, is irreducible to brain processes. Since the introduction of the DSM-III in 1980, American psychiatry in particular has sidestepped this issue by circumventing etiological theories in favor of a descriptive approach. Disorders are pigeonholed by sets of symptoms that are mainly elicited by patient report and observation. One advantage of adopting the descriptive approach to classification is its improved reliability over prior systems.

However, while the descriptive approach was able to improve reliability, it was not designed to establish the validity of classifications. The expectation was that

identifying and descriptively grouping covarying symptoms in clinical populations would be a major step toward explaining them by a common underlying etiology. Robins and Guze (1970) predicted that the validity of descriptively defined syndromes could be incrementally improved through increasingly precise clinical description, laboratory studies, delimitation of disorders, follow-up studies of outcome, and family studies. "Once fully validated, these syndromes would form the basis for the identification of standard, etiologically homogeneous groups that would respond to specific treatments uniformly" (Kupfer et al. 2002: xviii). However, the goal of explaining these syndromes with reference to an underlying etiology has not been achieved.

Reflecting upon the noteworthy development of the neurosciences in the second half of the twentieth century, thinkers such as Christopher Boorse (1975) claimed that psychiatry would inevitably evolve into a form of applied biological science. Today some think that the future psychiatry is destined to be "clinical neuroscience" (Reynolds et al. 2009). Many believe that the scope of mechanisms active in mental disorders can be confined to biological mechanisms. When those mechanisms are discovered, it is believed that psychiatry will merit the same scientific status as other areas of medicine. Neuroscience, it seems to many, is the solution to the problems of validation.

However, at the beginning of the twenty-first century, despite the early optimism of the "Decade of the Brain" neuroscientists have not discovered any biomarkers or laboratory tests for the most common psychiatric disorders. Those disorders with a confirmed biological disease or genetic defect listed in the DSM-5 (for, e.g., Alzheimer's and other forms of dementia) fall under the scope of neurology. Critical voices from prominent psychiatrists have diminished the widespread hope that neuroscience will soon provide solutions to psychiatric questions. One of the problems is, as Miller (2010: 718) puts it, that "a mental disorder need not be triggered by, due to, or explained by brain pathology any more than a software bug must be a consequence of hardware failure." Even in a seemingly clear-cut case in which it could be demonstrated that the etiology of a disorder involves causally active brain mechanisms, it remains a possibility that the respective mechanisms are causally affected by psychological events. Expressing his own pessimism about a simple biological etiology of mental disorders, Frances argues that a wide variety of pathways likely lead to the development of a disorder such as schizophrenia (Frances 2010). Frances somewhat pessimistically declares that diagnostic classification is the result of historical accretion and accident, and is not grounded on scientific necessity: "Our mental disorders are not more than fallible social constructs (but nonetheless useful if understood and applied properly)" (Frances 2010).[2]

Given a widespread (but not universal) skepticism of biological reduction, and the status of psychiatry as a hybrid discipline that embraces both the sciences and the humanities, making the case for realism and realist approaches to validity in psychiatry is—at least currently—out of reach. In this chapter, we attempt to draw the contours of a concept of *translational validity*, which is a non-conventional and instrumentalist approach to validation.

8.3 Translational Validity

In this section we claim that there are substantial differences between validation in the natural sciences and humanities, and because psychiatry is a hybrid discipline that embraces both the sciences and the humanities, a non-conventional approach to validation is called for. We name this approach *translational validity*. We will further argue that neuroimaging is an important instrument for establishment of translational validity under some conditions, e.g., simultaneous administration of the diagnostic assessment tools and brain scan.

8.3.1 Foundations of Validity and Validation Procedures in the Disciplines Constituting Psychiatry

The disciplines we will focus on are clinical psychology, psychopathology, and neuroscience. Clinical psychology is a discipline that studies the reports of patients using interviews and inventories, and that often relies on ideographic methods for understanding mental phenomena. Although contemporary clinical psychology is usually considered to be quantitative and thus scientific, in fact the items of its different assessment tools represent decontextualized narratives composed from excerpts of the patients and/or professional narratives (Stoyanov et al. 2012, 2013).

Clinical psychology and psychopathology are considered distinct fields of inquiry in this chapter. Clinical psychology operates within a predominantly humanistic framework and is therefore more *dimensional*, whilst psychopathology is understood as an attempt to impose *medical categorization* upon mental phenomena which are described as "symptoms" and "syndromes," unified in nosological blocks.

To some extent this distinction is provisional, since contemporary clinical psychology has already been mixed up with psychopathology and vice versa. However, they are still regarded as two distinct fields of expertise.

8.3.2 Validation in Clinical Psychology

Validation in clinical psychology is based on two kinds of comparisons. In the first, a score on the rating scale *under validation* is correlated with another score

on a rating scale, which is already *assumed to be valid*. The prototype for this approach is the *Minnesota Multiphasic Personality Inventory* (MMPI). Stoyanov et al. (2012) argued that the MMPI items were extracted statements/questions from patients' narratives as they emerged in qualitative psychiatric interviews. The items were empirically sorted into scales on the basis of their ability to distinguish between a specific diagnostic group (such as people with depression) and a non-psychiatric population. In this comparison the psychiatric diagnosis was *presumed to be correct* and uncontestable. In the second kind of comparison the clinical scales are administered to many people. The statistical index called Cronbach's alpha measures the extent to which all the items are measuring the same construct. Cronbach's alpha in particular is usually classified as a measure of internal consistency reliability, but it can also be considered as a measure of (factorial) validity as well whenever the rating scale is supposed to be homogenous.

Although psychological tests are quantified and represent generalizations across persons, their explanatory power is limited by the qualitative patient reports on which they are based. In this way psychological assessments tend to lack explanatory potential located outside the clinical measures to underpin them and could therefore benefit from independent cross-validation, especially with the methods of neuroscience.

8.3.3 Validation in Neuroscience

Contemporary neuroscience encompasses genetics, physiology, and functional neuroimaging. As a robust natural science, it seeks to discover objective scientific evidence about the mechanisms underlying mental disorders. Yet neuroscience suffers from many methodological limitations in terms of its validity and clinical utility (Borgwardt 2012; Stoyanov et al. 2012). To address validation, neuroimaging parameters need to demonstrate discriminative power at the single-subject level. Moreover, MRI modalities have to be calibrated across different scanners and centers, and provide good test–retest, inter-subject, and cross-scanner reliability. After reliability has been established, to achieve internal validity it needs to be determined that what is being measured is actually a clinically relevant psychopathological process. Also, neuroimaging needs to be applicable beyond research laboratory settings to clinical psychiatric situations (Rusconi and Mitchener-Nissen 2013).

8.3.4 Validation in Psychopathology

Validation in psychopathology occupies the very borderline area in between clinical psychology and neuroscience. It is operating with *hybrid objects*, called

"phenomena" (Berrios 2011), which cannot be exactly identified in the conventional operational languages of clinical psychology or neuroscience. Over the past decades after DSM III-R (1973), psychopathology has been operationalized with structured clinical interview protocols such as the Structured Clinical Interview for DSM (SCID, First et al. 2012), the Positive and Negative Syndrome Scale (PANSS, Kay et al. 1989), and the Montgomery–Asberg Depression Rating Scale (MADRS, Montgomery and Asberg 1979). These protocols are also based on patient narratives. From an epistemological point of view there is no substantial difference in cognitive content between psychological tests and structured clinical interviews; nonetheless, they remain distinguished in clinical practice. This leads to certain *epistemic circularity* in which dimensional rating scales (like MMPI) are validated backwards on structured clinical interviews (SCID), which are themselves validated on tests like the MMPI. Another problem with both tests and structured interviews is that they have led to what Andreasen (2006) called the "death of phenomenology." In effect, the careful description of patients' experiences has been replaced with conventional lists of reported symptoms. Yet this agenda misses the rationale for successful interplay and integration of the psychopathological quantitative assessment with neurobiological measures.

8.3.5 Epistemology of Meta-Language in Psychiatry: The Explanatory Gap

Meta-language (Berrios 2006) is a methodological tool for integrating the divergent disciplinary languages of psychiatry. The fundamental problem in integrating the different sources of psychiatric knowledge (clinical psychology, psychopathology, and neurosciences) is termed the "explanatory gap." The explanatory gap refers to the incommensurability of the nomothetic and ideographic disciplinary languages (Broome 2008). From a practical standpoint this means that the construct of "depression" in clinical psychology/psychopathology and in neuroscience are defined and measured in sometimes incompatible ways.

In the tests of the clinical psychologists, depression represents a dimensional measure. A high score (above a certain cut-point) on a dimensional depression scale is taken to be a valid indicator of a depressive disorder.

Structured interviews used in psychopathology differ from the abovementioned scales mainly in their observational, therefore presumably "objective" segment. This observation typically describes both verbal and nonverbal behavior of patients. As it has already been argued, the latter represents just another kind of structured "professional" narrative.

Neuroscience attempts to identify biological bases of *disorders* (e.g., genome-wide association studies, biomarkers at the level of serotonin transport and receptors, etc.), which are in turn practically *untranslatable* into clinical reality, especially into the assessments of descriptive psychopathology. This is one reason why it has not been possible to incorporate data from neuroscience into diagnostic criteria and contemporary classifications. This means that from an epistemological perspective, validity and validation in psychiatry are left at *mono-disciplinary* levels, either neurobiological or psychopathological.

In other words, patient narratives are hermeneutic but not explanatory and the measures obtained in neuroscience are potentially explanatory but not hermeneutic/meaningful, and that is the so-called explanatory gap. To manage this gap (or rather to escape from it) and avoid inter-paradigm controversies, contemporary psychiatry has adopted an instrumentalist approach to clinical taxonomy.

Therefore, the major issue which complicates the dialogue across the different disciplinary languages constituting psychiatry appears to be *translation* among them.

8.3.6 The Translational Validity and the Role of Neuroscience as External Validator

As a strategy for bridging the explanatory gap we propose a program of translational validation, which would use neuroimaging as a tool for *improvement of cross-disciplinary instrumental psychiatric validity*.

As has been stated elsewhere (Stoyanov, Machamer, et al. 2012, 2013; Stoyanov, Stanghellini, et al. 2013), clinical and neurobiological measures are considered valid for different reasons *inside* their own domains. All disciplines concerned with mental health establish internal or *intra-correlative validity*, i.e., psychological measures are validated with other psychological measures, and neurobiological measures are validated with other neurobiological tests. What is still missing is the *inter-correlative* or *inter-disciplinary validity, which entails consistent inter-domain translation*. Since the issue of translation is involved, we prefer the term "trans-disciplinary."

As a potential source of external validity for the scales of clinical psychology, neuroscience can contribute information from two major biological databases: (epi-)genetic risk factors and neuroimaging abnormalities. Unfortunately, most of the efforts to discover behavioral-genetic and epigenetic biomarkers in psychiatry are too inconsistent and unstable to underpin any translational validity (Yosifova et al. 2009; Betcheva et al. 2013).

One critical consideration against the implications of genetic markers in psychiatric diagnosis is their "*state independence*." The latter has been incorporated

into the "endophenotype strategy" and is defined as independence of the biomarkers from the current mental state, which means that endophenotypes are lifetime stable, present in both clinical episodes and remissions (Hasler et al. 2006). Whilst state independence might be a useful assumption for some retest stable mental phenomena such as the traits in the psycho-biological model of personality (Cloninger et al. 1993), it is less relevant for clinical states like bipolar depression which are in fact determined by instability of emotional regulation. In those cases, "state independence" would be a shortcoming rather than an advantage. On the contrary, we argue that "state dependence" should be rendered as an alternative and sounder approach to translation of the neurobiological mechanisms of mental disorders into clinical reality. State dependence means that certain correlations are directly relevant and specific to the current mental state. This is why the clinical and biological measures should be performed simultaneously in our paradigm.

8.3.7 Neuroimaging as a Translation Validity Operation

Structural and functional neuroimaging as potential external validators are also exposed to critical queries about their validity and clinical utility (Fusar-Poli and Broome 2006; Borgwardt et al. 2012; Korf et al. 2011; Stoyanov, Stanghellini, et al. 2012). On one hand, functional MRI is considered in our perspective as a translation validation tool because of the following reasons:

1. In comparison with other imaging methods, fMRI can capture very close to real-time brain response to psychological stimuli of diagnostic significance.
2. It has enhanced spatial resolution as compared to other neuroimaging techniques (e.g., Positron Emission Tomography—PET) and can penetrate the substrate of mental function—the oxygen metabolism of cortical and sub-cortical neural substrates. Cortical regions are usually easier to reach with neuroimaging; however, there are advanced methods in structural/functional MRI (PET and Magnetic Resonance Spectroscopy) that can specifically address sub-cortical brain regions.
3. Modern upgrades in fMRI facilities allow multimodal imaging which can integrate further modalities like receptor expression and quantitative electroencephalography (qEEG); the latter can substantially contribute to time resolution.

On the other hand, we have identified so far several major shortcomings, which seem to undermine the data translation from neuroimaging to psychiatry:

1. The psychological stimuli (such as emotional pictures processing) administered during functional brain scanning are specifically designed to study

general psychological processes and not day-to-day diagnostic constructs in clinical psychiatry (such as International Affective Picture System—IAPS).

2. Clinical assessment inventories, both observational and self-assessment (such as MADRS, BDI), are administered outside the brain scanner and are thus discrepant from the imaging findings. This argument concerns bipolar depression first, since one of the cardinal features of bipolar disorder is the instability of the circadian rhythm of emotions, which may vary significantly, especially in depression.
3. Statistical correlations between neuroimaging measures and clinical assessment are performed *post-hoc*, are very often unstable (not replicated) in larger cohorts, and cannot be regarded as cross-validation operations. In this way no validity connections are traced across the explanatory and ideographic knowledge in psychiatry.

Those shortcomings, however, can be managed with some modifications in the experimental paradigm, in particular with simultaneous—concordant in real time—and full-length administration of clinical measures during a functional neuroimaging session. One way to do this would be to project one by one the items from the selected clinical evaluation scale (e.g., Depression-Scale by Von Zerssen 1986 or Beck Depression Inventory 1988, etc.) in real time on a screen above the patient inside the scanner during fMRI. The patient's rating responses will be recorded by a button click of a four-button response panel that is installed in the fMRI system anyway. Should the two measures correspond, they are regarded as convergent translational validity operations.

The explicit objective of such protocol is aiming at cross-validation as a complementary approach for the establishment of bi-conditional rules for translation of the data of neuroscience to clinical psychology and psychopathology. Following the state dependence argument as exposed earlier, we set aside re-test stable *personality traits* as measured by MMPI, EPQ (Eysenck Personality Questionnaire), or TCI (Temperament and Character Inventory). Those are complex intentional structures which are difficult to assess simultaneously with fMRI. Besides, much progress has been achieved in the paradigm of C. R. Cloninger with TCI without time synchronization of the measures (Gusnard et al. 2003). This was our reason to focus on frequently employed in diagnostic practice brief *state measures* instead, such as the Beck Depression Inventory and the Von Zerssen Depression Scale.

It is suggested bi-directional cross-validation in our model: using the functional neuroimaging measures as external validator of psychological clinical test scores and using the psychological test scores as a way to bring a more hermeneutic dimension into the procedures of validation in psychiatric neuroimaging.

The translation takes place on two levels: empirical and meta-empirical. First, the corresponding empirical measures are cross-validated (e.g., depression clinical rating score on BDI and fMRI blood oxygenation level dependent (BOLD) activation); then the entire constructs and relevant theoretical models.

Those rules or "manual for translation" may provide a synergistic explanation for the mechanism of production of the disorder and facilitate the inter-domain dialogue.

8.4 Methodological Underpinnings and Limitations of Functional Neuroimaging

Much hope in psychiatry has been directed toward functional neuroimaging approaches, which promise to identify core neurobiological alterations. A non-invasive technique that can be used repeatedly in a clinical population, neuroimaging could in principle support diagnosis and effective interventions in psychiatry (Borgwardt and Fusar-Poli 2012; Borgwardt 2013).

8.4.1 Functional Brain Imaging Methods

Functional brain imaging methods such as functional magnetic resonance imaging (fMRI), which allow the in vivo investigation of human brain function, have been increasingly employed to examine the neurophysiological substrate of cognitive processes and psychopathological features. As the signals of the human brain functions are universal, fMRI studies that explore the neural substrates of psychopathology theoretically no longer rely on subjective measures, resulting in numerous publications of fMRI studies employing task- and non-task-related paradigms.

8.4.2 Methodological Considerations

Methodological factors may account for the considerable heterogeneity in findings across fMRI studies. These factors include differences in relevant acquisition design, lack of statistical power due to small sample sizes, different methods of image analysis (i.e., parametric versus non-parametric), differences in the demographic and sociodemographic group characteristics, and confounding effects of medication or illness chronicity. Analysis of the consistency and comparability of the results obtained using different fMRI acquisition and analysis methods on the same set of neuroimaging data is a crucial prerequisite for accurate localization of various brain functions. To reliably apply fMRI in clinical settings, stable and consistent results, irrespective of the particular image acquisition and analysis methods used, are needed. In addition, a cognitive frame shift is required from the empirical level of investigation to trans-disciplinary validation of the clinical assessment tools and imaging data.

For a program of translational validity to succeed, it is crucial that the results of psychiatric neuroimaging can become more reliable. In what follows we offer some practical guidelines to conduct or evaluate functional neuroimaging studies in a program of translational validity (Borgwardt et al. 2012):

- Implementation of an increased number of ways of pre-processing the data Regions of Interest (ROIs) studies (employing preselected masks or adopting Small Volume Corrections) should first report standard whole brain results and acknowledge if no significant clusters were detected at whole brain level before presenting the ROI findings;
- Both ROIs and whole brain studies should first report the results significant at $p<0.05$ corrected for multiple comparisons (i.e., FWE, FDR, Montecarlo) and then employ more liberal thresholds;
- When several ROIs are used, correction for multiple comparisons should be based on a mask which includes all of them rather than considering each ROI separately;
- Authors should be encouraged to blind the statistical analyses of the imaging datasets to avoid ROIs analyses being built post-hoc on the basis of the results;
- All studies should report a statistical analysis modeling an agreed set of possible confounding variables; these could include, for instance, gender, age, and handedness. In addition, studies would have the option of reporting further statistical analyses modeling additional study-specific confounding variables;
- All studies should acknowledge the number of analyses or brain correlations performed, giving a clear rationale for each, to avoid conducting exploratory analyses and reporting the most significant result;
- The potential overlapping of the patient and control group with previously published studies should be clearly acknowledged, and the spatial coordinates always reported, to assist future voxel-based meta-analyses in the field;
- Peer reviews should be as strict when assessing the methods of a study reporting abnormalities in expected brain regions, as when assessing the methods of a study not finding any expectable finding;
- Acceptance or rejection of a manuscript should not depend on whether abnormalities are detected or not, or on the specific brain regions found to be abnormal.

In summary, neuroimaging methods may help to understand the pathogenesis of brain changes to clarify the onset and dynamic neurobiological processes underlying psychiatric disorders. However, for neuroimaging to be a clinically useful and valid tool, a framework linking basic, clinical research and target-specific treatments for people with psychiatric disorders should be developed. Translational validation is one vehicle to enhance this link (Stoyanov 2009, 2011).

8.5 Empirical Findings: Toward Translational Validation

To support the feasibility of our theoretical model of translational validation, we will review the empirical findings in the paradigm of "*high risk*" *for psychosis* (Koutsouleris et al. 2012). Early clinical intervention in schizophrenia has become a major objective of mental health services, and the finding that structural and functional alterations in the cingulate cortex during a first episode of psychosis are related to outcomes is of great interest (Bora et al. 2011). However, cingulate function and structure has also been reported to be especially sensitive to remedial antipsychotic treatment in psychosis (Lahti et al. 2009; Stip et al. 2009) and there is evidence indicating that a few weeks of antipsychotic treatment modulate the functional response in this region (Lahti et al. 2004; Snitz et al. 2005).

Previous reviews and meta-analyses have shown that significant brain changes driven by antipsychotic exposure can play a prominent confounding role in psychiatric imaging, thus preventing its translational clinical application (Smieskova et al. 2010; Fusar-Poli et al. 2013, published online). One possible approach to circumvent this problem is to selectively analyze drug-naïve first-episode subjects. For example, in a recent meta-analysis of untreated first-episode subjects, structural alterations in the cingulate cortex appear to be present before the initiation of antipsychotic treatment (Fusar-Poli, Radua, et al. 2011).

An alternative option would be to endorse "close in" clinical high risk (HR) approaches to identify a group of individuals with higher transition rates to psychosis (18 percent after six months of follow-up, 22 percent after one year, 29 percent after two years, and 36 percent after three years [Fusar-Poli, Bonoldi, et al. 2011]) than those observed in the general population. This clinical strategy aims at identifying neural changes occurring prior to the onset of psychosis and may improve translational ability of neuroimaging to predict schizophrenia outcomes. The presence of individuals who are high risk but not psychotic is consistent with evidence that schizophrenia results from the interaction of environmental with both genetic and neurodevelopmental factors, with the latter associated with clinical, neurobiological, and neuropsychological features before the onset of psychosis.

In recent years, a broad range of functional imaging methods have rapidly developed as powerful tools to explore the neurophysiological basis of the HR (Fusar-Poli, Borgwardt, et al. 2011; Fusar-Poli et al. 2007). Overall, these studies have shown that several abnormalities in brain function and neurophysiology that are fundamental to schizophrenia are also present in people at HR of psychosis, and may therefore represent vulnerability markers (Fusar-Poli et al. 2007).

Meta-analyses of whole brain structural studies comparing HR subjects with controls have confirmed reduced gray matter volume in the HR as compared to controls in the cingulate cortices as well as in temporal, prefrontal, parahippocampal/hippocampal regions (Fusar-Poli, Borgwardt, et al. 2011; Fusar-Poli et al. 2013). Volumetric reductions in cingulate and temporal, insular, prefrontal cortex, and in cerebellum have been also associated with the development of psychosis over follow-up (Smieskova et al. 2010).

The largest study published to date showed that the non-converting HR group demonstrated significant improvement in attenuated positive symptoms, negative symptoms, and social and role functioning, with more than 50 percent of this non-converting sample no longer presenting with any HR symptoms (Addington et al. 2011). However, this group remained on average at a lower level of functioning than non-psychiatric comparison subjects, suggesting that initial HR categorization is associated with persistent disability for a significant proportion for at least two years (Addington et al. 2011). It would be very useful to address functional changes associated with remission status within the HR cohort to identify protective neurobiological markers of later development of illness. Additionally, there is evidence from functional imaging and neurochemical HR studies that the extent of abnormality at baseline is predictive of subsequent conversion to psychosis (Smieskova et al. 2010).

These neurofunctional abnormalities of the At-Risk-Mental-State (ARMS) were not only related to different duration of ARMS, but also to gray matter reductions (GMV) (Smieskova et al. 2011) and the GMV itself was positively correlated with clinical outcomes as global functioning, negative symptomatology, and hallucinations (Smieskova et al. 2011). In particular, MRS studies have revealed reduced neuronal density and increased membrane turnover in cingulate as well as in frontal and insular lobe in HR subjects who later developed psychosis. Overall, the burden of functional imaging research into the HR state for psychosis has progressed exponentially, sustaining preventive interventions in clinical psychiatry (Ruhrmann et al. 2010).

However, despite these promises, validity of HR criteria is still highly discussed and the problem of the high number of false positives undermines the benefits of preventive interventions. In order to transcend over mere reliability and convert into valid inter-disciplinary paradigm, AMRS needs to employ the rationale of the translational cross-validation of the methods for clinical assessment and functional neuroimaging. In particular, the temporal gap between acquisition of clinical and functional brain measures may still present a limitation for the translation of the research findings (Korf and Gramsbergen 2007; Stoyanov et al. 2013; Stoyanov et al. 2012). Therefore, the management of the temporal discordance by simultaneous application of the two methods in

our paradigm may help to enhance the research designs of clinical neuropsychiatry (Stoyanov et al. in press).

There is thus an urgent need for psychiatric imaging to further develop linking neurobiological markers with longitudinal outcomes, including transition, remission, and response to preventative interventions.

8.6 **Conclusion**

The currently employed instrumental approaches are insufficient in terms of their validity and ability to integrate the domains of psychopathology and neuroscience in order to provide sound explanatory predictions of the mental phenomena. In this chapter we have explored the complex interdisciplinary structure of psychiatry as an amalgam of multifaceted sources of inquiry. The main sources, including psychopathological clinical assessment and neurobiological studies, have defined validity and validation procedures of their own and no particular approach to cross-disciplinary validation to foster the translation of data across the disciplines constituting psychiatry.

We proposed rules for making translations between clinical and neuroimaging data as one critical step forward to the introduction of the notion of translational validity.

The procedure of translational cross-validation entails simultaneous measurement of the brain activation measured by fMRI and self-report responses to psychological tests. The correlations between the two measures remain free of any ontological speculation about the mind–brain causation and are regarded as *ispo facto* real-time correspondence.

Further, we present substantial empirical and meta-empirical data from current neuroimaging investigations of at-risk-mental-state (AMRS). On one hand, this analysis provides critical insights into the limitations of functional brain imaging in psychiatry. On the other hand, the empirical findings of correlations between brain processes and clinical assessment support the thesis that if correlations are discovered with inert psychological stimuli (of no immediate diagnostic relevance), then they should exist between functional neuroimaging measures and clinical rating scales of diagnostic significance. The real-time diagnostic testing combined with Blood-Oxygenation-Level-Dependent (BOLD) fMRI may provide cross-disciplinary connections of translation and therefore back up sounder instrumental validation across mental health disciplines.

The critical analysis of the problem of validation as emerging in the interdisciplinary situation across psychiatry and neuroscience, as well as the current empirical results in other studies attempting to relate neurobiological data to clinical reality, do ascertain the feasibility of the model of translational validity.

Notes

1. This short introductory description is in many ways an oversimplification. The picture is more complex, and the question whether or not scientific statements are truth-evaluable will also depend on whether one embraces a correspondence theory of truth.
2. Of course, what we refer to here as a disappointment with neuroscientific progress is not a view that everybody shares. Indeed, it seems to be the case that the main tenor in current research is still the idea that mental disorders are to be understood and treated as brain disorders (Insel and Quirion 2005).

References

Addington, J., **Cornblatt, B. A.**, **Cadenhead, K. S.**, **et al.** (2011). At clinical high risk for psychosis: outcome for nonconverters. *American Journal of Psychiatry*, **168**, 800–5.

Andreasen, N. C. (2006). DSM and the death of phenomenology in America: an example of unintended consequences. *Schizophrenia Bulletin*, **33**(1), 108–12.

Beck, A. T., **Steer, R. A.**, **and Carbin, M. G.** (1988). Psychometric properties of the Beck Depression Inventory: Twenty-five years of evaluation. *Clinical Psychology Review*, **8**(1), 77–100.

Berrios, G. E. (2006). "Mind in general" by Sir Alexander Crichton. *History of Psychiatry*, **17**(4), 469–86.

Betcheva, E. A. G., **Yosifova, T.**, **Mushiroda, M.**, **et al**. (2013). Whole genome association study in Bulgarian population reveals HHAT as schizophrenia susceptibility gene. *Psychiatric Genetics*, **23 (**1), 11–19.

Boorse C. (1975). On the Distinction between Disease and Illness. *Philosophy and Public Affairs*, **5**(1), 49–68.

Bora, E., **Fornito, A.**, **Radua, J.**, **et al**. (2011). Neuroanatomical abnormalities in schizophrenia: a multimodal voxel-wise meta-analysis and meta-regression analysis. *Schizophrenia Research*, **127**, 46–57.

Borgwardt, S. (2013). 2989–Structural and functional neuroimaging in early schizophrenia: translating research evidence into clinical utility. *European Psychiatry*, **28**, 1.

Borgwardt, S., **and Fusar-Poli, P.** (2012). Third-generation neuroimaging in early schizophrenia: translating research evidence into clinical utility. *The British Journal of Psychiatry*, **4**, 270–2.

Borgwardt, S., **Koutsouleris, N.**, **Aston, J.**, **et al**. (2012). Distinguishing Prodromal from First-Episode Psychosis using Neuroanatomical Single-Subject Pattern Recognition. *Schizophrenia Bulletin*

Borgwardt, S., **Radua, J.**, **Mechelli, A.**, **and Fusar-Poli, P.** (2012). Why are psychiatric imaging methods clinically unreliable? Conclusions and practical guidelines for authors, editors and reviewers. *Behavioral and Brain Functions*, **8**(1), 46.

Broome, M. R. (2008). Philosophy as the science of value: neo-Kantianism as a guide to psychiatric interviewing. *Philosophy, Psychiatry & Psychology*, **15**(2), 107–16.

Cloninger, C. R., **Svrakic, D. M.**, **and Przybeck, T. R.** (1993). A psychobiological model of temperament and character. *Archives of General Psychiatry*, **50**(12), 975.

First, M. B., **Spitzer, R. L.**, **Gibbon, M.**, **et al**. (2012). *Structured Clinical Interview for DSM-IVo Axis I Disorders (SCID-I), Clinician Version, Administration Booklet*. Washington, DC: American Psychiatric Publishing.

Frances, A. (2010). Allen Frances Responds. *Bulletin of the Association for the Advancement of Philosophy and Psychiatry*, **17**(1), 21–5.

Fusar-Poli, P., Bhattacharyya, S., Allen, P., et al. (2010). Effect of image analysis software on neurofunctional activation during processing of emotional human faces. *Journal of Clinical Neuroscience*, **17**(3), 311–14.

Fusar-Poli, P., Bonoldi, I., Yung, A. R., et al. (2012). Predicting psychosis: meta-analysis of transition outcomes in individuals at high clinical risk. *Archives of General Psychiatry*, **69**(3), 220–9.

Fusar-Poli, P., Borgwardt, S., Crescini, A., et al. (2011). Neuroanatomy of vulnerability to psychosis: a voxel-based meta-analysis. *Neuroscience and Biobehavioral Reviews*, **35**(5), 1175–85.

Fusar-Poli, P., Borgwardt, S., and Valmaggia, L. (2008). Heterogeneity in the assessment of the at-risk mental state for psychosis. *Psychiatric Services*, **59**(7), 813.

Fusar-Poli, P., and Broome, M. R. (2006). Conceptual issues in psychiatric neuroimaging. *Current Opinion in Psychiatry*, **19**(6), 608–12.

Fusar-Poli, P., Broome, M., Barale, F., et al. (2009). Why is psychiatric imaging clinically unreliable? Epistemological perspectives in clinical neuroscience. *Psychotherapy and Psychosomatics*, **78**(5), 320–1.

Fusar-Poli, P., Perez, J., Broome, M., et al. (2007). Neurofunctional correlates of vulnerability to psychosis: a systematic review and meta-analysis. *Neuroscience and Biobehavioral Reviews*, **31**(4), 465–84.

Fusar-Poli, P., Radua, J., McGuire, P., et al. (2012). Neuroanatomical maps of psychosis onset: voxel-wise meta-analysis of antipsychotic-naïve VBM studies. *Schizophrenia Bulletin*, **38**(6), 1297–307.

Fusar-Poli, P., Smieskova, R., Kempton, M. J., et al. (2013). Progressive brain changes in schizophrenia related to antipsychotic treatment? A meta-analysis of longitudinal MRI studies. *Neuroscience and Biobehavioral Reviews*.

Gadamer, H.-G. (1996). *The Enigma of Health: The Art of Healing in a Scientific Age*. Stanford, CA: Stanford University Press.

Gusnard, D. A., Ollinger, J. M., Shulman, G. L., et al. (2003). Persistence and brain circuitry. *Proceedings of the National Academy of Sciences*, **100**(6), 3479–84.

Hasler, G., Drevets, W. C., Gould, T. D., et al. (2006). Toward constructing an endophenotype strategy for bipolar disorders. *Biological Psychiatry*, **60**(2), 93–105.

Insel, T. R., and Quirion, R. (2005). Psychiatry as a clinical neuroscience discipline. *JAMA: the journal of the American Medical Association*, **294**(**17**), 2221–4.

Ioannidis J. P. (2011). Excess significance bias in the literature on brain volume abnormalities. *Archives of General Psychiatry*, **68**(8), 773–80.

Kay, S. R., Opler, L. A., and Lindenmayer, J. P. (1989). The Positive and Negative Syndrome Scale (PANSS): rationale and standardisation. *The British Journal of Psychiatry*.

Korf, J., and Gramsbergen, J. B. (2007). Timing of potential and metabolic brain energy. *Journal of Neurochemistry*, **103**(5), 1697–708.

Koutsouleris, N., Borgwardt, S., Meisenzahl, E. M., et al. (2012). Disease prediction in the at-risk mental state for psychosis using neuroanatomical biomarkers: results from the FePsy study. *Schizophrenia Bulletin*, **38**(6), 1234–46.

Kupfer, D. J., First, M. B., and Regier, D. A. (eds) (2002). A Research Agenda for DSM–V. Washington, DC: American Psychiatric Association.

Lahti, A. C., Weiler, M. A., Holcomb, H. H., et al. (2009). Modulation of limbic circuitry predicts treatment response to antipsychotic medication: a functional imaging study in schizophrenia. *Neuropsychopharmacology*, **34**, 2675–90.

Miller, G. (2010). Mistreating psychology in the decades of the brain. *Perspectives on Psychological Science*, **5**(6), 716–43.

Montgomery, S. A., and Asberg, M. A. R. I. E. (1979). A new depression scale designed to be sensitive to change. *The British Journal of Psychiatry*, **134**(4), 382–9.

Radua, J., Mataix-Cols, D., Phillips, M. L., et al. (2011). A new meta-analytic method for neuroimaging studies that combines reported peak coordinates and statistical parametric maps. *European Psychiatry*

Reynolds, C. F., et al. (2009). The future of psychiatry as clinical neuroscience. *Academic Medicine*, **84**(4), 446–50.

Robins, E., and Guze, S. B. (1970). Establishment of diagnostic validity in psychiatric illness: its application to schizophrenia. *American Journal of Psychiatry*, **126**(7), 983–7.

Ruhrmann, S., Schultze-Lutter, F., Bechdolf, A., et al. (2010). Intervention in at-risk states for developing psychosis. *European Archives of Psychiatry and Clinical Neuroscience*, **260**, Suppl 2, S90–4.

Rusconi, E., and Mitchener-Nissen, T. (2013). Prospects of Functional Magnetic Resonance Imaging as Lie Detector. *Frontiers in Human Neuroscience*, **7**, 00594.

Simon, A. E., Velthorst, E., Nieman, D. H., et al. (2011). Ultra high-risk state for psychosis and non-transition: a systematic review. *Schizophrenia Research*, **132**, 8–17.

Smieskova, R., Allen, P., Simon, A., et al. (2011). Different duration of at-risk mental state associated with neurofunctional abnormalities. A multimodal imaging study. *Human Brain Mapping*, **33**(10), 2281–94.

Smieskova, R., Fusar-Poli, P., Allen, P., et al. (2010). Neuroimaging predictors of transition to psychosis—a systematic review and meta-analysis. *Neuroscience and Biobehavioral Reviews*, **34**(8), 1207–22.

Snitz, B. E., MacDonald, A., Cohen, J. D., et al. (2005). Lateral and medial hypofrontality in first-episode schizophrenia: functional activity in a medication-naive state and effects of short-term atypical antipsychotic treatment. *American Journal of Psychiatry*, **162**(12), 2322–9.

Stip, E., Mancini-Marie, A., Letourneau, G., et al. (2009). Increased grey matter densities in schizophrenia patients with negative symptoms after treatment with quetiapine: a voxel-based morphometry study. *International Clinical Psychopharmacology*, **24**, 34–41.

Stojanov, D., Korf, J., de Jonge, P., et al. (2011). The possibility of evidence-based psychiatry: depression as a case. *Clinical Epigenetics*, **2**(1), 7–15.

Stoyanov, D. (2009). The cross-validation in the dialogue of mental and neuroscience. *Dialogues in Philosophy, Mental and Neuro Sciences*, **2**(1), 24–8.

Stoyanov, D. (2011). A linkage of mind and brain: towards translational validity between neurobiology and psychiatry. *Biomedical Reviews*, **22**, 65–76.

Stoyanov, D., Machamer, P. K., and Schaffner, K. F. (2012). Rendering clinical psychology an evidence-based scientific discipline: a case study. *Journal of Evaluation in Clinical Practice*, **18**, 149–54.

Stoyanov, D., Machamer P., and Schaffner. K. F. (2013). In quest for scientific psychiatry: towards bridging the explanatory gap. *Philosophy, Psychiatry & Psychology*, **20**(3), 261–73.

Stoyanov, D., **Stanghellini, G.**, **and Broome, M.** (2012). Conceptual issues in psychiatric neuroimaging. *Current topics in medicinal chemistry*, **12**(21), 2348–56.

Stoyanov, D. S., Stieglitz, R. D., and Borgwardt, S. J. (in press). The translational validation as novel approach to integration of neuroscience and psychiatry. In A. L. Mishara, P. Corlett, P. Fletcher, A. Kranjec, and M. A. Schwartz (eds), *Phenomenological Neuropsychiatry, How Patient Experience Bridges Clinic with Clinical Neuroscience*. New York: Springer.

Von Zerssen, D. (1986). Clinical self-rating scales (CSRS) of the Munich psychiatric information system (PSYCHIS München). In *Assessment of depression* (pp. 270–303). Heidelberg: Springer Berlin.

Weinberger, D. R., **and McClure, R. K.** (date?). Neurotoxicity, neuroplasticity, and magnetic resonance imaging morphometry: what is happening in the schizophrenic brain? *Archives of General Psychiatry*, **59**(6), 553–8.

Yosifova, A., **Mushiroda, T.**, **Stoianov, D.**, **et al.** (2009). Case-control association study of 65 candidate genes revealed a possible association of a SNP of HTR5A to be a factor susceptible to bipolar disease in Bulgarian population. *Journal of Affective Disorders*, **117**(1), 87–97.

Chapter 9

Psychiatry, objectivity, and realism about value

Michael Loughlin and Andrew Miles

9.1 Introductory Remarks

When the editors of this volume asked us to supply a chapter outlining a "perspective" on psychiatric validation, they invited us to consider two questions:

1. How does your perspective compare and contrast with the other existing views/models of validation?
2. What are the prospects that your model can contribute to a single model of validation adopted by the whole field?

While the argument we go on to develop provides an answer, of sorts, to these questions, it is probably not the answer the editors were wanting or expecting. Indeed, it is an answer that might initially strike readers as bizarre, as it challenges certain pervasive background assumptions that, we argue, need to be revised before we can begin to make progress in this area. It is, if you like, the "groundwork" that needs to be done before we can attempt to give a sensible answer to the question of what is the right "model" of psychiatric validation.

We sketch the outline of an approach to validation, but it is one that converts questions about psychiatric validation into questions of a primarily moral nature, and our concluding comments make reference to the sort of epistemic and ethical virtues we need to develop via the education of practitioners, rather than suggestions for the development of formal guidelines, criteria, and unified processes. This is because we think that, before psychiatry can progress, we need to understand fully the underlying conceptual problems that led to what is sometimes termed the "crisis" in psychiatry (Loughlin et al. 2013b). Underlying assumptions, by no means exclusive to psychiatry, about the relationship between science and value generated quite specific problems for this area of practice. For psychiatry to defend and develop its intellectual framework we need to bring these assumptions out in the open, subject them to critical scrutiny, and, we argue, reject them.

So, in answer to question 2, we are precisely as far away from having a "single model" as we are from having a broad consensus on the nature of the human

good. But we can begin to defend different conceptions of the human good and use them as the basis for diagnosis—a diagnosis will be valid *contingent upon* the assumption of a normative framework, which will require defense in terms of moral arguments. In reply to question 1, what we offer here is more of a meta-perspective, a view on what is necessary for any model if it is to have a hope of being valid. We are not in a position to deliver the final word on any of the important practical issues other contributors to this volume discuss, but hope more modestly to "contribute" to the debate by providing a method for examining assumptions, reframing problems where necessary in an area that is going to remain extremely controversial for the foreseeable future.

9.2 **Don't Start Here**

There is a joke English tourists sometimes tell about asking directions in certain parts of Ireland. Supposedly, when you ask how to get from some remote place to a local landmark or vantage point, people will tell you, "Well, you don't start from here." Now, if that really is all the locals are prepared to say, then it is, arguably, a little unhelpful, but if followed by instructions on how to retrace one's steps, to get back to a place where it will be easier to get clear directions, then it may be the best, most practical advice it is reasonable to expect in the context.

Certainly, when it comes to matters more complex than the quest to find and photograph the Holy Stone of Clonrichert, there are questions to which the warning not to start from here is the best response that one can give (Loughlin 2007). In this chapter, we will argue that a cluster of questions surrounding the issue of psychiatric validation fall into this category, including how to classify mental disorders, and how to explain the relationship between mental and physical health and illness so as to be able to diagnose and care for the mental health needs of one's fellow human beings. Before we can give a full, satisfying, and truthful answer to these questions, we need to retrace the intellectual steps that led some astute contemporary thinkers to regard the very idea of "mental illness" with suspicion.

Questions about the scientific validity of psychiatric diagnosis derive their meaning and impetus from specific conceptions of science, value, and reality. It is possible to identify these conceptions and their origins in our intellectual history, and to examine the intellectual framework of which they form component parts. We propose that, instead of working within that framework, in this case what is needed is a revision of the framework itself—a redrawing of the conceptual map to describe different relationships between value, reality, and science. Sometimes, to solve particular problems, or even (more modestly) to discover a perspective upon the problems which enables us to view their solution as attainable, we need to accept that some fundamental feature of the way we see the world is wrong. In such cases, we do not need to gather further empirical

evidence, nor do we need a more astute analysis of that evidence, but rather we need a *philosophical* shift: a revision in the way the evidence is conceptualized or "framed" (Loughlin et al. 2010). Such a shift can change our views regarding what counts as evidence in the first place, and what methods of analyzing that evidence are appropriate. It will require us to step back from the current debate, to remind ourselves how we got to where we are now, and how certain dichotomies became part of our standard academic lexicon.

The feature of our contemporary world-view that stands in the way of progress, in the discussion of health care generally but most significantly in the discussion of mental health and illness, is a presupposition we will express as subjectivism with respect to value, or simply value-subjectivism. This presupposition is implicit in popular accounts of the key features distinguishing scientific analysis on the one hand, from moral judgment on the other (Loughlin 2013a), and it gives rise to what some authors characterize as "the myth of moral neutrality" in psychiatric diagnosis (Hamilton 2013) and in science in general (Loughlin 1998). Though it by no means originates in the modern era, today's pervasive subjectivism about value owes a good deal of its intuitive plausibility to the currently dominant and (in a sense we'll explain) characteristically "modern" view of the world and our place within it. Before we can arrive at a proper methodology in psychiatry, we must jettison those features of our conceptual framework that require authors either to deny the irreducibly moral nature of psychiatric diagnosis or to reject psychiatry as scientifically unsound.

Thus, we submit this chapter as a contribution to the philosophy of psychiatry, in that it does not represent a proof that subjectivism with respect to value is false (although, for independent reasons we think it is false), but it does tell us that we must believe this philosophical position to be false if we believe that psychiatric diagnosis can, in principle, be valid.

9.3 How We Got Here

Powerful criticisms articulated by exponents of the anti-psychiatry movement in the latter half of the twentieth century (Szasz 1960; Cooper 1967; Foucault 1987) led to what some authors have described as a "crisis" in psychiatry, one "sufficiently serious to jeopardize the constitution of psychiatry as a medical discipline" (Loughlin et al. 2013b: 418). While these arguments are well known, it is worth reminding ourselves that the key problem for characterizing psychiatric diagnosis as a valid branch of medicine was, for Szasz, the specific relationship between "the context of value" and the diagnosis of "mental illness":

> The concept of illness, whether bodily or mental, implies *deviation from some clearly defined norm*. In the case of physical illness, the norm is the structural and functional

> integrity of the human body . . . The norm from which deviation is measured whenever one speaks of a mental illness is a *psycho-social and ethical one*." (Szasz 1960: 114, emphasis in original)

While Szasz clearly recognizes that "the practice of medicine is intimately tied to ethics" (1960: 115), he maintains that psychiatry is "very much more intimately tied to problems of ethics than is medicine" (1960: 116) and attempts to capture the essential difference between each discipline's relationship with value by noting that, "although the *desirability* of physical health, as such, is an ethical value, what health *is* can be stated in anatomical and physiological terms" (1960: 114).

The point seems to be that while we cannot practice medicine in a way that is "free of ethical value" (and interestingly, we cannot do medical research without similarly becoming embroiled in "many ethical considerations and judgments" (Szasz 1960: 115)), we can at least explain the ontology of physical health in value-neutral terms, because "what health *is*" can be stated in terms of the language of anatomy and physiology. So, Szasz says:

> The notion of mental symptom is therefore inextricably tied to the *social* (including *ethical*) *context* in which it is made in much the same way as the notion of bodily symptom is tied to an *anatomical* and *genetic context* (1960: 114), [and] whereas bodily disease refers to public, physicochemical occurrences, the notion of mental illness is used to codify relatively more private, sociopsychological happenings of which the observer (diagnostician) forms a part. (1960: 116)

How is it that the observer "forms a part" when mental illness is being diagnosed, but not so when the illness being diagnosed is physical? There is an implied ontological distinction here: "bodily disease" is a "public" entity. The language of "public occurrences" suggests things that can be observed from any perspective, whatever the observer's private beliefs and values. In contrast, the identification of a mental illness requires engaging with norms of an "ethical" nature, which are, by implication, subject-dependent, being social constructs or subjective reactions to the reality observed. Having characterized the relevant norms as "ethical," Szasz feels this leads directly to the question (1960: 115): "Who defines the norms . . .?" swiftly giving rise to the follow-up question: "Whose agent is the psychiatrist?" Questions of agency and subjectivity are raised by the presence of ethical norms in a way that they are not immediately raised by diagnosis in (genuine) medical science, where what the thing observed "is" can be classified as a "bodily disease".

According to Szasz, the realization that the psychiatrist "does not stand *apart* from what he observes" but is already committed to a picture of the world that includes ethical norms "stands in opposition to a currently prevalent claim, according to which mental illness is just as 'real' and 'objective' as bodily illness"

(1960: 116). Szasz instantly qualifies this point by admitting some confusion as to exactly what is meant by such words as "real" and "objective," but he says he suspects "that what is intended by the proponents of this view is to create the idea in the popular mind that mental illness is some sort of disease entity, like an infection or a malignancy" (1960: 116).

We have quoted Szasz at some length here because it is important to establish that, for this leading figure in the anti-psychiatry movement, problems for the "objectivity" and "reality" of mental illness are closely related to the requirement for value-judgments (where the values in question are moral, or as Szasz prefers, "ethical"[1]) in the process of their diagnosis. While it is assumed that the "desirability" of physical health is an ethical matter, the ontology of disease is not: diseases are real "entities," and this means they can be identified without recourse to value-judgment. It would seem, then, that only that which is "objective" in this sense can be "real," though because Szasz expresses himself via speculation on what those he is criticizing might mean, we must be cautious about ascribing a clear thesis to him on this point. However, the idea that there is a close conceptual connection between objectivity and reality, and that both of them lie on the other side of a conceptual divide from "ethical value," does at least seem to be in influence.

So the extensive disputes in the contemporary philosophy of mind, about the relationship between specific mental states and brain states, while of great importance in their own right, do not in any immediate or obvious way impact on this particular problem. Even if we accept a strict identity theory, reducing any given mental state to some particular brain state, it will not follow that mental disorders are reducible to brain disorders, as what is at issue is the type of "norm" relevant to the diagnosis of the disorder—and as Szasz noted, that norm remains a moral one (Banner 2013).

The area in which to seek a solution, then, would appear to be ethics, or what is sometimes categorized as "meta-ethics," as it concerns the status of moral thinking and its relationship with other species of human thought. Human beings make value-judgments all the time, but is the making of a value-judgment a rational activity or some sort of alternative to rational thinking? Are "values" subjective reactions to the world, or is the making of certain value-commitments (or "evaluative perception") a prerequisite for understanding aspects of the world we encounter as they really are? (McDowell 1998; Dancy 2004).

In the decades following the publication of Szasz's arguments, authors such as Fulford (1989) convincingly argued that, even if we accept that psychiatric diagnosis is value-laden, this does not imply that the process is invalid, because there are reasons to believe that *all* medical diagnosis is value-laden. Fulford

(amongst many others) has been accused of employing something called "the likeness argument" (Pickering 2003, 2006) in inferring that because mental illness is relevantly similar to physical illness, and because we cannot plausibly give up on the concept of physical illness, we must conclude that mental illness is at least as "real" as physical illness. While the "likeness argument" is not, in our view, a fallacy (Loughlin 2003), we maintain that we need to go further than Fulford seems prepared to go. Giving a full defense of the intellectual legitimacy of diagnosis in both medicine and psychiatry entails adopting a view we will express as *realism with respect to value*.

This should not, we must note, be read as implying that by adopting this view we somehow render disputes about value less controversial, but simply that where there are controversies they are *bona fide* controversies, not expressions of "subjective opinion" disguised as substantive claims. Claims about value are contentious but *truth-apt*: the aim of such debates is to discover the truth. Value-judgments, we contend, can be genuinely true, or genuinely false. When a practitioner is making up her mind about whether a person has, or does not have, condition X, she is making a judgment that is value-laden. But she is also making up her mind about a real question, not simply bringing to bear her own "subjective feelings" on the matter. The ability of diagnosis in medicine and psychiatry to be genuinely correct or incorrect is conceptually tied to the status of the value-judgments underlying diagnosis: only if those judgments are truth-apt can it even be possible, in principle, for a diagnosis to be correct (or indeed, incorrect). Value-realism is a necessary presupposition of valid medical and psychiatric practice.

9.4 Science, Value, and Scientism

It follows that, to vindicate the necessary presuppositions of psychiatric diagnosis, we must believe two claims which, to many modern readers, may appear in tension if not outright contradiction. The first claim is that psychiatric diagnosis is inherently value-laden. The attempt to categorize a person's mental state as more or less healthy, or to consider a person as suffering from a mental illness or indeed as mentally healthy, logically presupposes taking up an evaluative stance, asserting certain normative statements to be the case, and this presupposes some normative framework. That is to say, when we describe someone as in good or poor mental health, or as suffering from a mental illness, we commit ourselves logically to a value-laden position, to the view that there are ways that people *should be* and ways that they *should not be*. Any attempt to reduce or eliminate the evaluative aspect of diagnosis must, therefore, fail. Diagnosis of mental health, and indeed diagnosis of health in general, is not a value-neutral

project. The normative judgments or claims involved are not reducible to statistical or other empirical claims.[2]

The second claim is that psychiatric diagnosis is an objective process in the specific sense that a diagnosis can be correct or incorrect. Claims about the mental health of persons are truth-apt: they can be true or false in the same way the claims about a person's weight can be true or false. Those who claim that *because* psychiatric diagnosis is a value-laden process it is therefore "subjective" or "relative" (such as those critics of psychiatry who claim that it is the unscientific imposition of arbitrary value-judgments upon human behavior) are mistaken. Psychiatric diagnosis can indeed be wrong, but this is because it can also be right. Wrong diagnoses can be extremely harmful, but even this judgment presupposes that claims about what is good or bad for persons are objective, in the sense that they are truth-apt.

Each of these claims might strike many readers as plausible in its own right. As Thornton (2011: 989) notes, "[t]o an unprejudiced eye, both the general concept of illness and specific instances of illnesses simply look to be evaluative," and claims that the "norms" in health are merely empirical and statistical just seem wrong because

> there is more to pathology in general than what is unusual . . . . Illness is *bad* for us. So unless there is a way to explain away that apparently evaluative or normative aspect of illness, there is good reason to believe appearances . . . . Merely statistical analyses of what is usual and unusual do not seem to capture the fact that high intelligence is in itself a good thing and low intelligence is a bad thing.

Trying to make something like the badness of borderline intellectual functioning objective by hand-waving in the direction of "value-free" evolutionary advantage doesn't help here. For instance, the relationship between having above average intelligence (by definition deviating from the statistical norm) and having more descendants than those with merely average intelligence, or indeed the just plain stupid, is by no means factually established.[3] However, citing Wakefield (1999), Thornton concedes that "[m]ore sophisticated attempts to use the notion of biological function have had the more modest aim of explaining away evaluative notions from the concept of *disorder*, rather than illness or disease, conceding that the latter notions also contain the ineliminable notion of harm"; but he notes that even with regard to that modest aim, "it is far from clear that the notion of failure of function presupposed explains away, rather than smuggling in, normative notions."

Although the attempt to make the badness of maladaptive behaviors value-free fails, such badness is not therefore merely a matter of opinion, if that means we cannot be right or wrong about what is bad or harmful to us. We aim to bring up our children to make sound judgments about what is and is not harmful, and to avoid harm because we want them to live well. Outside the

context of academic debate no serious person disputes the claim that it is possible to make correct and incorrect judgments about what is harmful to oneself (Loughlin 2002: 226–8).

Why, then, do we claim that many modern readers might find a tension or even contradiction between these two, independently plausible claims regarding the value-laden nature of diagnosis and its objectivity? Both the first and second claims can be true if, and only if, a specific philosophical view about the nature of value is correct. This is the view that normative claims, about what should be the case, can be true or false, just as empirical claims, about what is or is not the case, can be true or false. The process of diagnosis, to be possible and valid, presupposes a specific position in philosophical ethics, which we characterize as realism with respect to value.

The problem is that our uses of language, including the terms "objectivity," "subjectivity," "rationality," "science," and "value," are heavily influenced by a specific picture of the world and our place within it, which we have elsewhere characterized as "scientism" (Miles 2009; Miles and Loughlin 2011; Loughlin et al. 2013a). Scientism is sometimes equated with science, but this is a mistake. Scientism is not a scientific thesis but a philosophical thesis about the nature of science and "the relationship between science and either the truth, knowledge or reality" (Loughlin et al. 2013a: 131). So scientism can be understood as the view that science, *and only science*, "reveals the truth, such that all true claims are part of a true scientific theory, or are reducible to claims of this sort" (Loughlin et al. 2013a: 132). Scientism is distinguished from an alternative philosophical position called "scientific realism," which is the more modest view that the posits of true scientific theories are real. While the scientific realist believes that science reveals genuine aspects of reality, the believer in scientism goes further, asserting that science reveals the *essence* of reality, such that only the posits of true scientific theories are real, and all else must either be reducible to the posits of true scientific theories or consigned to the realm of fiction (Loughlin et al. 2013a: 135).

The influence of scientism explains why the quest to distinguish science from non-science became a major preoccupation of twentieth-century philosophy (Loughlin et al. 2013a: 132). If science, and only science, can reveal the nature of reality, then it becomes imperative to discover criteria distinguishing genuine science from non-science. According to the assumptions of scientism, disciplines that wish to be taken seriously as vehicles for the discovery of truth about the world are required to establish their scientific credentials or to be dismissed.

We have given numerous examples elsewhere of the pervasive influence of this particular world-view on popular debate and practice within a range of academic and professional areas (Miles 2009; Loughlin et al. 2013a). For our present purposes, the most significant implication concerns the relationship between

"objectivity" and "value." Scientism espouses what Nagel (1986: 91) called "an epistemological criterion of reality," defining what is real as that which is discoverable by science. The combination of this philosophical view with its account of the nature of science renders the idea of "objective value" a contradiction in terms:

> Descartes is often credited as one of the finest exponents of the "modern" world view. Writing at the dawn of the scientific age, he famously divided reality into two realms, the "inner" or "subjective" and the "outer" or "objective" realms. The external world was characterised in terms of the language of the emerging, physical sciences. The importance of quantification to the emerging sciences is fundamental to understanding Descartes' conception of the "external world". External reality is, by definition, something we can measure. In contrast, "phenomena" are internal, subjective properties dependent for existence on a perceiving subject. (Loughlin et al. 2013a: 137)

Thus modern thinkers see an absolute dichotomy between the subjective and the objective, with all properties assigned to one side of this divide or the other. Later versions of scientism turned on the "subjective" side, insisting on its denial or reduction to the objective side—hence the increasing tendency to equate the "objective" with (a) the properties of the "external world" (taken to be, exclusively, the measurable entities or properties posited by mechanistic science) and (b) claims that can be true or false (truth-apt).

By repeated association under the same term, based on the *assumption* that they are co-extensive, these two (logically distinct) senses of "objective" (publically observable and truth-apt) are effectively treated as equivalent. Eventually, the idea that all value-judgments are "subjective" acquires an almost self-evident status, as though it "just follows" from the meanings of "ordinary language" terms like "objective" and "true" (Loughlin et al. 2013a: 140). While the claim that Harry is 6ft tall refers to properties we can measure, the claim that Harry is a good person does not, so only the former claim is treated as truth-apt. If my criteria for calling someone a good person differ radically from yours, all that can be said is that we use the term in different ways, and there is no question that either usage (or associated criteria) can really be right or wrong: hence the modern dogma that all value-judgments are "mere expressions of opinion or preference."

Once this particular division between the subjective and the objective has been posited, a number of philosophical problems come into being. "Human beings are rendered inherently problematic entities as they seem to straddle both realms and have properties (such as cognition and choice) that are not easily assigned to either one realm or the other" (Loughlin et al. 2013a: 137).

Medicine is thereby rendered problematic, psychiatry even more so. Both concern the human good, so are deemed subjective. A natural inclination is to

rescue these disciplines by showing that the value-judgments they embody can be reduced to properly "scientific"—meaning value-neutral—properties and concepts. But this is a mistake. Scientism allowed human beings to focus on the measurable aspects of the world and this focus undoubtedly gave rise to massive intellectual and social progress as a direct consequence. But it would be hasty to conclude that, because a particular way of viewing the world gave rise to intellectual progress, it is therefore the conclusion of the intellectual evolution of the species: "We should be sceptical of the idea that intellectual history came to an end, that the definitive and final world view was discovered at just about the point that we arrived on the scene" (Loughlin et al. 2013a: 136).

The time to revise an underlying philosophy or conceptual framework is, precisely, when it ceases to facilitate progress and seems instead to be standing in its way.[4] Scientism's failure to accommodate the value-laden and "humanistic" aspects of clinical practice (Miles 2009) is a reason to revise this conceptual framework.

As we noted earlier, the employment by Fulford and others of what Pickering (2003) termed "the likeness argument" in support of the reality of mental illness need not be viewed as a fallacy, even though we concede that it does not, in itself, logically establish the conclusion that mental illnesses are real. The analogy with medicine serves to illustrate an important point. We would indeed have to give up far too much to maintain the absolute dichotomy between science and value presupposed by the framework of scientism. To maintain an absolute divide between our evaluative and "human" capacities on the one hand, and "objectivity" on the other, would make practice not only in psychiatry but in general medicine impossible. However extensive its empirical knowledge base, a robot could not be a good medical practitioner, unless we found a way to program it in addition with a sound normative framework, giving it the ability to make human value judgments (Gelhaus 2011).

It follows that, if "objectivity" means "value-neutrality," then it is a capacity of no use to, and in fact destructive of, good practice. To know the world it is necessary to be *engaged* with it, such that if "objectivity" excluded engagement it would have little or no epistemic value (McDowell 1998; Loughlin 1998). When we use "objectivity" to denote something positive, something worth having, we mean something like, the ability to see the world from perspectives other than one's own, or the ability to weigh arguments and reach a balanced conclusion. An objective person is not someone bereft of emotion, detached from and indifferent to the suffering of others (again, if it is a capacity we want practitioners of any sort to have), but rather it is someone with the mental discipline to find the level and manner of emotional engagement appropriate to respond compassionately and helpfully to the problem at hand (Marcum 2011).

Of course, such accounts of objectivity are value-laden, but to *complain* that such an account is evaluative is still to be caught up in the dichotomous framework which, we suggest, needs revising at this stage in our intellectual history, if debates about good practice are to move forward.

9.5 **Reclassifying Psychiatry**

Having retraced the intellectual steps that led to what some called the "crisis" in psychiatry, we have arrived at the conclusion that the discipline must abandon all pretentions to value-neutrality, and reject value-subjectivism in favor of value-realism. Psychiatry is a discipline whose essential purpose is concerned with promoting the human good. The fact that this project is value-laden is not the problem. What we need in order to explain the reality of mental health and illness is a less restrictive conception of reality.

The problem is philosophical: the influence of scientism and the idea that "objective reality" consists only of that which is detectable and measurable according to certain methods. Only when we make that idea explicit, identify it as the problem, and reject it, can we move forward and start to talk about the sort of value-judgments that unavoidably inform diagnosis, and discuss their rationale with reference to a defensible conception of the human good. That's the point to which we must return, before we can recommence our journey to validate our notions of mental health and illness. The debate we need to have is within the field of ethics. Ethics is not a side issue but conceptually central to psychiatry.

This does not mean that we must abandon science, but instead we must move beyond the idea that there is an absolute dichotomy or incompatibility between science and morality. Scientific thinking, like all human thinking, takes place in the context of living a human life, and engaging with the world in ways that require the making of value-judgments. Psychiatry and other disciplines devoted to improving people's mental health are moral disciplines, and it is the modern misunderstanding of that truth—the sense that it is worrying or problematic—that calls out for explanation.

We noted earlier that Fulford would not join us in defending value-realism, and his own thoughts on the issue nicely illustrate this modern reaction. Commenting on three responses to his own work on "Values-based practice" (Brecher 2011, Hutchinson 2011, and Thornton 2011), he asserts that:

> there are clear hints of totalitarian leanings (understood as commitment to pre-set "good outcomes") in all three commentators' positions: Brecher's apparent endorsement of "moral objectivism", . . . Hutchinson's advocacy of *Eudemonia* as "*the* Good Life" (p. 1001, emphasis added but Hutchinson's capitalization), and Thornton's moral particularism . . . all suggest authoritarianism. (Fulford 2013: 539)

According to Fulford, the problem with Brecher's moral objectivism (he is a Kantian), Hutchinson's commitment to Aristotelian ethics (as evidenced by his usage of "the Good Life"), and Thornton's moral particularism would seem to be, simply, that they are all versions of what we have called realism with respect to value. The very fact that these authors, in their very different ways, think that moral judgments are truth-apt, is a sign, for Fulford, that they are "authoritarians" with "totalitarian leanings." How does this follow?

Fulford notes that "authoritarianism in the guise of totalitarian psychiatry" was "the basis of some of the worst abuses of medical practice in the twentieth century" (2013: 539). Referencing the treatment of political dissidents in the Soviet Union, he adds that: "Similar though less endemic forms of abuse have been driven in all areas of psychiatry by this or that authority imposing its own particular vision of what is right" (2013: 539).

To be accurate, he should also note that the views about "what is right" here have by no means been restricted to views about what is *morally* right or wrong. Nor have the oppressors consistently used psychiatry as their rationale or mechanism of imposition. People have been deprived of their autonomy and dignity for disagreeing with the approved viewpoint on almost any matter, by those wielding political power, throughout recorded history. Religion and genetics have similarly been abused to vindicate violence, persecution, and even the attempted eradication of whole castes deemed decadent or inferior.

Those of us who espouse the value-realism Fulford apparently deems symptomatic of "authoritarianism" are in a position to regard these abuses as genuinely *wrong*—in contrast to the value-subjectivist, who must regard these things as wrong only from a given perspective, such that "the holocaust was just the Nazi's way of doing things" (Clark 1988). We can only have a rational basis for condemning totalitarianism if value-subjectivism is false, so any argument moving from the evident wickedness of totalitarianism to a rejection of value-realism looks at risk of pulling the inferential rug from under itself (Loughlin 2002: 206–21).

So what is Fulford's argument here? He does seem to move from the observation that these authors hold the view that moral judgments are truth-apt, to the implication that they are somehow (logically?) committed to approving of practices that he rightly regards as reprehensible. Because Brecher, Hutchinson, and Thornton think that evaluative questions can have right answers, can we infer that they are more likely to imprison you for disagreeing with them than someone who thinks that all moral questions are fundamentally arbitrary? Is someone heavily influenced by Nietzsche's work on moral nihilism (for instance, a 1930s fascist) far less likely to imprison those who oppose his political agendas than a Kantian moral objectivist like Brecher? We assume this is not what Fulford is saying, as it is clearly false.

People have been imprisoned and tortured for believing that the Earth orbits the sun, rather than vice versa. Is the conclusion to be inferred from this abuse of power that the issue in question cannot be an objective one, that the question of which object orbits which is just a matter of opinion? Instead of rejecting the idea that the question has a right answer, we should instead conclude that the use of violence, repression, and torture is not the correct way to settle controversial questions, because that way of settling such questions is rationally invalid and morally wrong. I do not prove that you are wrong about any matter, scientific or moral, by locking you up. Indeed, the desire to lock up dissidents may betray a lack of rational arguments on the matter at hand.

Fulford's equation of "totalitarian leanings" with "a commitment to pre-set 'good outcomes' "[5] (in psychiatry and in medical practice more generally) suggests a different reading of his argument. We take it as read that he is not claiming that his opponents are committed to a view about which outcomes are good, prior to considering the arguments and evidence relevant to any specific case. If so, then he would surely be knocking down a straw man, and doing a great disservice to his three correspondents. If he is simply saying that totalitarians claim, incorrectly, that by repression they will improve the lives of the people they repress, then surely the problem is that this claim is typically *false*. Self-determination is a component of the human good. While we cannot rule out in principle the possibility that some psychiatric patients, given their specific problems and circumstances, will need to be restrained for their own good, the burden of proof should always be on those advocating such extreme measures to argue that, in this specific instance, such an extraordinary decision is the right one. The fact that totalitarians have claimed, falsely, to be restraining people for their own good when, in fact, the restraint simply served the totalitarian's own political agenda, does mean we should look at all such arguments with a *particularly* skeptical eye.

Despite struggling to find a valid reading of his argument, we think that Fulford's worries (about treating psychiatry as fundamentally a discipline dedicated to promoting the good) will be shared by many modern readers, and not because either Fulford or those readers are misguided. One clear intellectual advance brought about by the attack on the objectivity of value-judgments was a greater skepticism, a greater caution regarding pronouncing on matters of right and wrong.

Taken to the logical extreme of value-subjectivism, such an attitude becomes self-defeating, as if there really is no right answer to a question, then it strictly doesn't matter which answer you give, as none is better than any other. In that case, the caution inspired by a degree of skepticism disappears. Caution (as a mean between the extremes of unreflective certainty and paralyzing self-doubt) is the

virtue that makes us aware of our own fallibility. So the skepticism about making value-judgments regarding the lives and behavior of others, evident in the work of thinkers including both Szasz and Fulford, expresses a healthy attitude, and one that needs to be cultivated in the education of practitioners in many fields, including psychiatry. That said, contrary to the views of both of these authors, the correct theory explaining *why* this attitude is healthy is that the more practitioners possess the virtue of caution, the more likely we are to have genuinely good outcomes, and the less likely we are to have outcomes that are genuinely harmful.

The cautious attitude may be at work in Fulford's apparent (and mistaken) belief that Hutchinson's use of the definite article and capitalization in characterizing "the Good Life" is a "clear hint" of an "authoritarian" mind-set. Fulford is of course well aware that this use of terminology reflects Hutchinson's commitment to Aristotelian virtue ethics, but Fulford draws attention to Hutchinson's talk of "the" Good Life (as opposed, one assumes, to "a" range of possible good lives) because Fulford is also acutely aware that psychiatry has often helped repress difference, to regard diversion from the norms of belief and action in one's own society as a sign of "madness" (Fulford 2013: 539).

This is an easy mistake to make if one fails to make the distinctions noted in the passages cited in Thornton's article between statistical and normative conceptions of "the norm." The statistical norm—knowledge of "what people usually do around here"—is rarely a good indicator of what normative stance we ought to take up with regard to the behavior in question. There are notorious examples, such as the classification of homosexuality as a mental illness in our not too distant history, to demonstrate the fallacy of moving from empirical observations of the statistical norm to patently evaluative conclusions about the status of such "abnormal" behavior, in the absence of any independent moral argument that there is anything genuinely wrong or harmful about the behavior to be "corrected." A rudimentary education in meta-ethics should be sufficient to expose the fallacy here—the same one that would lead us to attempt to "correct" those with above average intelligence to make them more stupid, so as to "help" them achieve the statistical norm.

The correct point to conclude from this, we think, is not that psychiatrists and others should be taught to think of diagnosis as value-neutral (which it never is), but rather that they need the sort of education, in critical moral thinking, that will enable them to realize why such evaluations are fallacious, and more broadly will enable them to practice well in a professional context that requires them to confront irreducibly evaluative questions. A minimal requirement for an acceptable education of this sort would be that it should make them powerfully aware that the conventions in one's own society are not immune from criticism. Ryan (2011) has argued that the education of social workers should emphasize the critical skill

of knowing when they must challenge, rather than enforce, social norms. As there is no point denying that their behavior will have some value-base, it is worth enabling them to think rationally about that value-base, to become the autonomous and virtuous professionals that we need them to be. Similar arguments seem to us to apply to the education of psychiatrists.

To evoke Aristotelian virtue ethics in defense of the repression of diversity in society would, of course, be to employ a "bastardized" version of virtue ethics, and not one to which Hutchinson has ever subscribed. People have a wide range of different skills, interests, qualities, and preferences, and allowing a diversity of lifestyles is the best way to facilitate human flourishing—just as allowing diverse opinions to be openly debated facilitates social progress. Such diversity benefits, rather than harms, the community, allowing new ideas to be considered so that intellectual and social progress remain real possibilities, allowing diverse skills and insights to contribute to the meeting of the community's needs, and generally making life for its members a good deal more interesting. Thinking that talk of "the Good Life" implies believing in one, homogenous vision of how to live well, so ruling out diversity, is like thinking that because someone refers to "the Ocean" he can only see a flat surface, and is unaware of all of the different eddies, currents, and waves that the mass of water necessarily embodies. Virtue ethics as a commitment to promoting the human good (and the value-realism it presupposes) no more requires calling in the "totalitarian psychiatrist" to stamp out diversity of thought and action within society, than calling a mass of water "the Ocean" implies calling in King Canute to command it to be still. Similarly, the versions of value-realism presupposed by Brecher and Thornton provide no valid defense of the totalitarian psychiatry Fulford rightly abhors. Properly understood, they provide ways of validating Fulford's underlying intuition that this is the wrong way to practice psychiatry.[6]

9.6 **Conclusion**

The debate about the values that should inform psychiatric practice has always been a moral one, and if freed from the shackles of scientism it could be debated unapologetically in these terms. All judgments—in science, in morality, in any aspect of human life—are "subjective" in the trivial sense that they require a subject, but not in the sense that they are "merely" subjective reactions to the world, such that they cannot be truth-apt.

To fully validate our claims in psychiatric and indeed in general medical diagnosis, we need to discuss and defend our value-judgments about health and illness. We must reject scientism for an openly value-laden account of human functioning. Medical epistemology (including the epistemology

of mental illness) requires value-realism. The contentious nature of the value-judgments in the case of mental illness should not mislead us into concluding they are "subjective" or "relative."

Are some value-judgments better than others? We contend that this is manifestly the case, and that it is the modern skepticism of that assertion that represents the real intellectual puzzle. Such skepticism can be vindicated not as a thesis but (at least partially) as an attitude that informs the mind-set of a virtuous practitioner. We need an approach to the education of practitioners, in psychiatry and in other areas of medicine, that cultivates the crucial virtue of caution with respect to judgments that can have a profound effect on people's lives. This involves recognizing the diversity of lifestyles that can represent human flourishing, while being open to the possibility that some lifestyles are genuinely harmful.

Ethics is not a subsidiary component of psychiatry but is conceptually central to the subject. It is not as though one can study the epistemology of psychiatry and then, as a separate task, discuss its ethics, as the latter forms an inseparable part of the former: taking up an evaluative stance toward the nature of psychiatric disorders is an essential component of understanding what a psychiatric disorder *is*. Education in the mental health professions should encourage cautious, critical reflection on the value-judgments about health and illness that inform diagnosis, and discussion of their rationale with reference to their underlying conception of the human good. As we have by no means arrived at the end of intellectual history or moral evolution, discussion of the correct way to characterize the human good is ongoing. But any defensible conceptions of mental health, any efforts to categorize mental illness and to diagnose it in practice, must be framed with reference to a conception of the human good, so the more serious thought we put into this fundamental ethical project, the better for all of us—those who practice and those they treat.

Notes

1. Some authors may feel there is an important distinction between "moral" and "ethical," but we have never been able to work out what precisely it is (cf. Loughlin 2002: 27–31).
2. Given a sufficiently broad conception of "experience"—for instance that embraced by Husserl (1970)—we could arguably include the normative within the "empirical," treating evaluative perception as part of our experience. But we are using the term here in a sense more akin to that of the British empiricists: normative claims do not count as "empirical" in the sense intended by Ayer (1987).
3. Anyone who believes there to be a systematic, necessary link between high intelligence and having many children is invited to watch the American comedy *Idiocracy*.
4. Arguably, Aristotelian attacks on pre-Socratic atomism represented progress in their time, but they rightly did not preclude the reintroduction of atomistic thinking at a later stage in human history (Loughlin et al. 2013a: 142).

5. The insistence on putting terms like "good outcomes" in inverted commas, even when not directly quoting, may suggest the background belief that no outcomes are *really* good or bad—it's just that some people say/think of things as good and bad, as part of their subjective reaction to the world.
6. In fairness to Fulford, we should point out that we have only focused on his specific claims about the belief in "good outcomes" and "authoritarianism," and his use of the examples he cites from psychiatry. He makes these claims in the context of a discussion of Values-based Practice (VBP). While we do not think this invalidates anything we have said, he would no doubt want us to say a lot about his distinction between "good outcome" and "right process" to do justice to that broader debate. For a discussion of VBP's relationship to value-subjectivism, see Cassidy (2013).

References

Ayer, A. J. (1987). *Language, truth and logic*. Harmondsworth, Middlesex: Penguin Books Ltd.

Banner, N. (2013). Mental disorders are not brain disorders. *Journal of Evaluation in Clinical Practice*, **19**(3), 509–13.

Brecher, B. (2011). Which values? And whose? A reply to Fulford. *Journal of Evaluation in Clinical Practice*, **17**(5), 996–8.

Cassidy, B. (2013). Uncovering values-based practice: VBP's implicit commitments to subjectivism and relativism. *Journal of Evaluation in Clinical Practice*, **19**(3), 547–52.

Clark, S. R. L. (1988). Mackie and the moral order. *Philosophical Quarterly*, **39**, 98–144.

Cooper, D. G. (1967). *Psychiatry and Anti-Psychiatry*. Londres: Tavistock.

Dancy, J. (2004). *Ethics without Principles*. Oxford: Oxford University Press.

Foucault, M. (1987). *Mental Illness and Psychology*. Berkeley, CA: University of California Press.

Fulford, K. W. M. (1989). *Moral Theory and Medical Practice*. Cambridge: Cambridge University Press.

Fulford, K. W. M. (2013) Values-based practice: Fulford's Dangerous Idea. *Journal of Evaluation in Clinical Practice*, **19**(3), 537–46.

Gelhaus, P. (2011). Robot decisions: on the importance of virtuous judgment in clinical decision making. *Journal of Evaluation in Clinical Practice*, **17**(5), 883–7.

Hamilton, R. (2013). The frustrations of virtue: the myth of moral neutrality in psychotherapy. *Journal of Evaluation in Clinical Practice*, **19**(3), 485–92.

Husserl, E. (1970). *Logical Investigations*, Volume II (Translator: Findlay, J. N.). London: Routledge and Kegan Paul.

Hutchinson, P. (2011). The philosopher's task: VBP and "bringing to consciousness." *Journal of Evaluation in Clinical Practice*, **17**(5), 999–1001.

Loughlin, A. J. (1998). *Alienation and Value-Neutrality*. Ashgate: Aldershot.

Loughlin, M. (2002). *Ethics, Management and Mythology*. Abingdon, Oxon: Radcliffe Medical Press.

Loughlin, M. (2003). Contingency, arbitrariness and failure, *Philosophy, Psychiatry and Psychology*, **10**(3), 261–4.

Loughlin, M. (2007). Thinking: where to start. Chapter 9 of Roulston, S. (ed.), *Prioritising Child Health: Principles and Practice*. London: Routledge, 51–62. ISBN 978-0-415-37634-1.

Loughlin, M., Bluhm, R., Stoyanov, D., et al. (2013b). Explanation, understanding, objectivity and experience. *Journal of Evaluation in Clinical Practice*, **19**(3), 415–21.

Loughlin, M., Lewith, G., and Falkenberg, T. (2013a). Science, practice and mythology: a definition and examination of the implications of scientism in medicine. *Health Care Analysis*, **21**(2), 130–45.

Loughlin, M., Upshur, R., Goldenberg, M., et al. (2010). Editorial introduction and commentary: "Philosophy, Ethics, Medicine and Health Care: the urgent need for critical practice." *Journal of Evaluation in Clinical Practice*, **16**(2), 249–59.

Marcum, J. (2011). The role of prudent love in the practice of clinical medicine. *Journal of Evaluation in Clinical Practice*, **17**(5), 877–82.

McDowell, J. (1998). *Mind, Value and Reality*. Cambridge: Harvard University Press.

Miles, A. (2009). On a medicine of the whole person: Away from scientistic reductionism and towards the embrace of the complex in clinical practice. *Journal of Evaluation in Clinical Practice*, **15**(6), 941–9.

Miles, A., and Loughlin, M. (2011). Models in the balance: Evidence-based medicine versus evidence-informed individualized care. *Journal of Evaluation in Clinical Practice*, **17**(4), 531–6.

Nagel, T. (1986). *The View from Nowhere*. Oxford: Oxford University Press.

Pickering, N. (2003). The likeness argument. *Philosophy, Psychiatry and Psychology*, **10**(3), 243–54.

Pickering, N. (2006). *The Metaphor of Mental Illness*. Oxford: Oxford University Press.

Ryan, T. (2011). *Animals and Social Work—a Moral Introduction*. Basingstoke: Palgrave.

Szasz, T. S. (1960). The Myth of Mental Illness. *American Psychologist*, **15**(2), 113–18.

Thornton, T. (2011). Radical liberal values based practice. *Journal of Evaluation in Clinical Practice*, **17**(5), 988–91.

Wakefield, J. C. (1999). Mental disorder as a black box essentialist concept. *Journal of Abnormal Psychology*, **108**, 465–72.

Chapter 10

Scientific validity in psychiatry: Necessarily a moving target?

James Phillips

10.1 Validity in the DSM

With the publication of DSM-5, it is clear that an important goal—diagnostic validity—was not achieved. The quest for this Holy Grail began back in 1980 with DSM-III (American Psychiatric Association 1980). The immediate goal of that manual was to achieve the first step in a scientific nosology—diagnostic reliability—with the use of operational definitions and diagnostic criteria. With DSM-III we could be confident that clinicians and researchers in different countries would be talking about the same phenomenon when they discussed, for instance, schizophrenia. Built into the DSM-III process, however, was the understanding that reliable diagnoses could not yet claim to be valid; we could *not* feel comfortable that the diagnostic concept in question represented a distinct, real entity in the world. How did we know, for instance, whether the diagnostic entity called schizophrenia described one distinct illness or several? In that way the accomplishment of DSM-III immediately unleashed a new anxiety and a new goal—securing diagnostic validity.

In prioritizing reliability over validity, the architects of DSM-III assumed that ongoing research would lead to valid diagnostic constructs. The standard of validity was the set of criteria proposed by Robins and Guze in their 1970 article (Robins and Guze 1970), along with subsequent publication of the Feighner diagnostic criteria in 1972 (Feighner et al. 1972), and Robert Spitzer's Research Diagnostic Criteria in 1978 (Spitzer et al. 1978). The profound shock of the ensuing two decades was the inability, despite extensive research, to achieve the Robins/Guze criteria, exemplified by the failure to delineate the pathophysiology of the major psychiatric disorders, and the corresponding failure to find biomarkers for these disorders. The DSM-III-R, DSM-IV, and DSM-IV-R diagnostic categories remained plagued by fuzzy boundaries, excessive comorbidity, and excessive clinical use of NOS diagnoses. It turned out that psychiatric disorders are vastly more complicated than imagined by Robins, Guze, and other experts of the DSM-III era (Detre 1987).

In the face of this crisis of validity, the architects of DSM-5 recognized that a major goal of DSM-5 would be to accomplish what had not been accomplished with DSM-IV or DSM-IV-TR. As the DSM-5 leaders wrote in the 2002 white paper, *A Research Agenda for DSM-V* (Kupfer et al. 2002):

> In the more than 30 years since the introduction of the Feighner criteria by Robins and Guze, which eventually led to DSM-III, the goal of validating these syndromes and discovering common etiologies has remained elusive. Despite many proposed candidates, not one laboratory marker has been found to be specific in identifying any of the DSM-defined syndromes. Epidemiologic and clinical studies have shown extremely high rates of comorbidities among the disorders, undermining the hypothesis that the syndromes represent distinct etiologies. Furthermore, epidemiologic studies have shown a high degree of short-term diagnostic instability for many disorders. With regard to treatment, lack of treatment specificity is the rule rather than the exception. (p. xviii)

The authors' response to this crisis was an appeal for a "paradigm shift."

> All these limitations in the current diagnostic paradigm suggest that research exclusively focused on refining the DSM-defined syndromes may never be successful in uncovering their underlying etiologies. For that to happen, an as yet unknown paradigm shift may need to occur. Therefore, another important goal of this volume is to transcend the limitations of the current DSM paradigm and to encourage a research agenda that goes beyond our current ways of thinking to attempt to integrate information from a wide variety of sources and technologies. (p. xix)

As DSM-5 got under way, the leadership realized that ongoing research in the DSM-III/DSM-IV manner would not resolve the validity problem or produce the promised paradigm shift. With this realization in mind the leadership at first turned toward a large-scale, dimensional reframing of the diagnostic categories (Regier et al. 2011), and then later looked forward to the NIMH Research Domain Criteria project (RDoC) (Kupfer and Regier 2011) for possible answers to the validity crisis. Neither had an effect on the final DSM-5 document.

The final note in this background review is the DSM-5 field trials. They strike an ironic note, given that the challenge of DSM-5 was presumably to establish diagnostic validity, diagnostic reliability having been mostly accomplished in DSM-III and its successors (Kupfer 2002). What we find, however, is that the Field Trials are back to reliability. Oddly, validity was more or less taken for granted while the trials were being developed to again test reliability. In the reports of the trials, Clarke et al. write that "[t]he face and construct validity of the revised DSM-5 diagnoses were subjectively confirmed by the Work Groups that proposed the diagnostic changes" (2013: 43), and Regier et al. write that "[s]ince the 1970s, the validity of psychiatric diagnoses has largely been supported by expert clinical consensus, based on a wide range of clinical experience and increasingly buttressed

by basic, clinical, and epidemiologic research" (2013: 59). (Regier et al. do acknowledge the limitations of established diagnostic validity, writing that "reliability studies set the stage for validity studies beyond face/construct validity" (2013: 59) but not clarifying how that will be achieved.)

The reasoning of the Field Trials appears to be: we cannot achieve full validity without firmly established reliability; we do not have the latter, so we have to start over with serious reliability studies; in the meantime, we have enough validity of sorts to warrant testing the existing and new diagnostic constructs for reliability.

If this reversal of priorities—assume validity, test reliability—is the first surprise from the Field Trials, the second is the results. Using the statistical methodology of kappa values for testing inter-rater reliability, the Field Trials tested 15 adult and 8 child/adolescent diagnoses. Five diagnoses were in the "very good" range, nine in the "good" range, three in the "unacceptable" range, and eight with insufficient sample sizes to generate kappa values.

Interpreting these results is a matter of perspective. The architects of the trials consider them a triumph. Indeed, it is arguable that they are on par with the reliability of general medical diagnoses (Pies 2007). On the other hand, it is striking that major depression and generalized anxiety disorder do not have acceptable kappa values. Given the enormous prevalence of anxiety and depression disorders, these findings seem like a big hole in the Field Trials.

Furthermore, while we should certainly acknowledge the limited success of the trials, we need also remember that they say little about diagnostic validity. Schizophrenia, schizoaffective disorder, and bipolar disorder, for instance, all do well in DSM-5 reliability—i.e., can be distinguished with diagnostic criteria—but we still don't know whether they represent one disorder or three.

Leaving such questions regarding diagnostic validity aside, DSM-5 Chairman David Kupfer, along with Task Force member Helena Kraemer, wrote about the trials, "We ultimately tested the criteria for 23 disorders. The question we asked was a straightforward one: In the hands of regular clinicians, assessing typically symptomatic patients in no different way than they would during everyday practice, was a particular disorder reliable?" (Kupfer and Kraemer 2012: 10). They say nothing about the establishing of validity, and their conclusion leaves us questioning what we have accomplished with DSM-5—a handful of new diagnostic categories and hair-splitting changes of existing criteria sets, but, as with DSM-III and IV, categories with no established validity, and likely persistent problems of heterogeneous presentations, excessive comorbidity, and fuzzy boundaries. Thus several years of work by devoted Work Groups, ending with limited success in reliability, which we had thought were answered with DSM-III in 1980, and no word on validity.

10.2 Two Types of Validity

In pursuing the question of validity of psychiatric diagnoses, we need to step back and examine how validity is defined. Validity can refer to the validity of the diagnostic criteria, validity of the external measures used to confirm diagnoses, and finally, validity of the diagnostic constructs themselves. These dimensions of validity are intertwined and can lead to circular reasoning. For instance, the validity of a diagnostic construct may be supported by its high scores on one or more external validators, but that then depends on the validity strength of the validators themselves, which in turn can be defended on the basis of their strong association with what seems like a desirable construct (Kendler 1990).

In most psychiatric discussion of diagnostic validity, validity tends to mean validity of the diagnostic constructs. This was Robins and Guze's (1970) understanding of validity and implicitly that of DSM-III and all subsequent revisions. They judged validity with the use of their five validators—clinical description, laboratory findings, separation from other disorders, follow-up studies, and family history—but their emphasis was on validity of the diagnostic constructs, not the validity of their five validators.

In what follows I contrast two representative forms of diagnostic validity within the Robins/Guze framework, which I designate as strong and weak validity, the first, represented by Kendell and Jablensky (2003), focusing on validity of the constructs, the second, represented by the Guidelines (Kendler 2009), used by the DSM-5 Work Groups in their consideration of existing and proposed new diagnoses, focusing on validity of the validators.

10.2.1 Strong Syndromal Validity (SSV)

Kendell and Jablensky distinguish two criteria for assessing validity:

> We suggest, therefore, that a diagnostic category should be described as valid only if one of two conditions has been met. If the *defining characteristic* of the category is a syndrome, this syndrome must be demonstrated to be an entity, separated from neighboring syndromes and normality by a zone of rarity. Alternatively, if the category's defining characteristics are more fundamental—that is, if the category is defined by a physiological, anatomical, histological, chromosomal, or molecular abnormality—clear, qualitative differences must exist between these defining characteristics and those of other conditions with a similar syndrome. (2003: 8)

For diagnoses that are defined only in terms of signs and symptoms (i.e., virtually all DSM diagnoses), Kendell and Jablensky highlight the third Robins/Guze criterion, "separation from other disorders," which they redescribe as a "zone of rarity," and declare that syndromal diagnoses are valid only if they meet that standard of strict separation from other disorders. For diagnoses that are defined at a more fundamental level, e.g., Huntington's Disease, defined in

terms of a dominant gene, the defining physiologic or neuroscientific features must be distinct from other disorders with similar presentations. Effectively, Kendell and Jablensky use two of the five Robins/Guze standards for asserting diagnostic validity: separation for other disorders for syndromal validity, and laboratory studies for more fundamental validity.

According to these authors, a diagnostic construct either meets these standards or it does not. They emphasize that all DSM syndromal diagnoses merge and blur to such a degree with other diagnoses that they do not meet the standard of syndromal validity. But, they argue, this does not make the diagnostic constructs useless. Validity is present or not, whereas utility is a matter of degree, and can exist even if validity does not. For purposes of treatment and ongoing research they earn their place in the diagnostic manual as "useful" if not valid categories.

Given that they do not countenance notions such as partial validity or degrees of validity, Kendell and Jablensky would not be in agreement with the idea of weak validity to be described in the next section.

10.2.2 Weak Syndromal Validity (WSV)

In developing DSM-5, the Work Groups followed the "Guidelines for Making Changes to DSM-V," most recently revised in 2009 (Kendler et al. 2009). These guidelines may be viewed as an elaboration of the Robins/Guze criteria and certainly represent an excellent, state-of-the-art approach to achieving diagnostic validity with syndromal diagnoses. While Kendell and Jablensky use only the Robins/Guze criteria of laboratory studies and separation from other disorders, the Guidelines use all of the Robins/Guze criteria, plus another proposed by Kendler, "response to treatment" (1990). (For both, the criterion of laboratory studies is an empty set for most of the DSM-5 diagnoses.) The authors begin by stating that the desired paradigm shift and validity won't be achieved in DSM-5: "While DSM-V will not in itself represent a 'paradigm shift', it is intended to start a process that will lead to more useful ways of classifying and diagnosing mental disorders based on our current knowledge and reasoned predictions of where the science is heading" (2009: 1). They outline a list of validators, grouped into categories of antecedent, concurrent, and predictive. They provide careful guidelines for use of the validators in the introduction of new diagnoses, the deletion of existing diagnoses, and any changes in current criteria sets, as well as changes in the relational structuring of diagnoses.

It is important to bear in mind that, in the ongoing absence of definitive findings from basic science that would move a diagnosis toward something more scientifically basic than a symptom cluster, the putative validity of psychiatric diagnoses under the Guidelines remains that of syndromal validity. All

questions regarding the validity of a particular diagnosis address the evidence in support (or not) of a particular syndrome. For instance, the high concordance rate of schizophrenia in identical twins lends support to the syndromal validity of schizophrenia as a diagnostic construct.

In discussing syndromal validity in the Guidelines approach, it is important to bear in mind that, in contrast to the "strong" Kendell/Jablensky syndromal criterion of separation from other disorders that lends itself to an either/or, valid-or-not-valid approach, the "weak" syndromal validity of the Guidelines approach implies partial validity, or degrees of validity. To take the example of identical twins, the higher the identical twin concordance rate for, say schizophrenia, the stronger the partial syndromal validity for that disorder.

With the publication of the DSM-5, we know the results of Work Group decisions, although, regretfully, we don't know the details of discussions that led to the decisions. New diagnoses in the manual include disruptive mood dysregulation disorder and binge eating disorder. Reorganization of existing DSM-IV diagnoses led to combining autism and Asperger's disorder into a single diagnosis called autism spectrum disorder. Proposed disorders needing further work and listed in Section III include attenuated psychosis disorder and mixed anxiety/depression disorder. As indicated, these changes were all made on the basis of syndromal validity. None of the changes is grounded in neurobiologic evidence.

The strength of the Guidelines approach to syndromal validity is the use of updated validators and the incorporation of the huge body of scientific research carried out since the publication of DSM-TR in 2000, both of which result in a diagnostic manual with much more promise of syndromal validity than any of its predecessors. The weakness of the approach is that DSM-5 may leave us more than not in the same place as DSM-III—diagnoses with some reliability but minimal hope of achieving real validity with the new set of validators. We can recall the words of Kupfer et al. in 2002: "All these limitations in the current diagnostic paradigm suggest that research exclusively focused on refining the DSM-defined syndromes may never be successful in uncovering their underlying etiologies" (p. xx).

To illustrate the weakness, let us consider a couple of major DSM categories, schizophrenia and bipolar I disorder, and test them against the new validators. In the set of three predictive validators (all marked as "high priority")—*diagnostic stability, course of illness*, and *response to treatment*—schizophrenia and bipolar disorder score weakly on all three. The tendency toward diagnostic switches between schizophrenia, schizoaffective disorder, and bipolar disorder undermines the validator of *diagnostic stability* (Riecher-Rössler and Rössler 1998; Kupfer 2002). The varied course for both disorders challenges

the validator of *course of illness*, and finally, they both show a varied response to treatment, including responsiveness to the same medications. The other validator marked as high priority, *familial aggregation and/or co-aggregation*, produces a confusing result because with each disorder there is clear familial aggregation, but also aggregation with other disorders. Finally, the validator called *biological markers*, e.g., *molecular genetics* and *neural substrates*, again produces confusing results, inasmuch as hundreds of genetic polymorphisms are involved in these disorders and specific neural substrates have not been confirmed (Craddock et al. 2005, 2006; Hyman 2011).

It is of interest that in 1990 Kenneth Kendler, who was the lead author of the Guidelines report, wrote presciently about the problems in formulating a scientific basis for syndromal diagnoses in psychiatry. He pointed out that the choice of validators and of diagnostic criteria cannot be determined entirely on an empirical basis. The validity of one diagnostic construct versus another will depend on which validators are prioritized. Writing about competing constructs of schizotypal personality disorder, he wrote: "Moreover, empirical data *cannot* determine which of the two is better, for this decision is essentially a value judgment as to which construct of the disorder is more conceptually appealing" (1990: 971). And more generally, he wrote:

> Many important nosology questions in psychiatry are fundamentally nonempirical . . . In many cases, obtaining answers to the little questions will not provide unambiguous answers to the big questions. Before the answers to the little questions can be applied to the big questions, there is an important intervening step. This step often requires answers to other sorts of questions that are usually nonempirical (eg, what are the most important validators for this diagnosis? What are the criteria for defining disorder Q to be a subtype of disorder B? How do we distinguish an Axis I disorder from a Axis II disorder? (1990: 972)

Thus Kendler informed us over 20 years before DSM-5 that syndromal diagnoses within the Robins/Guze framework have their limitations, the main ones being that such syndromal validity is weak, partial, and something of a moving target.

In concluding this section, two remarks are in order. The first addresses the conflict between the two types of syndromal validity. In what I am naming "strong syndromal validity," Kendell and Jablensky argue for one standard (or validator)—zones of rarity (in Robins/Guze terms, clear separation from other disorders). By this standard, virtually all DSM diagnoses are syndromally invalid.

In contrast, the implicit argument of the Guidelines, using the other Robins/Guze criteria, is that validity is not an all-or-nothing phenomenon, and that we can think in terms of partial validity or degrees of validity. (I have used the term

"weak syndromal validity," but that term, as indicated earlier, implies concepts like partial validity and degrees of validity.) There is a resolution of sorts in this conflict in that the weak or partial validity of the Work Groups is covering much the same ground as the "syndromal utility" of Kendell and Jablensky.

In this disagreement between the two types of syndromal validity, there is probably no empirical way to determine which position is correct. The Kendell/Jablensky approach will leave the DSM diagnoses in a validity-less, never-never land. The DSM-5 Guidelines approach will allow us at least weak, partial validity, but a validity that is unstable and shifting. And of course, this dispute can be restated in terms of which Robins/Guze criteria will be prioritized, a question that does not readily admit of an empirical answer.

10.3 Validity and the NIMH Research Domain Criteria (RDoC) project

While the DSM-5 Work Groups were working on their revisions of the DSM diagnoses, a group at the NIMH were developing a unique approach to etiology, the NIMH Research Domain Criteria project (RDoC), that could have an impact on validity (Cuthbert and Insel 2010; Insel and Cuthbert 2009; Insel et al. 2010). Indeed, less than two years before the publication of DSM-5, Kupfer and Regier (2011) published an article in which, recognizing the looming failure of DSM-5 to fulfill its promises, they attempted to link the DSM-5 project to the newly developing NIMH RDoC project. The DSM-5 leaders expressed a hope that DSM-5 might achieve more validity, and the sought-after paradigm shift, through the accomplishments of the RDoC project.

The RDoC project focuses on neural circuitry as the target area of research, with a goal of correlating specific, observable malfunctions of cognitive and psychological functioning with particular disturbances of neural circuitry. In the preliminary working draft the NIMH scientists have identified five major domains of functioning, each containing a number of more specific constructs. In the RDoC matrix, these domains of functioning are represented as rows, while the units of analysis are represented as columns. Neural circuitry occupies one column as a unit of analysis, although the most important in this research project. Other units of analysis, represented in other columns, are genes, molecules, cells, physiology, behavior, and self-reports. (Please see Table 10.1.)

The goal of the project is to produce fundamental, scientific validity through the identification of specific disturbances in neural circuitry associated with discrete areas of psychological and cognitive malfunction. In the language of genetics these discrete areas of psychological and cognitive malfunction can be viewed as endophenotypes, in contrast to the DSM diagnostic syndromes,

Table 10.1 Research domain criteria matrix

Domain Construct	Units of Analysis							
	Genes	**Molecules**	**Cells**	**Circuits**	**Physiology**	**Behavior**	**Self-report**	**Paradigms**
Negative Valence Systems								
Acute threat ("fear")								
Potential threat ("anxiety")								
Sustained threat								
Loss								
Frustrative Non-reward								
Positive Valence Systems								
Reward valuation								
Effort valuation/ Willingness to work								
Expectancy/Reward prediction error								
Action selection/ Preference-based decision making								
Initial responsiveness to reward								
Sustained responsiveness to reward								

Reward learning								
Habit								
Cognitive Systems								
Attention								
Perception								
Visual Perception								
Auditory Perception								
Olfactory somato somatosensory								
Declarative memory								
Language behavior								
Cognitive (effortful) control								
Goal selection								
Updating								
Representation and maintenance								
Response selection, inhibition, or suppression								
Performance monitoring								

Reproduced from NIMH Research Domain Criteria (RDoC), Draft 3.1: June 2011 <http://www.nimh.nih.gov/research-priorities/rdoc/nimh-research-domain-criteria-rdoc.shtml> (accessed May 7, 2014).

which are phenotypes. The RDoC scientists hope to hand the nosologists of the future a list of these endophenotype/neural circuitry correlations, as well as findings from genetics, physiology, and so forth. How these research findings will be assembled into a diagnostic system remains to be seen. The goal is certainly to discover specific neural circuitry disturbances that would represent specific biomarkers with scientific validity for themselves and whatever larger diagnostic scheme they might be part of.

In describing types of validity we can thus include RDoC validity along with strong syndromal (SSV) and weak syndromal (WSV) validity. We should note, however, that RDoC validity is really a form of the strong diagnostic validity described by Kendell and Jablensky as based on defining fundamental characteristics—and not the Kendell/Jablensky strong *syndromal* validity defined by separation from other disorders. In the Robins/Guze framework, RDoC validity fulfills the criteria of "laboratory studies" that tends to trump the other, syndrome-oriented Robins/Guze criteria. What does, however, differentiate RDoC from the fundamental validity described by Kendell and Jablensky is that, while the latter is focused on validity of diagnostic constructs or categories, the RDoC project focuses on signs and symptoms, and tries to correlate these discrete elements of cognition, emotion, and behavior with discrete disturbances of neural circuitry.

In this discussion of diagnostic validity we must, finally, recognize that the NIMH project represents a huge promissory note. Current scientific support for the RDoC approach is minimal. Recent history of psychiatric nosology is replete with unredeemed promissory notes, and it's not obvious that the RDoC project will fare better than its precursors. With this hopeful but guarded attitude toward the project, we remain focused on the WSV approach to diagnosis, as that is the mode in which contemporary psychiatry works.

10.4 Validity in the Face of Complexity

Relevant to all forms of diagnostic validity thus far discussed—SSV, WSV, and RDoC validity—is the issue of complexity. The discussion of validity has proceeded as if complexity were not part of the discussion. In fact, it is, and I will try to explain the role of complexity in this section.

Among the various accounts of complexity (Nicolis et al. 1989; Mitchell 2003, I will focus on the straightforward presentation by Bechtel and Richardson (2010), who describe three levels of complexity: aggregative, component, and integrative.

An *aggregative* system is purely mechanical, as exemplified by a clock. A clock can be taken apart and reassembled. Each component part can be examined as a separate entity that, together with the other parts, produces

the functioning mechanism, the clock. The parts of a clock have no relation to each other except as pieces of a mechanism that, placed together in the proper manner, produce a functioning clock.

Biologic entities cannot be analyzed in this simple, mechanical manner, as they involve higher levels of complexity. What is needed, say Bechtel and Richardson, are two additional levels of complexity in which a particular part cannot be analyzed in isolation from other parts. What a part is and how it works is determined by its relation to the other parts and to the whole system. The whole is understood in terms of the parts, but the parts are also understood in terms of each other and of the whole.

In the simpler form of complex system, termed *component system*, the parts can still be studied independently, despite the fact that their actual functioning will depend on the total organization of the system. In a more complex system, termed *integrated system*, the parts lose their independence and can only be studied as components of the integrated system. What they are and how they work will change with the changing organization of the whole system.

I suggest that psychiatric disorders, like the individuals who suffer from them, cannot be analyzed like clocks. They are disorders of complex, biologic systems. It might be debated whether, in the terminology of Bechtel and Richardson, they are component or integrated systems; but they are certainly complex.

We can further relate the complexity of psychiatric disorders to questions of etiology, diagnosis, and validity. First, etiologic factors interact with one another in a complex manner. Second, this complex etiology can produce heterogeneous presentations and frequent comorbidities. Finally, the complexity of the disorders and their etiologies influences our efforts to classify them, and that inevitably implicates validity.

To follow this line of reasoning, let us take the example of schizophrenia. One of the differences in the larger picture of schizophrenia is bad and good outcome. In the terminology of complexity these divergent presentations are the "wholes" that need to be understood in terms of their "parts," factors such as genes. We know that the genetic underpinnings of schizophrenia are very complicated, with multiple sites and multiple genetic variants. As Hyman wrote, "Robins and Guze developed their view of relatively simple categorical diagnoses at a time when this dizzying level of complexity [in genetics] was barely imagined" (2011: 9). Thus we have to determine how the genetic variants combine to play a role in the heterogeneous presentations of schizophrenia. But we also have to pay attention to how epigenetic factors affect genetic expression—that is, how environmental factors may determine whether a relevant gene is expressed or not. Thus, in analyzing, say, how good outcome schizophrenia (the "whole") is determined by the shared genetic "parts" of this group, we will also have to recognize that elements of the

"whole" (e.g., psychological motivation, behavior, treatment, parental relationship, socioeconomic setting) are affecting how the genetic "parts" are expressed. We now have a situation in which the schizophrenic person is strongly affected by his/her genetic constitution but at the same time affects the genes that are affecting him. We cannot evaluate an individual's genetic constitution apart from understanding how that genetic structure is working in that individual. In the case of a shared presentation, e.g., "good outcome," we will not only look for a shared genetic picture (the part) but also a shared epigenetic/phenotypic picture (the whole). In this example the good outcome would be both a result of the genetic structure and a cause of the genetic structure, and the genetic structure would be a cause of the good outcome presentation and a result of it.

Let me further illustrate complexity in psychiatric diagnosis with two other examples. The first is a recent article by Kendler (2012) in which he analyzes the respective causal variance of schizophrenia, major depression, and alcohol dependence, all displayed in elegant column charts. (Please see Figures 10.1, 10.2, and 10.3). According to Kendler, each of these causal factors is a difference maker, meaning that when an outcome is the result of a variety of contributing factors, a difference maker (or risk factor) is one whose presence or absence changes the probability of the outcome. For each diagnosis the causal variance adds up to 100 percent. Kendler recognizes that he is adding the causal factors

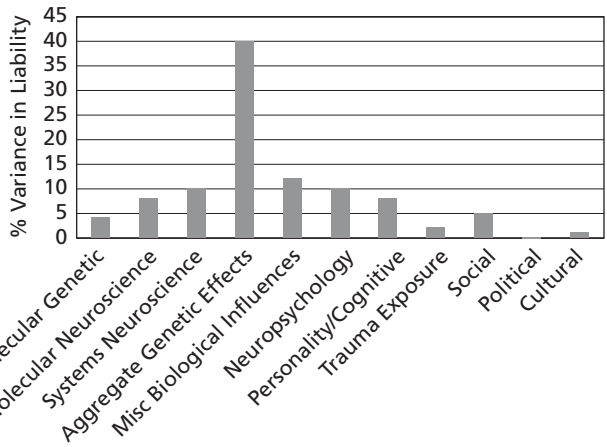

Fig. 10.1 Schizophrenia. Reprinted by permission from Macmillan Publishers Ltd: *Molecular Psychiatry*, 17(4), The dappled nature of causes of psychiatric illness: replacing the organic–functional/hardware–software dichotomy with empirically based pluralism, pp. 377–88, doi:10.1038/mp.2011.182 (c) 2012, Nature Publishing Group.

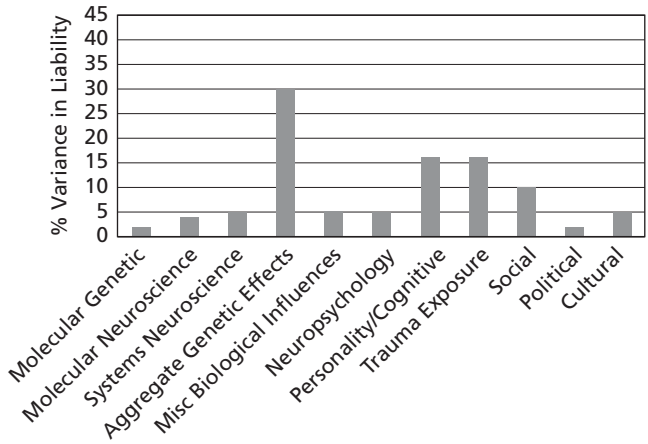

Fig. 10.2 Major depression. Reprinted by permission from Macmillan Publishers Ltd: *Molecular Psychiatry*, 17(4), The dappled nature of causes of psychiatric illness: replacing the organic–functional/hardware–software dichotomy with empirically based pluralism, pp. 377–88, doi:10.1038/mp.2011.182 (c) 2012, Nature Publishing Group.

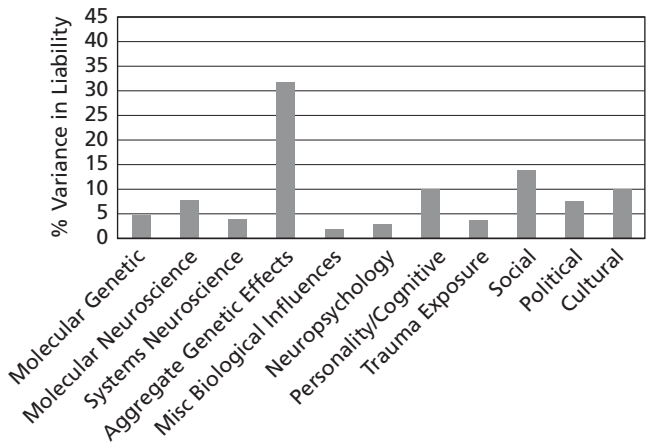

Fig. 10.3 Alcohol dependence. Reprinted by permission from Macmillan Publishers Ltd: *Molecular Psychiatry*, 17(4), The dappled nature of causes of psychiatric illness: replacing the organic–functional/hardware–software dichotomy with empirically based pluralism, pp. 377–88, doi:10.1038/mp.2011.182 (c) 2012, Nature Publishing Group.

in a simple, aggregative manner, as if each is acting independently of the others; and that this presents a false picture of factors that interact with one another. He writes: ". . . for pragmatic reasons, I initially assume an independence of difference-makers that does not exist in nature" (2012: 379). And at the end of the article he concludes:

> The results of the empirically based pluralistic analysis of the causes of SZ, MD and AD reinforce the conclusions from a prior essay that the commonly expressed wish to develop an etiologically based nosology for psychiatric disorders is deeply problematic. Psychiatric disorders are a result of multiple etiological processes impacting on many different levels and often further intertwined by mediational and moderational interactions between levels. It is not possible a priori to identify one privileged level that can unambiguously be used as the basis for developing a nosologic system. (2012: 385)

This study is focused on etiological complexity, but, as just noted, etiological complexity complicates our efforts to develop and validate classifications. For instance, if causal factor x is of a certain strength, but is also affected by other causal factors, and also judged non-empirically to be highly relevant in relation to other factors, how should we combine all this information to make a summary judgment about the "validity" of the diagnostic construct in question?

The second example is the RDoC matrix, as discussed earlier. The matrix describes domains of function as rows, and units of analysis as columns. The cells represent areas of analysis, where a particular subdomain is matched against a particular unit of analysis, e.g., neural circuitry. In its graphical presentation the matrix might look as simple as a clock. But it is not, and that's where the complexity comes in. Imagine taking the subdomain *attention* and matching it against the unit of analysis *circuits*. The area of study for that cell would be what kind of dysfunction in neural circuitry could be associated with a dysfunction in attention. But there are several other columns for studying the dysfunction of attention. Under *genes*, we would study genetic patterns associated with the defect in attention, and under *behavior* we would study the contribution of disturbed behavioral patterns to problems with attention.

Each of the units of analysis could themselves be complex. Under *genes*, for example, the goal would be a straightforward genetic pattern associated with the endophenotype/attentional dysfunction, but in reality we can expect to find several genetic polymorphisms, all affected by epigenetic factors, converging on the deficit in attention. And of course the factors from each of the units of analysis—*circuits, genes, behavior*—may all affect one another, e.g., behavior affecting how genes are expressed. The conclusion is that emphasizing a single unit of analysis (column) is ultimately not a realistic model, since for a particular subdomain of dysfunction the respective units of analysis (columns) will interact with one another, and in various combinations affect the emergence of the deficit. And finally, to make this analysis even more complicated, we should recognize that the various domain (and

subdomain) units (the rows) may be interacting and affecting one another in ways that we don't yet understand. These two examples of etiologic complexity illustrate how such complexity leads to a rather polymorphous view of nosology and validity. We have seen earlier in the chapter how the prioritizing of one validator over another can lead to the choice of one diagnostic construct over another (Kendler 1990). If we now add etiologic complexity, we can see how emphasis on one or another view of etiologic relationships could alter both validators and constructs. Etiological complexity thus worsens the validator and construct ambiguity already highlighted by Kendler in 1990, along with the inevitability of non-empirical decisions in deciding the issues in question.

10.5 The Biomedical Model and its Limitations

The "official" way of classifying in psychiatry is biomedical. Psychiatric disorders and diagnoses are expected to follow the model of the rest of medicine, with psychiatric disorders and diagnoses rooted in biomedical pathology. Ideally, the model would achieve discrete, medically founded, valid diagnoses.

Inasmuch as all forms of validity thus far discussed— strong syndromal validity (SSV), weak syndromal validity (WSV), RDoC, and other fundamental validity—work within the biomedical model, we have taken the model for granted. But are there in fact limitations in the model that bear on the question of diagnostic validity? Keep in mind that biomedical validity for the DSMs has meant adherence to the Robins/Guze standards of validity. Thus questioning limitations in the biomedical model is the same as questioning limitations in the Robins/Guze standards.

The limitation often noted is that many clinical presentations don't fit neatly into one of the biomedically oriented DSM diagnoses, creating problems of NOS diagnoses (now relabeled "unspecified," following the language of ICD-10 and ICD-11), excessive comorbidity, and failed separation from other disorders. From the biomedical perspective, such problems require only further refinement of the biomedical categories and do not represent any inadequacy of the model itself.

The limitation of the biomedical model I want to focus on in this section pertains to psychology. Psychological problems are the elephant in the room of biomedical nosology. The notion that there might be a significant psychological problem or conflict with resolution through some kind of psychological intervention has no place in biopsychiatry. The biomedical message tends to be, please leave your inner life at the door. Indeed, we could say that the DSM is designed for medically oriented psychiatrists. The rest—clinical psychologists, clinical social workers, psychotherapeutically oriented psychiatrists—live with

the manual as they must, but for many it does not reflect their way of understanding their patients nor their way of carrying out their clinical work.

Of course, defenders of the biomedical model would disagree with this statement. To cite one of biomedical psychiatry's pioneers, Samuel Guze articulated the biomedical attitude toward psychological etiology and treatment, arguing that a psychological dimension of treatment may be important as supportive therapy, but that psychological factors are neither causative nor curative (Guze 1989).

To begin a discussion of psychology and the DSM, let us keep in mind that the Robins/Guze criteria and the DSMs have always included psychological symptoms, and that the diagnostic criteria in great part describe such symptoms. But how to understand these symptoms? In a biomedical framework these "mental" or psychological symptoms always reflect an underlying biomedical condition, i.e., the "real" parts. We can question, however, whether symptoms are at times more than surface manifestations of underlying biomedical processes. Take borderline personality disorder (BPD). We have DSM diagnostic criteria describing behaviors such as "unstable and intense interpersonal relationships," feelings such as "emptiness," and the psychologically complex criterion of "identity disturbance." Is identity disturbance merely a surface symptom reflecting an underlying biomedical disorder? Is it not, rather, an indicator of a complicated psychological process—both a symptom and part of the process. To the biomedical psychiatrist who insists that identity disturbance is only a symptom in the sense of a surface manifestation, we might respond, pace Guze and others, that biomedical treatments don't work very well for BPD, and that effective treatment is psychological.

To understand the role of psychological factors in psychiatric disorders, their etiologies, and their treatments, we can reflect back to the previous section on complexity. To stay with the example of BPD, there are multiple etiological factors—biological, psychological, social, etc.—and, as suggested by Kendler in the article previously cited (2012), you can't simply add them up in an aggregative manner. Each affects the etiological power of the others. For BPD the psychological factor (e.g., early trauma) may be strong, but that factor will be affected by biological factors like constitutionally determined sensitivity to stress. The same complexity applies to treatment. For BPD the most effective treatment may be psychological, but for some patients medications may be very helpful, and in that situation the two forms of treatment interact in a complex manner, the medication supporting the psychotherapy and vice versa.

Let me illustrate these points with a case example. Linda, a single woman in her early forties, presented to me with symptoms that clearly warranted a diagnosis of PTSD. She did, however, also meet criteria for generalized disorder

and borderline personality disorder. Her array of symptoms was likely related to early sexual abuse, of which she was only minimally aware at the onset of treatment, as well as to a non-supportive parental background. The patient was educated as a medical doctor, but with the burden of intrusive flashbacks and anxiety symptoms, she found it very difficult to sustain the responsibilities of practicing as a physician, and would for periods of time stop working as a physician and do other medical work for which she was qualified.

The same problems encumbered her personal life. Issues of anxiety, distrust, and self-doubt prevented her from forming the kind of stable, intimate relationship she desired.

The treatment has been primarily psychotherapy, focused both on the early abuse and the current symptoms and life problems. Medication has not been very helpful and has been only a minimal aspect of the treatment. She has been in treatment for about three years and has made slow, steady progress; she spends more time working as a physician, and she tolerates better the anxiety of being in a relationship.

This woman illustrates the complexity of etiology, treatment, diagnosis, and psychology in psychiatry. Etiology includes the interweaving combination of early sexual abuse, poor parental support, and, probably, an inborn tendency toward heightened anxiety. Treatment has been primarily psychological, with minimal help from medications. Diagnosis is confusing, as indicated earlier. Of the three DSM diagnoses mentioned, we could prioritize one (e.g., PTSD) and name the other two as comorbid diagnoses. Or we could pick one diagnosis and argue that all the symptoms could be included within that diagnosis. Here we face the dilemma described earlier of making diagnostic decisions on a non-empirical basis. Finally, we are dealing with a case in which psychological factors play a significant role—in etiology, presentation, and treatment. Regarding presentation, we recognize psychological symptoms as well as psychological conflicts. The latter are multiple: conflicts over accepting the reality of the abuse, over facing her parents' failures, over tolerating the anxiety and other symptoms when working as a physician and developing a relationship, over transforming her self-image from impaired victim to competent adult, and of course over accepting the treatment and the treater as helpful rather than as causing more symptoms. We have worked on all these conflicts with understandably slow progress.

I invoke this patient as someone whose problems involve significant psychological factors in etiology, presentation, and treatment. Guze and others would not countenance as a psychiatric disorder a case where etiology and treatment are to a significant degree psychological, as opposed to "biomedical." The irony of this case is that the patient does fit more than one of the standard DSM

diagnoses. It's just that the respective diagnoses have to be understood more complexly than the strictly biomedical model would allow.

To conclude this section, let me summarize the argument. Psychological etiology and psychological treatment exist on a continuum in psychiatric disorders. At one end of the spectrum, e.g., schizophrenia, psychological etiology is minimal and psychological treatment is supportive and rehabilitative. In the middle of the continuum are the many psychiatric disorders, e.g., depressive disorders, in which the degree of psychological etiology varies from one patient to another, and psychological treatment is supportive and at times may include insight-oriented psychotherapy. At the other end of the continuum, as with Linda, psychological etiology is stronger, and psychological treatment may be primary.

As the psychological dimensions of etiology and treatment become stronger, they challenge both the biomedical model and the diagnostic validity based on it. We discuss both in the following section.

10.6 **Discussion**

The discussion of validity in this chapter has taken the following course. I began with the failure of the DSMs (including DSM-5) to meet the Robins/Guze standards of diagnostic validity in psychiatry. From there we moved to a distinction between strong (SSV) and weak (WSV) syndromal validity, the first, as developed by Kendell and Jablensky, highlighting the Robins/Guze criterion of separation from other disorders as the single standard for strong syndromal validity, the second, as developed in the DSM Guidelines, highlighting the other Robins/Guze criteria for partial, weak validity (the Robins/Guze criterion of "laboratory studies" has played a minimum role for both Kendell and Jablensky and the Guidelines, since for most DSM diagnoses such studies have not yielded anything like a definitive biomarker).

Given the inability of DSM nosology to achieve SSV, we have been left with WSV as the current standard of syndromal validity. A major consequence of this standard is that syndromal validity is vulnerable to the non-empirical prioritizing of one validator or diagnostic construct over another, thus rendering validity a moving target.

As a next step in the argument I introduced the RDoC project, which promises to bypass syndromal validity and achieve the Robins/Guze criterion of "laboratory studies" (described by Kendell and Jablensky as "fundamental validity"). Given that the RDoC project remains a promissory note, it is not in a position at this time to replace WSV as the standard of validity.

I next introduced the concept of complexity in psychiatric diagnosis. Although primarily focused on the complex etiologies of psychiatric disorders, complexity

inevitably involves presentation, diagnosis, and validity. The effect of recognizing complexity in the diagnostic process is that it renders weak syndromal validity even more complicated (e.g., validators interacting with one another), and still more vulnerable to shifting conclusions about diagnostic validity.

Finally, I took the question of complexity a step further in pointing out that the preceding discussion of diagnostic validity, developed in terms of the Robins/Guze criteria, had been framed within the biomedical model assumed by Robins and Guze. I questioned the limitations of that model, especially its resistance to including psychological phenomena in the etiology, nosology, and treatment of psychiatric illness. Inasmuch as the preceding discussion of diagnostic validity was predicated on an assumption of the biomedical model, any challenge to that model would involve consequences for our notions of validity.

Let us now follow the next steps in this train of thought. We have seen two problems with diagnostic validity in psychiatry. The first is that the prevailing standard of weak diagnostic validity (WSV) involves non-empirical decisions about which validators to prioritize, and thus what is to count as a valid diagnostic construct. The second is that the biomedically oriented DSMs allow insufficient attention to psychological dimensions of etiology, nosology, treatment, and validity.

How might we address these two problems, and perhaps relate them? Since WSV is already by necessity flexible in its attitude toward diagnostic constructs and validity, is there any reason not to make it still more flexible and include the missing psychological aspects in WDV diagnoses? Allowing such phenomena into WSV nosology, of course, would require a framework larger than the Robins/Guze biomedical framework—or perhaps an expansion of what a biomedical model is.

Let me focus on the latter possibility, proposing that we do not need another model, biopsychosocial or whatever, so much as a *complete* biomedical model that understands both the complexity of psychiatric disorders and the fact that such complexity includes psychological dimensions not included in the Robins/Guze biomedical framework. In that regard, we have already noted that the diagnostic criteria for some DSM-5 diagnoses contain psychological symptoms that are more than simple markers for underlying biomedical diseases, and that such criteria and symptoms suggest psychological problems and potential psychological treatment. Thus, for diagnoses like BPD and PTSD, DSM-IV had already expanded beyond what would be allowed in strict adherence to the Robins/Guze biomedical framework. Regarding treatment, a complete biomedical model would recognize that there is not biomedical treatment *and* psychological treatment; there is biomedical treatment that *is* psychological as well as "medical."

The philosophical point of this proposed revision of the biomedical model is that subjective or mental symptoms are not always surface manifestations of an underlying biomedical disease process; they may also be the surface manifestations of an underlying psychological process. What makes psychiatry complex is that symptoms are often both, as in many depressive disorders. Psychological problems do not exist without their biology, and biological conditions do not exist without their psychology.

When Kupfer et al. lamented in 2002 that validity would not be achieved with the current Robins/Guze DSM paradigm and would require a new paradigm, they certainly did not have in mind the mini-shift suggested here. The latter is simply a recognition that a syndromal diagnosis could be something more than a symptom cluster representing underlying biomedical disease, and that an expanded biomedical model could include psychological dimensions within WSV.

Regarding my proposal and the structure of the DSM manual, we could imagine creating new, psychologically oriented diagnoses, or simply reformulating the existing sets of diagnostic criteria to include psychological factors. As indicated earlier, the current diagnostic criteria for BPD already describe psychological symptoms that at least suggest underlying psychological disturbance. On the other hand, the diagnostic criteria for major depression present psychological symptoms that seemingly assume underlying biomedical illness. The controversy over the grief exclusion in DSM-IV—and its removal in DSM-5—suggests a reluctance to specify psychological factors in the underlying disorder.

Finally, any restructuring of the diagnostic constructs and diagnostic criteria to include a richer psychological aspect would have some effect on diagnostic validity. In the context of weak syndromal validity (WSV), the diagnostic constructs, already complex, would be even more complex.

10.7 **Conclusion**

The index of DSM-5 does not have a single entry for the word "biomedical." Use of that term in this chapter is then not warranted by its presence in the manual. Rather, use of the term is justified by the fact that the assumptions of the Robins/Guze biomedical model are so deeply embedded in the DSMs that they don't need to be mentioned. The goals of psychiatric research remain, as indicated by Kupfer et al. at the beginning of the DSM-5 era (2002), to fill in the Robins/Guze "standard of 'laboratory studies'" with neuroscientific findings and the infamous biomarkers, replacing WSV not with SSV, but with fundamental validity. In this chapter I have proposed an inclusion within WSV of psychological factors, and with that an expansion of the biomedical model beyond the narrow

Robins/Guze standards. Such a change would leave the DSM in the territory of WSV, and would do nothing to achieve fundamental validity. What it would do is make the DSM more useful for psychologically oriented clinicians.

Meanwhile, the architects of the RDoC project, in their quest for fundamental validity, define psychiatric disorders as "brain disorder," and attempt to locate the disorders in neural circuitry. In this RDoC world psychology would again be minimized. The RDoC matrix does have an analysis/column marked "psychological," but it's unclear what that might mean other than psychological, surface manifestations of the disturbed circuits—thus no different from the traditional biomedical framework as described earlier. The challenge in this new/old order of brain diseases would be to again find a place for psychology in the diagnostic framework.

References

American Psychiatric Association (1980). *Diagnostic and Statistical Manual of Mental Disorders (Third Edition)*. Washington, DC: American Psychiatric Association Press.

Bechtel, W. R., and Richardson, C. (2010). *Discovering Complexity. Decomposition and Localization as Strategies in Scientific Research*. Cambridge, MA: MIT Press.

Clarke, D., Narrow, W., Regier, D., et al. (2013). DSM-5 field trials in the United States and Canada, Part I: Study design, sampling strategy, implementation, and analytic approaches. *American Journal of Psychiatry*, **170**, 43–58.

Craddock, N., O'Donovan, M., and Owen, M. (2005). The genetics of schizophrenia and bipolar disorder: dissecting psychosis. *Journal of Medical Genetics*, **42**, 193–204.

Craddock, N., O'Donovan, M., and Owen, M. (2006). Genes for schizophrenia and bipolar disorder? Implications for psychiatric nosology. *Schizophrenia Bulletin*, **32**, 9–16.

Cuthbert, B., and Insel, T. (2010). The data of diagnosis: New approaches to psychiatric classification. *Psychiatry*, **73**, 311–14.

Cuthbert, B., and Insel, T. (2010). Toward new approaches to psychotic disorders: The NIMH Research Domain Criteria project. *Schizophrenia Bulletin*, **36**, 1061–2.

Detre, T. (1987). The future of psychiatry. *American Journal of Psychiatry*, **144**, 621–5.

Feighner, J., Robins, E., Guze, S., et al. (1972). Diagnostic criteria for use in psychiatric research. *Archives of General Psychiatry*, **26**, 57–63.

Guze, S. (1989). Biological psychiatry: Is there any other kind? *Psychological Medicine*, **19**, 315–23.

Hyman, S. (2011). Diagnosis of mental disorders in light of modern genetics. In D. Regier, W. Narrow, E. Kuhl, and D. Kupfer (eds), *The Conceptual Evolution of DSM-5*. Washington, DC: American Psychiatric Publishing, 3–18.

Insel, T., and Cuthbert, B. (2009). Endophenotypes: bridging genomic complexity and disorder heterogeneity. *Biological Psychiatry*, **66**, 988–9.

Insel, T., Cuthbert, B., Garvey, M., et al. (2010). Research Domain Criteria (RDoC): Toward a new classification framework for research on mental disorders. *American Journal of Psychiatry*, **167**, 748–51.

Kendell, R., and Jablensky, A. (2003). Distinguishing between the validity and utility of psychiatric diagnoses. *American Journal of Psychiatry*, **160**, 4–12.

Kendler, K. (1990). Toward a scientific psychiatric nosology: strengths and limitations. *Archives of General Psychiatry*, **47**, 969–73.

Kendler, K. (2012). The dappled nature of causes of psychiatric illness: Replacing the organic–functional/hardware–software dichotomy with empirically based pluralism. *Molecular Biology*, **17**, 377–88.

Kendler, K., Kupfer, D., Narrow, W., et al. (2009). Guidelines for Making Changes to DSM-V Revised 10/21/09. Available at: <http://www.dsm5.org/ProgressReports/Documents/Guidelines-for-Making-Changes-to-DSM_1.pdf>, (accessed January 22, 2014).

Kupfer, D., First, M., and Regier, D. (eds) (2002). *A Research Agenda for DSM-V*. Washington, DC: American Psychiatric Association Press.

Kupfer, D., and Kraemer, H. (2012). What we Learned from the DSM-5 Field Trials. *Psychiatric News* (December 16, 2012).

Kupfer, D., and Regier, D. (2011). Neuroscience, clinical evidence, and the future of psychiatric classification in DSM-5. *American Journal of Psychiatry*, **168**, 1–3.

Mitchell, S. (2003). *Biologic Complexity and Integrative Pluralism*. Cambridge: Cambridge University Press.

Nicolis, G., Nocolis, G., and Progogine, I. (1989). *Exploring Complexity: An Introduction*. New York: W. H. Freeman and Company.

Pies, R. (2007). How objective are psychiatric diagnoses? *Psychiatry*, **4**, 18–22.

Regier, D., Narrow, W., Clarke, D., et al. (2013). DSM-5 field trials in the United States and Canada, Part II: Test–retest reliability of selected categorical diagnoses. *The American Journal of Psychiatry*, **170**, 59–70.

Regier, D., Narrow, W., Kuhl, E., et al. (eds) (2011). *The Conceptual Evolution of DSM-5*. Washington, DC: American Psychiatric Publishing.

Riecher-Rössler, A., and Rössler, W. (1998). The course of schizophrenic psychoses: What do we really know? A selective review from an epidemiological perspective. *European Archives of Psychiatry and Clinical Neuroscience*, **248**, 189–202.

Robins, E., and Guze, S. (1970). Establishment of diagnostic in psychiatric illness: Its application to schizophrenia. *American Journal of Psychiatry*, **126**, 983–987.

Spitzer, R., Endicott, J., and Robins, E. (1978). Research diagnostic criteria: Rationale and reliability. *Archives of General Psychiatry*, **35**, 773–82.

Part 3

Matters (slightly) more clinical

Chapter 11

The importance of structural validity

Kathryn L. Jacobs and Robert F. Krueger

11.1 Introduction

In this chapter, we will be discussing the role of structural validity in current psychiatric nosology. Structural validity, as we define it, is how closely the organizational structure of a set of definitions of psychiatric disorders matches how the disorders present in patient samples. We will discuss how structural validity has been neglected in the current nosology in favor of what we label as external validity, and what the possible consequences are in regards to the diagnosis and treatment of psychiatric disorders. We will then suggest possible changes that could be made to the current nosology to improve the structural validity of psychiatric diagnoses.

The first step in discussing current psychiatric nosology is to examine how it was initially developed. We therefore choose to focus first on reliability, and the role it has historically had in shaping psychiatric nosology. We believe that a discussion of the significant influence that reliability had in the formation of current diagnostic categories will give our readers a base of knowledge necessary to move forward into the discussion about structural validity.

11.2 Reliability

Reliability is often a chief concern in the development or refinement of a diagnostic system. In the case of the development of the different iterations of the DSM, this is apparent when looking back at previous editions. In the first and second DSM publications, disorders were described in a very literary style in paragraph form. Diagnoses involved matching patients with the best fitting description. This made diagnoses highly subjective and hard to replicate from one clinician to another (Spitzer et al. 1978). An unreliable diagnostic system means that a patient cannot be sure of the diagnosis they are given, as it could change from clinician to clinician. This instability in diagnoses can complicate treatment, as different diagnoses presumably have different optimal treatments.

The shift toward greater reliability can be seen in the DSM-III, DSM-IV, and DSM-IV-TR. In these later editions, descriptions of mental disorders shifted away from the paragraph form seen in earlier publications. The new format presented mental disorders as polythetic-dichotomies. Mental disorders in the early versions of the DSM had previously been dichotomous, meaning that a patient was diagnosed with either having a given disorder or with not having it; there was no possible middle ground. The polythetic approach to mental disorders, however, was introduced in the DSM-III, and changed the way that mental disorders were described. This approach was applied to many of the new diagnostic categories described in the DSM-III, and carried over into the DSM-IV and DSM-IV-TR (text revision). In this new description, in order to have a given disorder, a patient had to present with a certain number of symptoms out of a larger group. For example, in order to be diagnosed with a major depressive episode, a patient would have to present with at least five of nine listed symptoms, including "depressed mood most of the day" and "markedly diminished interest or pleasure" (fourth ed.; text rev.; DSM-IV-TR; American Psychiatric Association 2000). The severity of the symptoms was not taken into account, nor was any specific combination of symptoms beyond the two required symptoms mentioned above. There is a specific boundary (from four to five symptoms) that a patient must cross before they can receive the diagnosis. The rigid use of these boundaries in clinical practice cut diagnoses into distinct, non-overlapping categories.

This new format was designed to improve the reliability of the DSM diagnoses, as it reduced the number of subjective decisions that a clinician had to make in regards to a patient (Grove et al. 1981). These reductions in subjectivity led to greater agreement amongst clinicians when given a particular patient to diagnose (Lobbestael et al. 2011). In specifying the definitions for mental disorders to a distinct set of required symptoms, the reliability of the diagnoses was improved.

11.3 Limits of External Validity

This new system, created in order to enhance reliability in diagnoses of mental disorders, focused on external validity to support its new definitions. In our definition of external validity, we are referring to judging diagnoses by how they predict external criteria (e.g., functioning). This is of course an important consideration in diagnosing a mental disorder, but is not entirely sufficient. This is because diagnostic concepts can predict external variables, yet still not be organized in a way that reflects the empirical organization of disorders. The shift in format from paragraph descriptions to polythetic-dichotomies in the

DSM made clear to clinicians what to focus on when considering a diagnosis. This was a major step forward in the mental health field, and helped clear up many ambiguities in definitions of mental disorders. This approach used external validity as the benchmark for judging the quality of these new definitions.

For a more concrete example, consider again the diagnosis of a major depressive episode. As noted before, in order to receive a diagnosis of a major depressive episode, a patient must present with five of the nine given symptoms listed in the DSM-IV-TR. In order to judge this diagnosis in terms of external validity, one would have to examine the patient's life outside of the clinic. Do they have impaired functioning? Are multiple aspects of their life affected, such as work, home, and school? If they answer yes to these questions, one might judge the diagnosis of a major depressive episode as valid. This kind of impairment is what a clinician would expect given a diagnosis of a major depressive episode; therefore, the diagnosis seems to fit.

There is one central problem with using external validity as the primary support for a current diagnostic system. This problem is that external validity uses circular reasoning to back up its claims. A diagnostic construct is considered valid when it can be shown that the patients present with the expected clinical impairments, but those impairments are only expected because that is how the diagnosis was conceptualized in the first place. Many different diagnoses—mental disorders as seemingly different as major depressive episode and cocaine intoxication—can present with very similar impairment symptoms (work functioning, sleep disturbances). Yet despite being different diagnoses, these same external correlates can be used as support for a diagnosis of either (fourth ed.; text rev.; DSM-IV-TR; American Psychiatric Association 2000). This example illustrates the problems inherent in using external validity as a sole source of support for a diagnostic system. External validity is too broad to be used as a primary means of validating a classification system. While it is useful for judging which cases should be included in the broader category of "mental disorders," alone it is insufficient to validate which specific diagnosis a given patient should receive.

11.4 **Structural Validity**

If external validity alone is not enough to validate a set of psychiatric diagnoses, then what can be done to improve these diagnoses? We believe a plausible solution would be to look at structural validity. The definition for structural validity originated in a 1957 monograph written by Jane Loevinger. In it, Loevinger describes a type of validity focused on evaluating and improving psychological test structure. Examining the structural validity of a test would involve looking at

the test questions and how they correlate with one another, and comparing those correlations to real-world behaviors. If the behaviors which correlated in real life corresponded to correlating test questions, then the test is structurally valid.

For example, consider a psychological test designed to measure aggression. For the purpose of this example, we will define aggression as a single, discrete concept, rather than a diffuse family of behaviors. In real life, one might expect a number of physically aggressive acts committed against a romantic partner in a given period of time to correlate positively with the number of verbal threats made to co-workers. In this example, in order to have a structurally valid test, the test questions aimed at measuring romantic aggression should correlate positively with the test questions aimed at measuring co-worker aggression.

In our case, we will be using a slightly different definition of structural validity. The idea is analogous to that of Loevinger, but has been modified somewhat to be relevant to the nosology of mental disorders rather than tests. Structural validity, as presented in the remainder of this chapter, is how closely the organizational structure of a set of definitions of psychiatric disorders matches how the disorders present themselves in clinical samples.

For an example, consider the diagnoses of Major Depressive Disorder (MDD) and Generalized Anxiety Disorder (GAD). In current organizational structure, these are defined as separate, unrelated illnesses. The recent publication of the DSM-5 has arranged the disorders to be located next to each other in the text, but they are still considered separate diagnoses (American Psychiatric Association 2013). To examine the structural validity of these diagnoses, one must look at how they manifest in patient populations. A study conducted via survey to examine comorbidity found the prevalence of MDD in a general population to be 8.5 percent (Kessler et al. 1999). Generalized Anxiety Disorder occurred at a rate of 1.3 percent (Kessler et al. 1999). According to probability theory, the joint probability of any two independent events is the probability of the first multiplied by the probability of the second. Given the individual probabilities listed above, if MDD and GAD were indeed independent, one would expect the prevalence of patients with co-occurring MDD and GAD to be, at most, 0.085 * 0.013, which is equal to 0.0011 or 0.11 percent. The actual comorbidity, according to the survey, was 2.3 percent, approximately 20 times the expected rate (Kessler et al. 1999).

This seems to indicate that the definitions of MDD and GAD need to change if they are to be considered structurally valid. We will explore a possible alternative for the diagnoses of MDD and GAD later on in this chapter. By separating disorders and making them as specific as possible, the current nosological system for psychiatric disorders fails to consider structural validity, instead opting for external validity to support its definitions.

11.5 Where is Structural Validity in Current Psychiatric Nosology?

We mentioned in the previous section that Loevinger's definition of structural validity was originally written to examine the validity of psychological tests. Her definition of structural validity fits with the manner in which psychological tests are developed. In Loevinger's definition, structural validity is determined by looking at how the test structure correlates with how behaviors are structured in the real world. When psychological tests are first developed, researchers come up with a broad array of questions that could possibly relate to the psychological construct being tested. They use these questions with an initial group of subjects, and then narrow down the question pool by looking at the correlations between the answers and the real-life behavior they are trying to study.

In test development, structural validity is defined as "the degree to which scores of a questionnaire are an adequate reflection of the dimensionality of the construct to be measured" (Elbers et al. 2012). This type of validity is conceptually different from external validity. In external validity, a diagnosis is validated simply based upon the fact that it correlates with expected external criteria (e.g., functioning), without consideration as to the structure of these external correlates. In considering structural validity, researchers and clinicians must also focus on the underlying structure of the diagnoses, and whether or not the patterns in the diagnostic space match the patterns seen in patient populations.

Structural validity is considered very important in test development. This is seen in a review of a self-report questionnaire examining fatigue in patients suffering from Parkinson's disease. Elbers et al. (2012) examined 31 different questionnaires focusing on fatigue in patient populations. The researchers evaluated the different questionnaires based on a number of criteria with the goal of more clearly defining fatigue in a clinical setting. In this evaluation, structural validity, or how well the scores of the questionnaire are an adequate reflection of the dimensionality of fatigue as a construct, was considered crucial in order to consider the questionnaire a good indicator of fatigue symptoms.

This method of test development is a data-driven method, meaning that the test structure changes accordingly when unexpected correlations are found in the initial test subjects. Naming of new psychiatric disorders, on the other hand, is an expert-driven approach. This means that instead of drawing from data to develop criteria, experts in the field create delineation between symptoms as they perceive them to be arranged in real life.

For example, consider a disorder that has been conceptualized and studied by a small group of researchers. This disorder is characterized by the intense

desire to have one's limb(s) amputated (First 2004). The limbs are not diseased in any way, and the researchers claim that the desire is not systematically related to any documented sexual fetish. In the consideration of adding this desire for amputation as a separate disorder to future publications of the DSM, we can see the expert-driven approach taken to naming new psychiatric disorders. In this case, a small group of researchers noticed a trend among a group of patients. This trend was the desire to have a healthy limb removed. The researchers then sought out other people who shared this desire. They posted on forums, searched chat rooms, and asked current patients if they knew of anyone else who shared their desire for amputation. In doing so, they amassed a small group of people who seemed to share similar symptoms. The researchers categorized this disorder, considering it separate from any disorder included in the current nosological system, and proposed that it be considered for inclusion in future nosology.

There are several differences between this expert-driven approach seen in the proposal of the addition of an amputation-affinity disorder and the data-driven approach used to create new psychological tests. In the development of tests, a battery of questions is created with the goal of capturing all aspects of a construct. The construct is then refined based upon the patterns of correlations (between items, and between items and behaviors) that occur in the population.

In contrast, in the expert-driven development of the definition of the amputation disorder, researchers actively sought out patients who showcased the set of symptoms that the researchers were *looking for*. The definition or categorization of the amputation disorder was not changed based on varying symptoms in a large group of people, because anyone who displayed symptoms that were not consistent with what the researchers were looking for was excluded from the study.

The problem with this expert-driven method is that it can miss many of the intricacies and variations in human behavior, normal and abnormal. By artificially delineating between people who have an affinity for amputation and people who do not (according to the definitions they themselves created), researchers miss out on a possible spectrum of thoughts present in subjects not considered for the study. If there was a person who had once considered amputation, but had decided against it, they would not be included anywhere in the scientific documentation, even though this could be an important middle ground in studying the phenomenon of feeling detached from one's own limbs. Instead, researchers looking to name a new psychiatric disorder pick only those subjects who fit the description that the researchers themselves created.

As a contrast, consider an example of how to approach this diagnosis from a data-driven, structurally valid perspective. In this approach, researchers or clinicians looking to create a new diagnosis would need to look at a much

broader range of the population. Instead of focusing only on those who are currently considering voluntary amputation, they would broaden their search to, for example, people who had once considered amputation, people who felt separated from their limbs, and any number of other related criteria that the researchers proposed. They would then collect data on all of the subjects exhibiting these symptoms, including data on all other psychiatric symptoms not included in the original data set. This data could include possible depressive symptoms, anxious symptoms, and any other psychiatric symptoms of note. The researchers would then look at the structure of the data in the sample collected, examining correlative trends between symptoms. This would allow the researchers to discover if this diagnosis was simply a part of a larger group of symptoms (like anxiety), or if it did indeed represent a separate and distinct set of symptoms warranting a new diagnosis.

11.6 Why is a Focus on Structural Validity Necessary?

To further explore the importance of a structurally valid system of nosology, we will focus on the definitions of personality disorders in the DSM-IV-TR and DSM-5. The personality disorders, like many of the other diagnoses in the DSM-IV-TR, were treated as polythetic-dichotomies. As described earlier, a polythetic-dichotomy is an organizational system in which a diagnosis is either present or not (there is no middle ground), and a certain number of criteria have to be met in order to qualify for the diagnosis. Even if, as mentioned in the DSM-IV-TR, the diagnoses were not meant as hard, non-negotiable categories (fourth ed.; text rev.; DSM-IV-TR; American Psychiatric Association 2000), clinicians and research often reified these categories in practice, despite there being evidence that such reifications were premature, given the state of our understanding of mental disorders (Hyman 2010).

In the case of the personality disorders, this organizational system proved to be particularly troubling to many in the field. The personality disorders in the DSM-IV-TR were split into ten categories a priori, a pattern which does not seem to represent what is found in nature (Krueger et al. 2011a; Widiger et al. 2009). These boundaries, inconsistent with what seems to occur naturally, lead to large comorbidity between different personality disorders (Skodol et al. 2011), as well as frequent use of the Not Otherwise Specified (NOS) category (Widiger et al. 2005). This lack of specificity leads to poor coverage in the field of personality disorders, leaving 40 percent of patients with a PD not covered by current criteria boundaries (Livesley 2012). This inconsideration of structural validity is beneficial to neither patients nor researchers, as a substantial portion of the clinical population is being either missed or misrepresented. Through the

pursuit of data-derived diagnoses, the DSM could have its structure reflect the natural organization of mental disorders.

11.7 Consequences of Structural Invalidity

One possible consequence of a structurally invalid nosology is that actual correlations between diagnoses are hidden because clinicians believe that these diagnoses are separate entities, leading them to ignore areas of overlap. As mentioned in the previous section, current nosology and improper use of the DSM in regards to personality disorders has led to not only a very high comorbidity rate, but also a high percentage of patients categorized as having a Personality Disorder Not Otherwise Specified (Widiger et al. 2005). This suggests that these categories are not properly representing how personality disorders are structured in the population. If these disorders were indeed unrelated, such a high comorbidity rate would not occur.

Use of the DSM verbatim, without consideration as to the underlying intentions of the authors, has the potential to be extremely costly to the scientific community. If a system of classification is not examined for structural validity, many years and millions of dollars in grant money could be wasted examining differences between diagnoses that in reality show no distinct boundary. This is especially true if research groups exclude subjects who exhibit comorbid diagnoses. If two diagnoses are related, showing high comorbidity rates, excluding subjects who are comorbid for both diagnoses artificially separates subjects into two groups that should be more closely linked. If new diagnostic criteria are created by expert opinion rather than data driven, possible links are more likely to be missed if the said experts are used to seeing only one type of patient.

All of these negative consequences of a structurally invalid nosology hurt not only researchers, but patients themselves. Consider from our examples, a patient who displays both Avoidant Personality Disorder and Dependent Personality Disorder. Depending on the clinician that the patient visits, they may receive a diagnosis of a disorder of Avoidant PD, a diagnosis of Dependent PD, both, or neither. Their diagnoses may change depending on which clinician they see, when they are seen, or what symptoms they choose to highlight when being interviewed. This burden of uncertainty and multiple disparate diagnoses does not help the patient to understand their mental illness, and is not helpful in leading towards a treatment. Given this example, one can see how having a structurally valid and concise diagnosis containing the symptoms shared by both personality disorders could help greatly in reducing the stigma associated with multiple diagnoses, as well as helping in developing more specified treatment options.

11.8 Developing a Structurally Valid Nosology

From the previous examples given, one can see that having a structurally valid nosology would be greatly beneficial to the field of mental health. Having the terminology of diagnoses follow the patterns that are displayed by the data would indicate a more structurally valid nosology. However, given how data is collected, one can see that this goal is not easy to accomplish. In order to begin data collection, researchers must have a hypothesis in place to build upon. In the past, this has led to data collection aimed at confirming mental health diagnoses that were initially developed by expert opinion. This limits the type of data that can be collected, as the data will follow the patterns that are laid out by the diagnoses delineated in the hypothesis. In order to create a more structurally valid system of diagnoses, a more fluid system of diagnostic development is needed. Taking from the methods of test development described earlier in this chapter, researchers can use available data to modify the current system of nosology into a system more focused on structural validity.

11.9 How can Structural Validity be Achieved in Psychiatric Nosology?

With the recent development of the DSM-5, many researchers have been addressing the question of how to develop a more structurally valid system of nosology. In regards to personality disorders, one of the large changes was made based on the fact that patterns of comorbidity in PDs mimics patterns seen in personality traits (Krueger et al. 2011b). Using this information, work group members developed a new "trait based" system to categorize personality disorders (American Psychiatric Association 2013). Rather than representing the symptom space with ten categorically distinct syndromes (borderline, histrionic, etc.), it is modeled using a profile of twenty-seven pathological traits (emotional liability, anxiousness, etc.) that can be grouped into six broader domains (e.g. negative affectivity). This new system both explained a good portion of the variance in the DSM-IV PDs, as well as providing incremental information about an individual's disorder using a measure of severity of impairment (Hopwood et al. 2012).

This new trait-based system for personality disorders presented in the DSM-5 is a large step forward for those interested in creating a structurally valid nosological system. However, there are still some problems with the way the DSM-5 presents personality disorders. The personality disorders are split into two sections, with the new trait-based system printed in a later section of the book, labeled as the "alternative model" (American Psychiatric Association 2013). Written along with all of the other diagnoses are the original ten

personality disorders, reprinted from the DSM-IV-TR. This poses a problem, as the empirically flawed DSM-IV diagnostic criteria are presented as a valid diagnostic system. In order to get closer to a structurally valid system of nosology, current terminology needs to be allowed to change in the direction that is informed by the data available.

As a possible example for how structural validity could inform the organization of mental disorders in future DSM publications, consider the example from earlier in the chapter involving Major Depressive Disorder and Generalized Anxiety Disorder. As stated earlier, these two disorders are considered separate and distinct by current DSM nosology. Despite being considered independent, MDD and GAD co-occur at a rate much higher than would be expected by chance. In a structurally valid nosology, this overlap would have to be taken into account. For example, a new organizational structure could focus on a general pool of both depressive and anxious symptoms. These symptoms would be weighted by severity, with more severe symptoms considered to be of more importance. A patient would be rated on the number and severity of their symptoms, and given two scores.

The first score would represent the type of symptoms present (on a scale of depressive–anxious, with a midpoint representing an even ratio of depressive to anxious symptoms). The idea of combining depressive and anxious symptoms was supported by Goldberg (2010), who suggested that considering the two categories simultaneously could increase the effectiveness of treatment in a clinical setting. Goldberg proposes that a diagnosis such as "anxious depression" could be given in an applicable situation, saying that failing to take into account the anxiety of a clinically depressed patient may lead to improper treatment.

The second score would represent severity (from mild to severe). The idea of dimensional ratings based on severity was supported in Brown and Barlow (2009). The authors stated that by keeping current categories of mental disorders intact, meanwhile adding a dimensional assessment of severity, would allow diagnoses to be more accurately based on data while keeping the clinical utility of hard categories (Brown and Barlow 2009). This proposed system would better take into account the large overlap between depressive and anxious symptoms, while still preserving the fact that there are important differences between the two categories.

This example could also be considered on a broader scale. In their review, Barlow et al. (2013) highlight the underlying role of neuroticism in a large cluster of diagnoses represented in the current DSM, including depressive and anxiety disorders. They suggest that treatment of neuroticism directly via much broader treatment methods may benefit a large patient population. This method of treatment, like the hypothetical example presented earlier, incorporates the

information about the high comorbidity rates seen in depressive and anxious disorders by targeting the personality style linking these diagnoses together.

11.10 Conclusion

We believe structural validity to be extremely important to consider when developing and evaluating psychiatric nosology. Current approaches to the development of mental disorder definitions focus primarily on reliability and external validity, which has led to diagnoses with considerable clinical overlap. This overlap, with many patients receiving two or more diagnoses, could conceivably lead to stigmatization, increased health care costs, and confusion regarding the mental health care field in general. It is therefore imperative that the organizational structure of mental disorder definitions matches as closely as possible to how the symptoms present in a research setting. The expert-driven approach to diagnoses, with experts providing definitions by consensus, was abandoned years ago by the medical community (Sackett et al. 1996). It is time for the psychiatric community to follow suit, and utilize a data-driven approach to the classification of mental disorders.

References

Aina, Y., and Susman, J. L. (2006). Understanding comorbidity with depression and anxiety disorders. *The Journal of the American Osteopathic Association*, **106**(5),

American Psychiatric Association (2000). *Diagnostic and statistical manual of mental disorders*, Fourth edition, text rev.). Washington, DC: American Psychiatric Association.

American Psychiatric Association (2013). *Diagnostic and statistical manual of mental disorders* Fifth edition. Arlington, VA: American Psychiatric Publishing.

Barlow, D. H., Sauer-Zavala, S., Carl, J. R., Bullis, J. R., and Ellard, K. K. (2014). The nature, diagnosis, and treatment of neuroticism: Back to the future. *Clinical Psychological Science*, **2**, 344–65.

Brown, T. A., and Barlow, D. H. (2009). A proposal for a dimensional classification system based on the shared features of the DSM-IV anxiety and mood disorders: Implications for assessment and treatment. *Psychological Assessment*, **21**(3), 256–71.

Elbers, R. G., Van Wegen, E. E. H., Verhoef, J., et al. (2012). Reliability and structural validity of the Multidimensional Fatigue Inventory (MFI) in patients with idiopathic Parkinson's disease. *Parkinsonism & Related Disorders*, **18**(5), 532–6.

First, M. (2004). Desire for amputation of a limb: paraphilia, psychosis, or a new type of identity disorder. *Psychological Medicine*, *(***6***)*, 919–28.

Goldberg, D. (2010). Should our major classifications of mental disorders be revised? *The British Journal of Psychiatry*, **196**, 255–6.

Grove, W. M., Andreasen, N. C., McDonald-Scott, P., et al. (1981). Reliability studies of psychiatric diagnosis theory and practice. *Archives of General Psychiatry*, **38**(4), 408–13.

Hopwood, C. J., Thomas, K. M., Markon, K. E., et al. (2012). DSM-5 personality traits and DSM-IV personality disorders. *Journal of Abnormal Psychology*, **121**(2), 424–32.

Hyman, S. (2010). The diagnosis of mental disorders: The problem of reification. *Annual Review of Clinical Psychology*, **6**, 155–79.

Kessler, R. C., DuPont, R. L., Berglund, P., et al. (1999). Impairment in pure and comorbid generalized anxiety disorder and major depression at 12 months in two national surveys. *The American Journal of Psychiatry*, **156**(12), 1915–23

Krueger, R. F., Eaton, N. R., and Clark, L. A., et al. (2011). Deriving an empirical structure of personality pathology for DSM-5. *Journal of Personality Disorders*, **25**(2), 170–91.

Krueger, R. F., Eaton, N. R., Derringer, J., et al. (2011). Personality in DSM-5: Helping delineate personality disorder content and framing the metastructure. *Journal of Personality Assessment*, **93**(4), 325–31.

Livesley, J. (2012). Tradition versus empiricism in the current DSM-5 proposal for revising the classification of personality disorders. *Criminal Behaviour and Mental Health*, **22**(2), 81–90.

Lobbestael, J., Leurgans, M., and Arntz, A. (2011). Inter-rater reliability of the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID I) and Axis II Disorders (SCID II). *Clinical Psychology & Psychotherapy*, **18**, 75–9.

Loevinger, J. (1957). Objective tests as instruments of psychological theory. *Psychological Reports*, **3**, 635–94.

Sackett, D. L., Rosenberg, W. M., Gray, J. A., et al. (1996). Evidence-based medicine: what it is and what it isn't. *British Medical Journal*, **312**, 71–2.

Skodol, A. E., Clark, A., Bender, D. S., et al. (2011). Proposed changes in personality and personality disorder assessment and diagnosis for *DSM-5* Part I: Description and rationale. *Personality Disorders: Theory, Research, and Treatment*, **2**(1), 4–22.

Spitzer, R. L., Endicott, J., and Robins, E. (1978). Research Diagnostic Criteria Rationale and Reliability. *Archives of General Psychiatry*, **35**(6), 773–82.

Widiger, T. A., Livesley, J., and Clark, L. (2009). An integrative dimensional classification of personality disorder. *Psychological Assessment*, **21**(3), 243–55.

Widiger, T. A., and 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.

Chapter 12

Validation of psychiatric classifications: The psychobiological model of personality as an exemplar

C. Robert Cloninger

12.1 Introduction

Confidence in current psychiatric classifications has fallen sharply because they fail to identify the underlying psychobiological causes of syndromes and fail to guide specific prescriptions for treatment. The operational checklist approach has proliferated from 14 useful diagnoses in 1972 (Feighner et al. 1972) to more than 300 sets of criteria for the highly overlapping disorders in DSM-5 (APA, 2013). It had been hoped that the number of atypical cases would be reduced by the addition of more categories, but that has not happened, suggesting that the added categories are redundant and doubtful in utility and validity (Feighner et al. 1972; Kendell and Jablensky 2003; Martin et al. 1985a, b).

In fact, the putative mental disorders in current classifications do not have sharp natural boundaries with points of rarity separating them, so they are not validated as discrete disease entities or taxa (Cloninger 2007; Kendell and Jablensky 2003; Kendell 1982). Some evidence of partial separation has been presented, but even in these cases the separation is weak and inconsistent (Cloninger et al. 1985; Cloninger et al. 1984). The partial separation observed can be explained as the consequences of meta-stable configural states of complex multidimensional systems (Cloninger 2007).

The occurrence of meta-stable configural states is a consistent feature of the dynamics of complex adaptive systems (Waldrop 1992), and has an important implication that mitigates the criticism of categorical classification systems by advocates of purely dimensional systems: self-organizing interactions among dimensions of complex adaptive systems lead to syndromes that are dysfunctional and relatively stable with fuzzy boundaries (Cloninger et al. 1997). Essentially people get stuck in a local optimum rather than the global optimum of well-being, as illustrated in Figure 12.1. There is no direct path to global

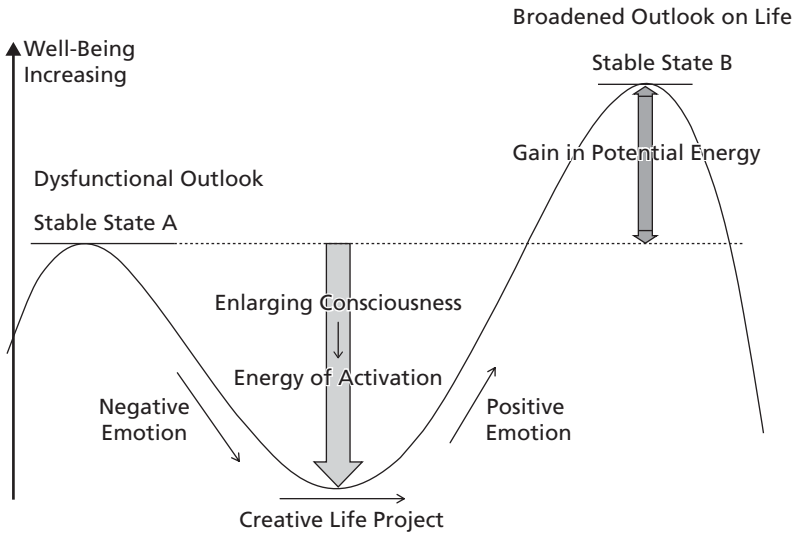

Fig. 12.1 Dynamics of well-being

well-being. For example, in order to advance in well-being, people often have to tolerate deprivation and give up local benefits (e.g., transient intoxication for an addict, or transient positive emotions from binging for a bulimic, illusory entitlement for a narcissist). In other words, no pain, no gain aptly summarizes the dynamics of the "rugged landscape" of complex psychobiological systems. Consequently, many people resist change and persist in poorly adapted lives, causing much suffering for themselves and/or others.

Much useful person-centered clinical information is lost if such meta-stable configural states are ignored and people are only described in terms of quantitative dimensions (Cloninger et al. 2012). Consequently, multiple approaches to case descriptions are needed, including dimensions, configural profiles, and narrative accounts of development over the course of a person's life. In other words, something like categories are useful in classifications, even though they are not discrete disease-entities. However, criteria for category-like prototypes also need to be understood systematically in terms of underlying dimensions and described in part in terms of narratives of the life course of development, which has not been included in current classifications. When underlying dimensions, meta-stable configural states, and life narratives are simultaneously considered as aspects of an integrated system of description and classification, focus can return to the health promotion of the developing person who has self-organizing goals and values, rather than focusing on abstract definitions of heterogeneous syndromes (Cloninger 2013a).

Unfortunately, the developers and users of categorical classification systems often seem to believe that they are dealing with discrete disease entities. Despite the acknowledgment of extensive overlap among disorders, the categories in current classifications have often been reified and misused as if they were homogeneous and discrete disease entities despite the lack of precise natural boundaries between conditions or between what is considered normal and abnormal. The specious reification is facilitated by the clinician's focus on abstract criteria for syndromes rather than on the developing person as a whole and by the lack of a systematic understanding of the underlying causal processes that underlie the development of personality and psychopathology. The number of disorders enumerated continues to proliferate from 106 in DSM-I in 1952 to 297 in DSM-IV and more than 300 in DSM-5 in 2013 (Rosenberg 2013). As the range of disorders has widened, estimates of the prevalence of mental disorders has risen to nearly half of the U.S. population, but are much lower when impairment criteria are carefully applied (Narrow et al. 2002).

The developers of the DSM have acknowledged that "no definition adequately specifies precise boundaries for the concept of mental disorder" (APA 1994). DSM-5 defines a mental disorder as "a syndrome characterized by clinically significant disturbance in an individual's cognition, emotion regulation, or behavior that reflects a dysfunction in the psychological, biological, or developmental processes underlying mental functioning." It further notes that disorders are "usually" associated with significant distress or disability, and that "expectable or culturally approved response to a common stressor or loss, such as the death of a loved one, is not a mental disorder." However, the definition appears to be an afterthought added as a loose generalization rather than a set of strict requirements because each element of these supposedly "required" features has exceptions in practice. For example, in DSM-5 Somatic Symptom Disorder can involve only a single symptom, so it is not a syndrome (i.e., a group of consistently associated symptoms). Nevertheless, previously accepted criteria for Somatization Disorder were omitted from DSM-5 despite their having well-established validity from longitudinal, family, and adoption studies (Cloninger et al. 1986; Cloninger et al. 1984). Bereavement (i.e., loss of a loved one) is not an exclusion criterion for Major Depressive Disorder in DSM-5, even though there are well-established differences between these conditions (Clayton 1990; Clayton et al. 1974). Of course, a single specific phobia could also be a mental disorder and syndromes can be loosely defined to include judgments about distress and impairment, but the APA Task Force is correct that it does not have a "consistent operational definition that covers all situations (APA 1994, 2013)." Without clear guidance from a precise definition of mental disorder or standards of scientific evidence for what is a useful and

valid distinction within the classification system, decisions to add or subtract a diagnosis have often degenerated to non-scientific political discussions of what groups of users like.

As a result of an unmanageable number of redundant criteria, clinicians tend to justify their initial unreliable impressions based on presenting complaints by listing symptoms that fit their impression; and they often fail to obtain a thorough person-centered life narrative (Cloninger et al. 2006). Consequently, the quality of life histories has been impoverished inadvertently by the emphasis on cross-sectional checklists in DSM-III through 5, leading to poorer understanding of the full course of development of illness by many recently trained clinicians. Perhaps the large number of redundant criteria sets accounts for the disappointingly low reliability observed for most diagnoses in DSM-5 field trials (Regier et al. 2013).

These developments call into question the adequacy of the natural history approach to validation of mental disorders that Robins and Guze (1970) modeled on the work of Sydenham in the seventeenth century and Kraepelin in the eighteenth century (Cloninger 1989). Robins and Guze recommended what they regarded as an atheoretical empirical approach in which valid diagnoses could be established by studies involving five phases: clinical description to describe a syndrome of associated signs and symptoms, exclusion of other disorders, laboratory studies to identify causes and distinguishing biological processes, follow-up studies to characterize outcome and show the stability of diagnoses, and family studies to show familial aggregation of people with the same disorders in families (Robins and Guze 1970). Originally, 14 categories were distinguished on the basis of four of these phases. No laboratory test reliably distinguished among the disorders in the early classifications and that remains true to this day (APA 2013), despite the perennial promise that such laboratory findings will be shortly forthcoming (de Freitas Araujo 2011; Moreira-Almeida and Santos 2011).

Robins and Guze (1970) emphasized the importance of exclusion criteria so that their approach would be person-centered and allow only one diagnosis of mental disorder per person (except for depressions that could be secondary to a pre-existing illness). Unfortunately, exclusion criteria have been systematically weakened or dropped in DSM with the initial justification that classifications should be comprehensive and provide a label for anyone with a mental disorder if at all possible (Cloninger 1990, 2002; Maser and Cloninger 1990). This has led to extensive "comorbidity" with overlap between redundant criteria sets, which is so extensive that DSM-IV and DSM-5 explicitly acknowledge that the lack of discriminant validity means that the putative disorders cannot be regarded as discrete disease-entities (APA 1994, 2013). In practice, under the current

diagnostic rules, people who qualify for one diagnosis usually satisfy criteria for multiple disorders (Cloninger 2002). There is so much heterogeneity within categories and overlap among categories that neurochemical, genetic, and brain imaging findings have failed to distinguish putative mental disorders from one another reliably, primarily because of a lack of specificity (Craddock and Owen 2005).

What does the disappointing state of psychiatric classification mean for future work at establishing the validity of psychiatric assessments? Why has it been so difficult to develop a valid psychiatric classification? The fundamental challenge is that common mental disorders are complex phenomena that result from non-linear interactions among multiple biological, mental, social, and spiritual processes that influence development (Arnedo et al. 2013; Cloninger 2004; Svrakic et al. 2013). These interactions are complex in the sense that specific combinations of the same antecedent causes can lead to different clinical outcomes ("multi-finality") and different antecedent can lead to the same outcome through common developmental pathways ("equi-finality") (Cicchetti and Rogosch 1996; Cloninger et al. 1997). Reciprocal feedback interactions are pervasive characteristics of psychobiological processes, so that models are inadequate if they assume linearity (i.e., consider unidirectional chains of causes whose effects are additive). Linear thinking from "top-down" (i.e., phenotype to cause) or "bottom-up" (i.e., cause to phenotype) has never and can never prove an accurate understanding of the pathophysiology of the reciprocal feedback interactions among the underlying causes of mental disorders, despite the illusory promises of reductionist materialism (de Freitas Araujo 2011).

Nevertheless, psychiatry now has the tools needed to systematically begin to understand the processes that lead to both well-being and ill-being. The work over the last two decades on the validation of the psychobiological model of personality illustrates the way an integrative understanding of biological, psychological, and social processes underlying health can permit the development of valid methods for assessing personality and its disorders. Extensive research has shown that understanding the structure, function, and development of personality provides a framework for understanding the determinants of health as well as vulnerability to personality disorders and psychopathology (Cloninger 2013a; Cloninger, Salloum, et al. 2012). Although this is a sweeping claim, at a minimum, consideration of the psychobiological model of personality provides an example of a systematic way to validate psychiatric classifications and points out key observations that have been neglected in alternative approaches, thereby leading to the inadequacies of current and recently proposed systems for personality and its disorders.

Many approaches that have been advocated for personality assessment have failed to produce models and theories that account for the facts about personality that will be discussed here. Personality models have been based on factor analysis of descriptors (Eysenck and Eysenck 1969; Thomas et al. 2012), lexical content of languages to describe personality (Benet and Waller 1995; Goldberg 1990), prototypic descriptions (Samuel and Widiger 2004), and a wide range of reductive approaches emphasizing biology, emotionality, cognition, or socialization. Consequently, much can be learned from the way an integrative psychobiological approach allows the establishment of validity. Of course, meaningful approaches to theoretical and practical understanding require integration of intuition, observation, and tests of predictions, so that many divergent facts are carefully considered. People always want to find systematic methods that can be applied as algorithms with little creative thinking or understanding; consequently, they end up relying excessively on methods that are inadequate to deal with all the hidden complexities of natural phenomena. Albert Einstein warned us about such hidden complexities when he said, "There is, of course, no logical way leading to the establishment of a theory, but only groping, constructive attempts controlled by careful considerations of factual knowledge" (Shankland 1964).

12.2 The Psychobiological Approach to Personality Assessment

12.2.1 Defining Personality

It is useful in my experience to begin any effort at understanding a phenomenon to try to define its properties clearly and precisely. In the simplest definition, personality is the distinctive way that an individual learns to adapt (Cloninger 2004). Personality can also be defined more specifically as the characteristics of the self plus the internal and external forces that pull on the self. After exhaustive consideration of hundreds of suggested definitions, Gordon Allport offered the following definition: "Personality is the dynamic organization within the individual of those psychophysical systems that determine his unique adjustments to his environment" (Allport 1937). In order to more fully recognize self-directed processes in development (Rogers 1995), Allport's definition can be revised to "the dynamic organization within the individual of the psychobiological systems by which the person both shapes and adapts uniquely to an ever-changing internal and external environment" (Cloninger and Cloninger 2011). Thus personality is not a set of fixed differences between people as construed by trait theory, but is a complex set of functions by which a person both shapes and adapts to experience in the proactive development of his or her life narrative.

12.2.2 Non-Linear Dynamic Systems

The foregoing definition of personality entails that assessment should be directed toward mechanisms of learning and memory that occur within the person, rather than merely superficial behaviors that differ between people (Cervone 2005). Jeffrey Gray pointed out that Eysenck's personality models derived by factor analysis described the architecture of between-person differences but did not correspond to the underlying learning processes within the person (Cloninger 1986). For instance, Eysenck's neurotic extraverts (i.e., novelty seekers) learn best in response to reward conditioning, whereas neurotic introverts (i.e., harm-avoidant individuals) learn best in response to aversive conditioning (Cloninger 1986, 1987; Corr et al. 1997). The effects of behavioral conditioning interact with personality type (that is, specific combinations of personality and conditioning have unique effects): a person who is highly harm avoidant (i.e., worried, fearful, and shy) has an augmented startle when they are thinking of something unpleasant, whereas a person who is low in harm avoidance (i.e., risk-taking, relaxed, and outgoing) has a reduced startle when they are thinking of something pleasant (Corr et al. 1997; Corr, Pickering, et al. 1995; Corr, Wilson, et al., 1995).

However, pleasant thoughts do not reduce the startle responses of people who are high in harm avoidance, and unpleasant thoughts do not augment the startle responses of people low in harm avoidance. Nevertheless, most personality psychologists were trained to rely on the traits that emerge from linear factor analysis and persist in this practice despite the questionable validity of using only additive traits to understand the interactive processes underlying personality (Cloninger 2008). In other words, most people think of traits as composed of separate units that can be added together to describe a total personality, whereas traits interact with each other and with the person's situation in ways that are not additive. The whole of personality is more than the sum of its parts, so personality cannot be reduced to a set of individual parts. Statistical methods that assume such additivity, as does linear factor analysis, produce distorted models of personality structure that are not true to life and impede understanding of the learning processes within a person by which he or she adapts to their situations in life.

12.2.3 Normal Functional Variability

A fundamental question about personality is whether abnormal traits are extremes of normal variation or results of particular combinations of traits in situations for which they are poorly adapted. The decisive fact is the observation that the (correlational) structure of personality traits is the same in clinical samples as it is in the general population, whether or not the test is designed to

measure normal traits (Bayon et al. 1996; Cloninger 2008; Strack 2006; Svrakic et al. 1993) or abnormal traits (Fossati et al. 2013). In other words, abnormal personality profiles are more prevalent in clinical samples than in the general population, but the correlations among these traits are the same regardless of the average values of scores. Therefore, abnormal personality traits must be extreme combinations of normal variation in personality. Even schizotypal, cyclothymic, and depressive personality traits are fully accounted for by variation in normal personality traits (Cloninger 1999). This finding supports the view that personality traits involve adaptive functions that enable people to learn how to deal with the full range of life functions, including sexuality, materiality, emotion, intellect, and spirituality (Cloninger 2004). Consequently, much can be learned about personality in relation to health by considering facts related to its function, structure, evolution, and development. In fact, emphasis on abnormal extremes may be therapeutically counterproductive, thereby undermining the therapeutic utility of personality assessment (Cloninger and Cloninger 2011).

12.3 What do we Know about Personality with Reasonable Certainty?

What we have learned about personality helps us to understand the relations between personality and health. These useful observations can be divided into information about its functions, structure, evolution, and development. Here the key observations that have been evaluated and confirmed are listed, and the process that led to their discovery will be discussed in the next section.

12.3.1 Functions of Personality

(1) Personality refers to adaptive processes of learning and memory within individuals (Cloninger 2004):
   (a) There are three major systems of learning and memory in human beings: behavioral conditioning of habits and skills, semantic learning of facts and propositions, and self-aware learning of intuitions and narratives (Tulving 1987, 2002).
   (b) These three systems of learning distinguish among the properties of three distinct domains of human personality (temperament, character, and the narrative self) (Cloninger 1994, 2004). Specifically, temperament varies quantitatively in terms of strengths of habits and irrational emotional drives (e.g., fear, anger), whereas character varies qualitatively in terms of the concepts that organize our goals and values in a more or less rational way. The narrative self develops in

self-aware consciousness in a way that is more or less self-organizing and creative in that people can develop in ways that are not fully predictable by temperament and character.

(c) Temperament, character, and the narrative self represent the corporeal, mental, and spiritual components of personality respectively and have distinctive functional properties (Cloninger 2004).

(2) Adaptive functions of personality are strongly related to individual differences in activation of brain circuitry (Cloninger 2009; Cloninger and Kedia 2011; Sussman and Cloninger 2011).

(3) Specific functions can be measured as facets of personality (personality subscales) that involve adaptation in specific situations of life (Cloninger 2009; Cloninger and Kedia 2011).

12.3.2 Structure of Personality

(4) Variation in personality traits varies quantitatively along multiple dimensions:

(a) Personality traits are weakly to moderately correlated, thereby influencing one another during development.

(b) There are no sharp boundaries separating people into taxa or discrete personality subtypes (Cloninger 2007).

(5) Personality prototypes or clusters of people with similar personality profiles occur as meta-stable configurations of extremes on multiple dimensions (Cloninger and Cloninger 2011).

(6) The correlations within domains of temperament or character are weak but there are strong interactions (i.e., reciprocal feedback) among these domains during development (in other words, the emotional drives of temperament influence the development of character, and vice versa) (Cloninger et al. 1997).

These structural relationships are depicted in Figure 12.2.

12.3.3 Evolution and Development of Personality

(7) Components of personality in the psychobiological model are summarized in Figure 12.3. Behavioral conditioning of habits underlying four temperament dimensions evolved in a stepwise fashion and was well developed in early vertebrates (fish to reptiles) (Cloninger and Gilligan 1987). These temperament dimensions are called Harm Avoidance (i.e., anxious, shy vs. risk-taking, outgoing), Novelty Seeking (i.e., impulsive, disorderly vs. rigid, orderly), Reward Dependence (i.e., warm, attached vs. aloof, detached),

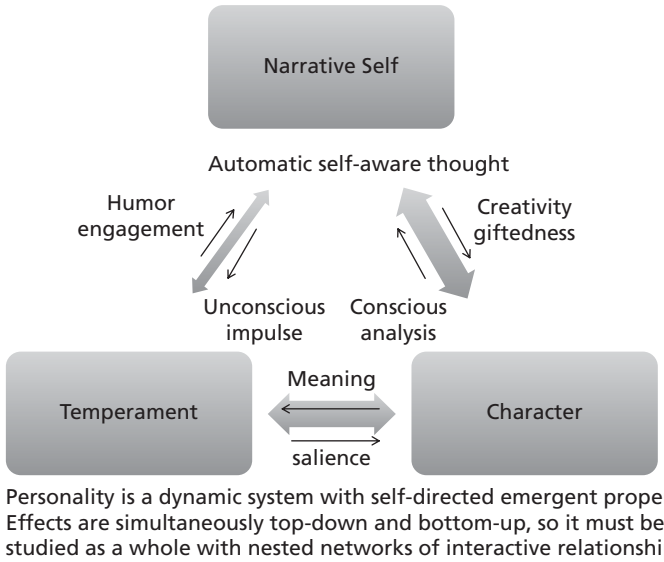

Fig. 12.2 Cycle of reciprocal interactions among the domains of personality

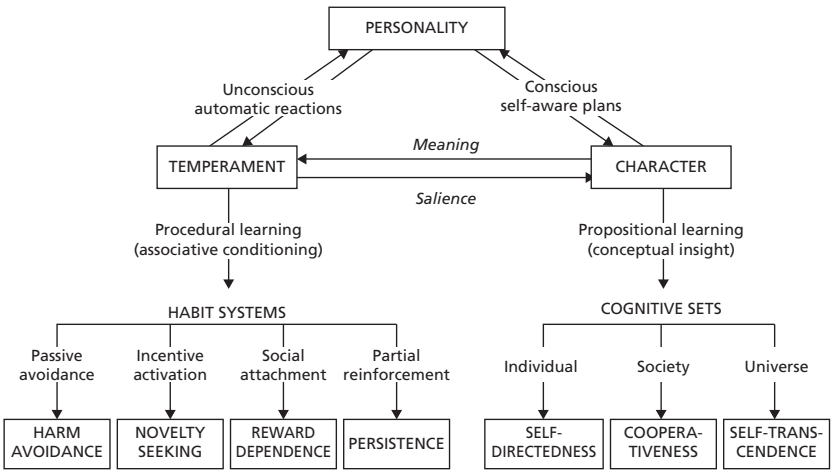

Fig. 12.3 Schematic summary of the psychobiological model of personality

and Persistence (i.e., determined, ambitious vs. easily discouraged, vacillating). Earlier questionable speculations about such evolutionary paths have recently been tested and refined empirically. This has been possible by means of detailed studies that integrate molecular phylogenetics and the comparative neuroanatomy of the ancestors of human beings (Cloninger 2009; Cloninger and Kedia 2011). The coincident changes in structure and

function of brain organization in the unique chain of ancestors leading to human beings provides a solid foundation for understanding structure–function relations in the evolving human brain. This evolutionary basis for organization of human brain structure–function relations has also been tested by results of functional brain imaging, which show strong interactions among circuits that instantiate distinct functions measured by different personality dimensions (Cloninger, Zohar, et al. 2012; Gusnard et al. 2001; Gusnard et al. 2003; Pezawas et al. 2005).

(8) In mammals, the structures and functions of semantic learning evolved with the neocortical control of the reptilian brain to regulate emotional drive (which underlies coope rativeness) and analytical reasoning (which underlies self-directedness); later self-transcendence emerged with self-aware consciousness in modern human beings (Cloninger 2009; Cloninger and Kedia 2011).

(9) The development of personality in human beings is a complex adaptive system whose dynamics is shaped by reciprocal feedback relations among multiple processes interacting with one another differentially in various situations (Cloninger et al. 1997; Josefsson, Jokela, et al. 2013).

12.3.4 Relation of Personality to Health

(10) Personality traits strongly modulate susceptibility to the full range of psychopathology (Cloninger 1999; Cloninger et al. 2010).

(11) Personality traits strongly modulate susceptibility to physical disorders, particularly the major chronic diseases such as atherosclerosis (Cloninger 2013a; Rosenstrom et al. 2012).

(12) A healthy personality is characterized by average temperaments and strong character development (high Self-directedness, Cooperativeness, and Self-transcendence) (Cloninger 2013b). Self-directedness and Cooperativeness are insufficient for optimal health, at least under current world conditions (Cloninger 2013b). Self-transcendence is also needed for flourishing of the narrative self with creative functioning, plasticity, and fulfilling values (Cloninger and Cloninger 2011; Seligman 2011).

12.4 Development and Validation of the Assessment Model

The psychobiological model of personality was developed in three major steps. Initially, personality was limited to four temperament dimensions that were based on individual differences in behavioral conditioning of habits or emotional drives.

The model was based on extensive research in operant learning, experimental manipulation of brain systems, and twin studies of the genetic structure of personality (Cloninger 1986, 1987). The temperaments were called Harm Avoidance, Novelty Seeking, Reward Dependence, and Persistence. The identified traits were called temperaments because they were presumed to be moderately heritable and developmentally stable from an early age, which was later consistently confirmed (Heath et al. 1994; Josefsson, Jokela, et al. 2013; Stallings et al. 1996).

The temperament dimensions were able to distinguish variants of personality disorder in terms of profiles (i.e., configurations of extremes of the dimensions) (Cloninger 1987) (see Figure 12.4). For example, the antisocial type of personality profile is high in Novelty Seeking (i.e., impulsive, quick-tempered), low in Harm Avoidance (i.e., risk-taking, uninhibited), and low in Reward Dependence (i.e., cold, aloof). However, this same profile could occur in people who were mature and responsible citizens despite their adventurous emotional style. So other features were needed to specify whether someone had a personality disorder or not, and to quantify how mature or immature the person was.

An empirical search was first carried out to identify any personality traits that could not be explained by the four temperament dimensions. This yielded clues to three traits that were then elaborated into character traits of Self-directedness (i.e., resourceful, purposeful, responsible), Cooperativeness (i.e., tolerant, helpful,

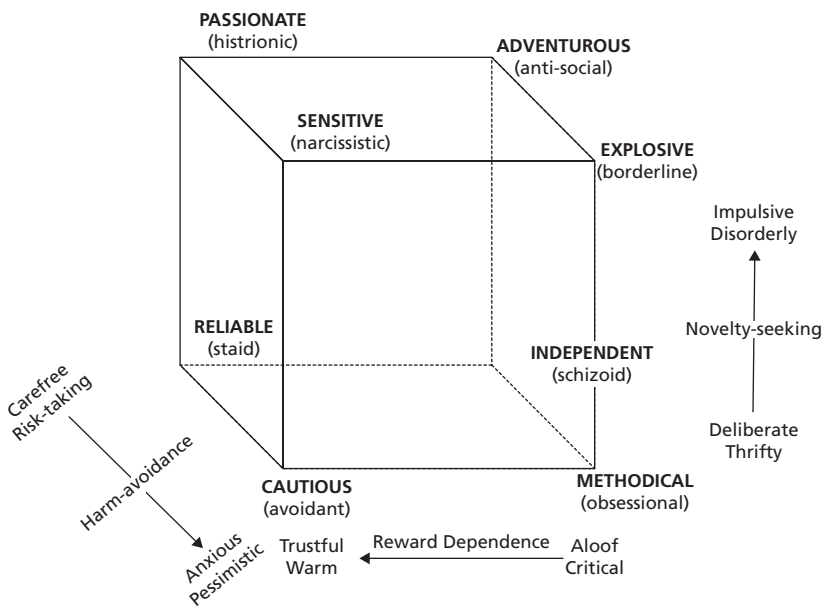

Fig. 12.4 The temperament cube

empathic), and Self-transcendence (i.e., intuitive, imaginative, spiritual), each expressed in somewhat different ways in five types of life situations (i.e., sexual, material, emotional, intellectual, and spiritual) (Cloninger 2004; Cloninger et al. 1993). Studies of the resulting Temperament and Character Inventory (TCI) were then carried out in both the general population and in clinical samples of psychiatric in-patients and outpatients for purposes of validation (Bayon et al. 1996; Cloninger et al. 1994; Cloninger et al. 1993; Svrakic et al. 1993). Findings showed that the three character dimensions allowed measurement of whether someone had a personality disorder and how severe the immaturity of personality was (Svrakic et al. 1993). Specifically, low scores in Self-directedness and Cooperativeness provide a measure of the DSM concept of personality disorder, which has subsequently been confirmed consistently in many studies in many cultures. In fact, high Self-directedness and Cooperativeness were recently proposed by the DSM-5 Personality Disorder Study Group to be indicators of a healthy personality (APA 2013; Cloninger 2010).

Nevertheless, two surprises were observed about character. First, character traits were found to be as heritable as temperament traits (Gillespie et al. 2003). This finding called into question the hypothesis made in 1993 that character was less heritable and more influenced by sociocultural influences than temperament (Cloninger et al. 1993). The distinction between temperament and character cannot be based on the heritability of the dimensions, so I suggested that it was based on differences in the mechanism of learning. Subsequent research has confirmed that character traits are regulated by differences in the activation of specific zones of the human neocortex, particularly components of the semantic and self-aware systems of learning and memory (Cloninger 2004, 2008). For example, individual differences in Self-directedness are strongly correlated with activation of the medial prefrontal cortex, Cooperativeness with activation of the orbitofrontal cortex, and Self-transcendence with activation of the anterior frontal polar cortex (Cloninger 2004; Cloninger and Kedia 2011). In contrast, temperament dimensions are regulated by differences in activation of zones for regulation of habits and emotion in the limbic system (Gardini et al. 2009; Gusnard et al. 2003; Pezawas et al. 2005).

In addition to character being related to more recently evolved brain regions than temperament, character is also distinguished from temperament by its greater sensitivity to the effects of parental care-taking and sociocultural influences. Longitudinal studies from childhood through adulthood have confirmed the original hypothesis that character development is influenced by parental care-taking and social norm-favoring (Josefsson, Jokela, Cloninger, et al. 2013; Josefsson, Jokela, Hintsanen, et al. 2013). Temperament dimensions do not

have a consistent direction of change over the life course; average scores stay the same because equal numbers of individuals increase or decrease as a result of individual differences in conditioning. In contrast, character traits develop in the direction favored by social norms.

Longitudinal studies also revealed a second surprise about character development (Cloninger et al. 1997). When character did change, the correlations among temperament and character dimensions allowed prediction of the direction of those changes as a complex adaptive process (Cloninger et al. 1997). However, it was not possible to foretell who would change, and most people did not change substantially.

What accounted for the capacity of some people to mature and to increase in well-being, others to become less well, and others to stay the same? The answer required an appreciation of the distinction between semantic and self-aware learning. Semantic learning is analytical and logical, so it is based on assumptions from which conclusions are deduced. Most people are as resistant to changing their assumptions and their outlook on life as they are to changing their habits. In contrast, self-aware learning has been called "mental time-travel" and involves the unique human capacity to change one's perspective rapidly and freely by imagining and contemplating future developments in our personal narratives of life (Cloninger 2004; Tulving 2002).

Interactions among the three character traits produce meta-stable profiles with distinctive clinical features, as depicted in Figure 12.5. The relationship between the three character traits to learning can be understood by thinking of them as branches of mental self-government. Self-directedness is the executive branch, pursuing accomplishment of goals in a purposeful and responsible manner. Cooperativeness is the legislative branch, making the rules by which we get along with one another in a considerate and principled manner. Self-transcendence is the judicial branch, using its capacity for imagination, contemplation, and insight to recognize and understand when the rules apply. From this perspective, the capacity to identify with what is beyond one's individual self (i.e., Self-transcendence) also allows us to imagine and contemplate change in outlook (i.e., perspective shifting). Self-transcendence, then, would have a key role in the theoretical thinking of scientists, the appreciation of artistic beauty, as well as the philosophical contemplation of values and the meaning of life. Could self-transcendence, then, be an indicator of who would grow and mature in character? In fact, longitudinal studies of people between 20 and 45 years of age have now shown that personality change is most likely to occur in people who are high in Self-transcendence, Novelty Seeking, and Persistence (Josefsson, Jokela, et al. 2013).

Self-directedness and Cooperativeness are consistently associated with maturity, health, and happiness, but the role of Self-transcendence is more complex because

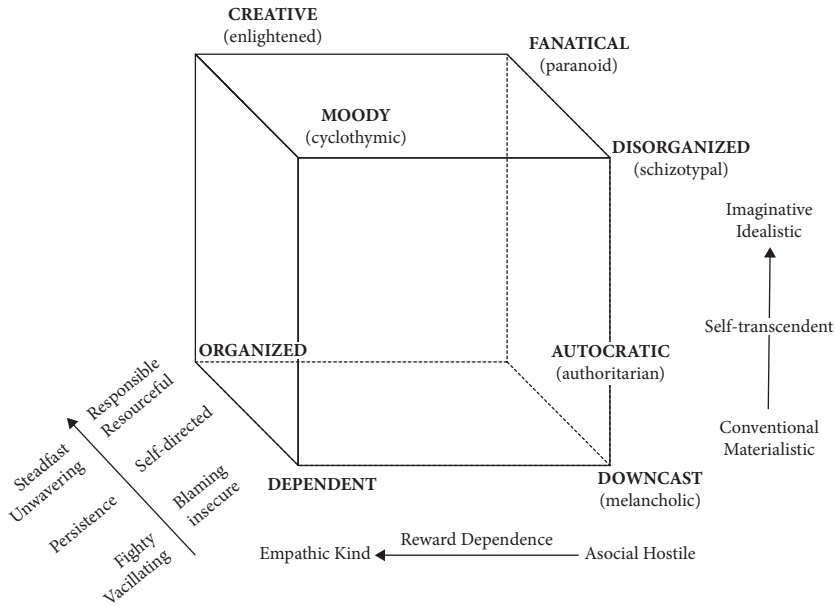

Fig. 12.5 The character cube

it depends on the full configuration of character traits. If Self-directedness is low, a person is unrealistic (i.e., represses true facts that are unpleasant and inconvenient). If a person has a vivid imagination (i.e., is high in Self-transcendence) and is unrealistic (i.e., is low in Self-directedness), then they have schizotypal traits with prominent magical thinking (Smith et al. 2008). Self-transcendence is always associated with increasing positive affect regardless of whether a person is high or low in the other two character traits. Therefore, the healthiest character profile results when all three character traits are elevated, a profile described as creative or enlightened (Cloninger 2013b; Cloninger and Cloninger 2011). A creative character profile is realistic, helpful, and flexibly adaptive.

Ongoing research is evaluating the mechanisms by which this combination of traits facilitates psychobiological coherence and well-being (Cloninger and Zohar 2011; Cloninger, Zohar, et al. 2012; Zohar et al. 2013). A review of the TCI correlates of psychopathology is available elsewhere (Cloninger et al. 2010).

12.5 Differences from Factor Analytic Models of Personality

The most obvious difference between the psychobiological model and other modern personality models is the distinction between temperament and character, which are confounded in factor-analytic models. Factor analysts argue

that the distinction between temperament and character is both unnecessary and mistakenly produces a more complex structure than is obtained under the simplifying assumption of linearity (Cloninger 2008). However, this simplified model is itself based on the false assumption of additivity. It does not account for the incontrovertible facts of personality development, such as sudden stepwise growth at critical tipping points and the phenomena of multi-finality and equi-finality (Cloninger et al. 1997).

The consequences of using invalid simplifying assumptions for clinical assessment can be seen by considering the heterogeneity of the content within the traits identified in factor-analytic models. The most consistent trait in factor-based personality models is usually called Neuroticism or Negative Affectivity, and it is strongly correlated with both High TCI Harm Avoidance and Low TCI Self-directedness. That means that Neuroticism confounds two traits with different genetic influences, different brain circuitry, and different roles in learning and adaptation.

Psychiatrists and psychologists recognize that personality disorders are not the same as anxiety disorders, but these patients groups are both described as high in Neuroticism in factor-analytic models. Specifically, high Harm Avoidance is correlated with augmented startle responses and Mismatch Negativity, but not with dysfunctional cognitive attitudes (Corr et al. 1997; Hansenne et al. 2003). In contrast, low Self-directedness is associated with dysfunctional attitudes but not with augmented startle or Mismatch Negativity (Luty et al. 1999; Richter and Eisemann 2002). Hence factor-analytic models falsely assume homogeneity in whatever is correlated, an error that can be avoided by attention to extra-statistical information about biology and development that are crucial for valid psychiatric classification.

12.6 Differences from the Proposed DSM-5 Personality Criteria

The criteria proposed by the DSM-5 working group on personality disorder were not accepted, because the APA's Scientific Review Committee regarded the scientific evidence as insufficient for the major changes proposed and the APA's Clinical and Public Health Review Board regarded the criteria as too unwieldy for routine clinical use.

The proposed criteria were not merely complex; they were made up of three components that were developed separately and are not coherently related to one another. The three components of the proposed criteria were a reduced list of personality disorder types, a description of healthy personality, and

a list of five pathological traits like those derived by factor-analysis (APA 2013). However, the categories were not systematically derived from the set of quantitative traits, as is done in the psychobiological model of personality. Likewise, the healthy personality traits were not healthy poles of the pathological personality traits. In fact, the description of healthy personality included measures closely related to Self-directedness and Cooperativeness. Self-directedness is not the healthy pole of Negative Affectivity because Negative Affectivity includes low Self-directedness combined with high Harm Avoidance.

As a result of the apparent inconsistencies, the subcommittees tried to integrate the components of the proposed criteria. Unfortunately, the result that is listed in Section III of DSM-5 is obviously an awkward effort at retrofitting disparate elements to one another. The absence of a coherent theory and the use of analytic methods that make invalid simplifying assumptions about personality (specifically linear factor-analysis) reflects the scientific disarray afflicting psychiatric classification and the field of abnormal personality in particular (Cloninger 2007).

12.7 Similarity with Prototypic and Narrative Models

Personality develops as a complex adaptive system, thereby leading to sudden stepwise growth, multi-finality, and equi-finality. This means that development is a non-linear process in which individuals are often stuck in a meta-stable "attractor" state (Cloninger 2004), as depicted in Figure 12.1. They stay in these states for long periods of stasis until they reach a critical tipping point, at which time they may rapidly shift into another attractor state with higher or lower well-being. Once in this state, they remain there even if the factors that led to the transition subside. This provides a precise quantitative account of the dynamics of personality development, and it also provides a bridge to prototypic models of personality.

In essence, configurations of multiple temperament or character traits define particular profiles that correspond to traditionally recognized personality prototypes like the example of antisocial personality disorder given earlier. Hence multidimensional profiles are a person-centered approach to description similar to prototypes (Cloninger 2004; Cloninger and Cloninger 2011). Dividing each dimension at its median into high and low extremes is sufficient to cluster individuals to the prototype they most resemble, thereby providing remarkably more information than linear analyses can provide (Cloninger and Zohar 2011; Cloninger, Zohar, et al. 2012).

The psychobiological model provides both quantitative measurements and prototypic profiles in a person-centered perspective, thereby providing an explanation for the power of prototypic formulations. Perhaps this is what Robins and Guze sought to recognize by insisting that a person could have only one diagnosis, which they found adequate in 80 percent of mental patients. Relaxing exclusion criteria in DSM created the appearance of comorbidity as a result of what are overly loose and redundant criteria.

In addition, the psychobiological model provides a way to recognize the great importance of both traits and situations. A person's behavior varies more between situations than between persons (Fleeson 2001). The facets of the dimensions correspond to the expression of similar functions in different situations (Cloninger 2004). Hence extreme high and low scores in particular facets identify outstanding strengths and weaknesses that a person has in particular situations, much as is done in schema-based cognitive therapy and in narrative therapy. The emphasis in the integrative approach to treatment guided by the psychobiological model is on reflection and contemplation of goals and values so that a person takes the initiative to accept their situation and commits to work toward valued actions, as in evidence-based third-wave psychotherapies (Vowles et al. 2011).

12.8 Implications for the Validation of Psychiatric Classifications

The development and validation of the psychobiological model of personality shows that there is no purely empirical or purely logical way that consistently leads to establishing the validity of a system of assessment and classification. All approaches to valid knowledge require the use of intuition, reasoning, and observations to test, correct, and refine partial insights (Cloninger 2004; Godel 1981). Algorithmic approaches vary in their adequacy for understanding different phenomena under particular conditions. No system of classification should be regarded as a "bible" because they are certainly not infallible and have limited reliability and validity in practice. Because they stifle creative investigation of alternative approaches, requiring use of certain criteria like the DSM-5 or the Research Domain Criteria of the National Institute of Mental Health in the USA for funding in research is a seriously flawed science policy.

Psychiatry now has remarkable bioinformatic tools for the development of valid classifications (Arnedo et al. 2013; Arnedo et al. in press), but the complexity of nature is humbling. We must approach human nature with creativity and humility, avoiding rigid preconceived protocols and adapting ourselves to what we can observe, interpret, and contemplate about the inexhaustible mysteries of human nature.

References

Allport, G. W. (1937). *Personality: A psychological interpretation*. New York: Holt, Rinehart & Winston.

APA (1994). *Diagnostic and Statistical Manual*, Fourth edition. Washington, DC: American Psychiatric Association.

APA (2013). *Diagnostic and Statistical Manual (DSM-5)*, Fifth edition. Washington, DC: American Psychiatric Association.

Arnedo, J., del Val, C., de Erausquin, G. A., et al. (2013). PGMRA: a web server for (phenotype x genotype) many-to-many relation analysis in GWAS. *Nucleic Acids Res*, *41* (Web Server issue), W142–149.

Arnedo, J., Svrakic, D. M., del Val, C., et al. (in press). Molecular Genetics of Schizophrenia Consortium, Fanous A, Pato M, Pato CN, de Erausquin GA, Cloninger CR, Zwir I. Uncovering the Hidden Risk Architecture of the Schizophrenias: Confirmation in three independent genome-wide association studies. *American Journal of Psychiatry.*

Bayon, C., Hill, K., Svrakic, D. M., et al. (1996). Dimensional assessment of personality in an out-patient sample: relations of the systems of Millon and Cloninger. *Journal of Psychiatric Research*, **30**(5), 341–52.

Benet, V., and Waller, N. G. (1995). The Big Seven factor model of personality description: Evidence for its cross-cultural generality in a Spanish sample. *Journal of Personality and Social Psychology*, **69**(4), 701–18.

Cervone, D. (2005). Personality architecture: within-person structures and processes. *Annual Review of Psychology*, **56**, 423–52.

Cicchetti, D., and Rogosch, F. A. (1996). Equifinality and multifinality in developmental psychopathology. *Development and Psychopathology*, **8**, 597–600.

Clayton, P. J. (1990). Bereavement and depression. *Journal of Clinical Psychiatry*, **51** Suppl, 34–8; discussion 39–40.

Clayton, P. J., Herjanic, M., Murphy, G. E., et al. (1974). Mourning and depression: their similarities and differences. *Canadian Psychiatric Association Journal*, **19**(3), 309–12.

Cloninger, C. R. (1986). A unified biosocial theory of personality and its role in the development of anxiety states. *Psychiatric Developments*, **4**(3), 167–226.

Cloninger, C. R. (1987). A systematic method for clinical description and classification of personality variants. A proposal. *Archives of General Psychiatry*, **44**(6), 573–88.

Cloninger, C. R. (1989). Establishment of diagnostic validity in psychiatric illness: Robins and Guze's method revisited. In L. N. Robins and J. Barrett (eds), *Validity of Psychiatric Diagnosis* (pp. 9–18). New York: Raven Press.

Cloninger, C. R. (1990). Comorbidity of anxiety and depression. *Journal of Clinical Psychopharmacology*, **10**(3 Suppl), 43S–46S.

Cloninger, C. R. (1994). Temperament and personality. *Current Opinion in Neurobiology*, **4**(2), 266–73.

Cloninger, C. R. (ed.) (1999). *Personality and Psychopathology*, First edition. Washington, DC: American Psychiatric Press.

Cloninger, C. R. (2002). Implications of comorbidity for the classification of mental disorders: The need for a psychobiology of coherence. In M. Maj, W. Gaebel, J. J. Lopez-Ibor, and N. Sartorius (eds), *Psychiatric Diagnosis and Classification* (pp. 79–105). New York: Wiley.

Cloninger, C. R. (2004). *Feeling Good: The Science of Well-Being*. New York: Oxford University Press.

Cloninger, C. R. (2007). Foreword. In W. T. O'Donohue, K. A. Fowler, and S. O. Lilienfeld (eds), *Personality Disorders: Toward the DSM-V* (pp. vii–xvi). Los Angeles: Sage Publications.

Cloninger, C. R. (2008). The psychobiological theory of temperament and character: comment on Farmer and Goldberg (2008). *Psychological Assessment*, **20**(3), 292–9; discussion 300–4.

Cloninger, C. R. (2009). The evolution of human brain functions: the functional structure of human consciousness. *Australian and New Zealand Journal of Psychiatry*, **43**(11), **994**–1006.

Cloninger, C. R. (2010). Personality and Temperament: New and Alternative Perspectives *Focus*, **8**(2), 161–3.

Cloninger, C. R. (2013a). Person-centered health promotion in chronic disease. *International Journal of Person-centered Medicine*, **3**(1), 5–12.

Cloninger, C. R. (2013b). What makes people healthy, happy, and fulfilled in the face of current world challenges? *Mens Sana Monogr*, **11**, 16–24.

Cloninger, C. R., and Cloninger, K. M. (2011). Person-centered Therapeutics. *International Journal of Person-centered Medicine*, **1**(1), 43–52.

Cloninger, C. R., and Gilligan, S. B. (1987). Neurogenetic mechanisms of learning: a phylogenetic perspective. *Journal of Psychiatric Research*, **21**(4), 457–72.

Cloninger, C. R., and Kedia, S. (2011). The phylogenesis of human personality: identifying the precursors of cooperation, altruism, and well-being. In R. W. Sussman and C. R. Cloninger (eds), *The Origins of Cooperation and Altruism* (First edition, pp. 63–110). New York: Springer.

Cloninger, C. R., Martin, R. L., Guze, S. B., et al. (1985). Diagnosis and prognosis in schizophrenia. *Archives of General Psychiatry*, **42**(1), 15–25.

Cloninger, C. R., Martin, R. L., Guze, S. B., et al. (1986). A prospective follow-up and family study of somatization in men and women. *American Journal of Psychiatry*, **143**(7), 873–8.

Cloninger, C. R., Przybeck, T. R., Svrakic, D. M., et al. (1994). *The Temperament and Character Inventory (TCI): A Guide to its Development and Use*. St. Louis: Washington University Center for Psychobiology of Personality.

Cloninger, C. R., Salloum, I. M., and Mezzich, J. E. (2012). The dynamic origins of positive health and wellbeing. *International Journal of Person-centered Medicine*, **2**(2), 1–9.

Cloninger, C. R., Sigvardsson, S., von Knorring, A. L., et al. (1984). An adoption study of somatoform disorders. II. Identification of two discrete somatoform disorders. *Archives of General Psychiatry*, **41**(9), 863–71.

Cloninger, C. R., Svrakic, D. M., and Przybeck, T. R. (1993). A psychobiological model of temperament and character. *Archives of General Psychiatry*, **50**(12), 975–90.

Cloninger, C. R., Svrakic, D. M., and Przybeck, T. R. (2006). Can personality assessment predict future depression? A twelve-month follow-up of 631 subjects. *Journal Affective Disorders*, **92**(1), 35–44.

Cloninger, C. R., Svrakic, N. M., and Svrakic, D. M. (1997). Role of personality self-organization in development of mental order and disorder. *Developmental Psychopathology*, **9**(4), 881–906.

Cloninger, C. R., and Zohar, A. H. (2011). Personality and the perception of health and happiness. *Journal of Affective Disorders*, **128**(1–2), 24–32.

Cloninger, C. R., Zohar, A. H., and Cloninger, K. M. (2010). Promotion of well-being in person-centered mental health care. *Focus*, **8**(2), 165–79.

Cloninger, C. R., Zohar, A. H., Hirschmann, S., et al. (2012). The psychological costs and benefits of being highly persistent: personality profiles distinguish mood disorders from anxiety disorders. *Journal of Affective Disorders*, **136**(3), 758–66.

Corr, P. J., Kumari, V., Wilson, G. D., et al. (1997). Harm Avoidance and affective modulation of the startle reflex: A replication. *Personality and Individual Differences*, **22**, 591–3.

Corr, P. J., Pickering, A. D., and Gray, J. A. (1995). Personality and reinforcement in associative and instrumental learning. *Personality and Individual Differences*, **19**, 47–71.

Corr, P. J., Wilson, G. D., Fotiadou, M., et al. (1995). Personality and affective modulation of the startle reflex. *Personality and Individual Differences*, **19**, 543–53.

Craddock, N., and Owen, M. J. (2005). The beginning of the end for the Kraepelinian dichotomy. *British Journal of Psychiatry*, **186**, 364–6.

de Freitas Araujo, S. (2011). Materialism's Eternal Return: Recurrent patterns of materialistic explanations of mental phenomena. In A. Moreira-Almeida and F. S. Santos (eds), *Exploring Frontiers of the Mind–Brain Relationship*. New York: Springer.

Eysenck, H. J., and Eysenck, S. B. G. (1969). *Personality Structure and Measurement*. San Diego, CA: Robert R. Knapp.

Feighner, J. P., Robins, E., Guze, S. B., et al. (1972). Diagnostic criteria for use in psychiatric research. *Archives of General Psychiatry*, **26**(1), 57–63.

Fleeson, W. (2001). Toward a structure- and process-integrated view of personality: traits as density distribution of states. *Journal of Personality and Social Psychology*, **80**(6), 1011–27.

Fossati, A., Krueger, R. F., Markon, K. E., et al. (2013). Reliability and Validity of the Personality Inventory for DSM-5 (PID-5): Predicting DSM-IV Personality Disorders and Psychopathy in Community-Dwelling Italian Adults. *Assessment*, **20**(6), 689–708.

Gardini, S., Cloninger, C. R., and Venneri, A. (2009). Individual differences in personality traits reflect structural variance in specific brain regions. *Brain Research Bulletin*, **79**(5), 265–70.

Gillespie, N. A., Cloninger, C. R., Heath, A. C., et al. (2003). The genetic and environmental relationship between Cloninger's dimensions of temperament and character. *Personality and Individual Differences*, **35**, 1931–46.

Godel, K. (1981). *Collected Works*. New York: Oxford University Press.

Goldberg, L. R. (1990). An alternative "description of personality": the big-five factor structure. *Journal of Personality and Social Psychology*, **59**(6), 1216–29.

Gusnard, D. A., Akbudak, E., Shulman, G. L., et al. (2001). Medial prefrontal cortex and self-referential mental activity: relation to a default mode of brain function. *Proceedings of the National Academy of Sciences of the United States of America*, **98**(7), 4259–64.

Gusnard, D. A., Ollinger, J. M., Shulman, G. L., et al. (2003). Persistence and brain circuitry. *Proceedings of the National Academy of Sciences of the United States of America*, **100**(6), 3479–84.

Hansenne, M., Pinto, E., Scantamburlo, G., et al. (2003). Harm avoidance is related to mismatch negativity (MMN) amplitude in healthy subjects. *Personality and Individual Differences*, **34**(6), 1039–48.

Heath, A. C., Cloninger, C. R., and Martin, N. G. (1994). Testing a model for the genetic structure of personality: a comparison of the personality systems of Cloninger and Eysenck. *Journal of Personality and Social Psychology*, **66**(4), 762–75.

Josefsson, K., Jokela, M., Cloninger, C. R., et al. (2013). Maturity and change in personality: Developmental trends of temperament and character in adulthood. *Developmental Psychopathology*, **25**(3), 713–27.

Josefsson, K., Jokela, M., Hintsanen, M., et al. (2013). Parental care-giving and home environment predicting offspring temperament and character traits after 18 years. *Psychiatry Research*, **209**(3), 643–51.

Kendell, R., and Jablensky, A. (2003). Distinguishing between the validity and utility of psychiatric diagnoses. *American Journal of Psychiatry*, **160**(1), 4–12.

Kendell, R. E. (1982). The choice of diagnostic criteria for biological research. *Arch Gen Psychiatry*, **39**(11), 1334–9.

Luty, S. E., Joyce, P. R., Mulder, R. T., et al. (1999). The relationship of dysfunctional attitudes to personality in depressed patients. *Journal of Affective Disorders*, **54**(1–2), 75–80.

Martin, R. L., Cloninger, C. R., Guze, S. B., et al. (1985a). Mortality in a follow-up of 500 psychiatric outpatients. I. Total mortality. *Archives of General Psychiatry*, **42**(1), 47–54.

Martin, R. L., Cloninger, C. R., Guze, S. B., et al. (1985b). Mortality in a follow-up of 500 psychiatric outpatients. II. Cause-specific mortality. *Archives of General Psychiatry*, **42**(1), 58–66.

Maser, J. D., and Cloninger, C. R. (1990). *Comorbidity of Mood and Anxiety Disorders*. Washington, DC: American Psychiatric Press.

Moreira-Almeida, A., and Santos, F. S. (eds) (2011). *Exploring Frontiers of the Mind–Brain Relationship*. New York: Springer.

Narrow, W. E., Rae, D. S., Robins, L. N., et al. (2002). Revised prevalence estimates of mental disorders in the United States: using a clinical significance criterion to reconcile 2 surveys' estimates. *Archives of General Psychiatry*, **59**(2), 115–23.

Pezawas, L., Meyer-Lindenberg, A., Drabant, E. M., et al. (2005). 5-HTTLPR polymorphism impacts human cingulate–amygdala interactions: a genetic susceptibility mechanism for depression. *Nature Neuroscience*, **8**(6), 828834.

Regier, D. A., Narrow, W. E., Clarke, D. E., et al. (2013). DSM-5 field trials in the United States and Canada, Part II: test–retest reliability of selected categorical diagnoses. *American Journal of Psychiatry*, **170**(1), 59–70.

Richter, J., and Eisemann, M. (2002). Self-directedness as a cognitive feature in depressive patients. *Personality and Individual Differences*, **32**(8), 1327–37.

Robins, E., and Guze, S. B. (1970). Establishment of diagnostic validity in psychiatric illness: its application to schizophrenia. *American Journal of Psychiatry*, **126**(7), 983–7.

Rogers, C. R. (1995). *On Becoming a Person: A therapist's view of psychotherapy*. Boston: Houghton Mifflin.

Rosenberg, R. S. (2013). Abnormal is the New Normal. *Slate*.

Rosenstrom, T., Jokela, M., Cloninger, C. R., et al. (2012). Associations between dimensional personality measures and preclinical atherosclerosis: The cardiovascular risk in Young Finns study. *Journal of Psychosomatic Research*, **72**(5), 336–43.

Samuel, D. B., and Widiger, T. A. (2004). Clinicians' personality descriptions of prototypic personality disorders. *Journal of Personality Disorders*, **18**(3), 286–308.

Seligman, M. E. (2011). *Flourish*. New York: Free Press.

Shankland, R. S. (1964). Michelson–Morley Experiment. *American Journal of Physics*, **32**, 16–35.

Smith, M. J., Cloninger, C. R., Harms, M. P., et al. (2008). Temperament and character as schizophrenia-related endophenotypes in non-psychotic siblings. *Schizophrenia Research*, **104**(1–3), 198–205.

Stallings, M. C., Hewitt, J. K., Cloninger, C. R., et al. (1996). Genetic and environmental structure of the Tridimensional Personality Questionnaire: three or four temperament dimensions? *Journal of Personality and Social Psychology*, **70**(1), 127–40.

Strack, S. (ed.) (2006). *Differentiating Normal and Abnormal Personality*, Second edition. New York: Springer Publishing.

Sussman, R. W., and Cloninger, C. R. (eds) (2011). *Origins of Cooperation and Altruism*, First edition, Vol. 36. New York: Springer.

Svrakic, D. M., Whitehead, C., Przybeck, T. R., et al. (1993). Differential diagnosis of personality disorders by the seven-factor model of temperament and character. *Archives of General Psychiatry*, **50**(12), 991–9.

Svrakic, D. M., Zorumski, C. F., Svrakic, N. M., et al. (2013). Risk architecture of schizophrenia: the role of epigenetics. *Current Opinion in Psychiatry*, **26**(2), 188–95.

Thomas, K. M., Yalch, M. M., Krueger, R. F., et al. (2012). The convergent structure of DSM-5 personality trait facets and five-factor model trait domains. *Assessment*, **20**(3), 308–11.

Tulving, E. (1987). Multiple memory systems and consciousness. *Human Neurobiology*, **6**(2), 67–80.

Tulving, E. (2002). Episodic memory: from mind to brain. *Annual Review of Psychology*, **53**, 1–25.

Vowles, K. E., McCracken, L. M., and O'Brien, J. Z. (2011). Acceptance and values-based action in chronic pain: a three-year follow-up analysis of treatment effectiveness and process. *Behavior Research and Therapy*, **49**(11), 748–55.

Waldrop, M. M. (1992). *Complexity: The Emerging Science at the Edge of Order and Chaos*. New York: Simon & Schuster.

Zohar, A. H., Cloninger, C. R., and McCraty, R. (2013). Personality and Heart Rate Variability: Exploring pathways from personality to cardiac coherence and health. *Open Journal of Social Science*, **1**(6), 32–9.

Chapter 13

Person-centered integrative diagnosis: Bases, models, and guides

Juan E. Mezzich and Ihsan M. Salloum

13.1 Introduction

The international and institutional development of person-centered psychiatry and medicine has made compelling the development of a diagnostic approach relevant to this new perspective in medicine and health. And the methodological unfolding of diagnostic schemas over recent decades provides some of the tools for the conceptualization and design of a suitable diagnostic approach for person-centered medicine. The next sections briefly describe the bases for a person-centered integrative diagnostic model and the subsequent practical guides that are emerging.

13.2 Bases for the Development of Person-Centered Integrative Diagnosis

Highlighted here as major bases for Person-centered Integrative Diagnosis is the development of person-centered medicine and new comprehensive diagnostic methodology.

13.2.1 Person-Centered Medicine

The first bases preceding and supporting the development of Person-centered Integrative Diagnosis are the coalescing initiatives for person-centered medicine and health. Historical roots for person-centered care can be found in major Eastern civilizations, particularly Chinese and Ayurvedic, which are still alive and practiced today as traditional medicine. Both of them articulate a comprehensive and harmonious framework of health and life, and promote a highly personalized approach for the treatment of specific diseases and the enhancement of quality of life (Patwardhan et al. 2005). In the West, the need for holism in medicine has been strongly advocated by ancient Greek philosophers and

physicians. Socrates and Plato taught that "if the whole is not well it is impossible for the part to be well" (Christodoulou 1987). This position was enriched by Aristotle, the philosopher and naturalist *par excellence* (Ierodiakonou 2011), and by Hippocrates, who brought theory, emotion, and individuality into the practice of medicine and delineated its ethical and person-centered foundations (Jouanna 1999).

Such broad and enlightened concept of health (full well-being and not only the absence of disease) has been incorporated into WHO's (1946) definition of health. This notion has maintained its vitality throughout the vicissitudes of contemporary health care. Modern medicine has brought a number of important advances in the scientific understanding of diseases and the development of valuable technologies for diagnosis and treatment. At the same time, it has led to a hyperbolic, impersonal, and dehumanizing focus on disease, over-specialization of medical disciplines, fragmentation of health services, weakening of the doctor–patient relationship, and commoditization of medicine (Heath 2005).

In response, proposals for re-prioritizing medicine as person-centered are emerging, which cover a wide range of concepts, tasks, technologies, and practices that aim to put the whole person in context as a center of clinical practice and public health. The World Psychiatric Association, which was born from the articulation of science and humanism (Garrabe and Hoff 2011), established at its 2005 General Assembly an Institutional Program on Psychiatry for the Person (Mezzich 2007; Christodoulou et al. 2008).

This initiative expanded into general medicine through a series of Geneva Conferences since 2008 in collaboration with the World Medical Association, the World Health Organization, the International Council of Nurses, the International Federation of Social Workers, the International Pharmaceutical Federation, the European Federation of Families of Persons with Mental Illness, and the International Alliance of Patients' Organizations, among a growing number of other international health institutions (Mezzich 2011a). The process and impact of the Geneva Conferences led to the emergence of the International Network (recently renamed College) of Person-centered Medicine (INPCM, ICPCM) (Mezzich, Snaedal, et al. 2009; Mezzich 2011b). The ICPCM launched the *International Journal of Person-Centered Medicine* in collaboration with the University of Buckingham Press (Miles and Mezzich 2011). It is promoting research and scholarship on person-centered medicine across the world.

The unfolding of the core concept of person-centered medicine is taking several forms. One is that it represents a medicine *of the person* (of the totality of the person's health), *for the person* (aimed at promoting the person's total health and well-being and facilitating the fulfillment of his/her life project),

by the person (with health professionals extending themselves as full persons professionally competent and with high ethical aspirations), and *with the person* (collaborating respectfully and in an empowering manner with all persons involved) (Mezzich, Snaedal, et al. 2010). Another form, through a broad international consultation project aimed at elucidating the key concepts underlying person-centered medicine, encompasses ethical commitment, holistic scope, cultural sensitivity, relationship focus, individualized treatment, common ground for diagnosis and care, people-centered systems of care, and person-centered health education and research (Mezzich et al. 2013).

13.2.2 Comprehensive Diagnostic Methodology

Addressing the nature of diagnosis, the eminent historian and philosopher of medicine Lain Entralgo (1982) cogently argued that diagnosis goes beyond identifying a disease (*nosological diagnosis*) to also involve *understanding* of what is going in the body and mind of the person who presents for care. Understanding an individual's clinical condition also requires a broader assessment of his/her experience and life context. As health may be conceived as a person's capacity to continue to pursue his or her goals in an ever-challenging world (Canguilhem 1991), this encompassing perspective must incorporate a thorough diagnosis of health. There are indeed compelling reasons, in consistency with WHO's definition of health, for including health-promoting or *salutogenic* factors (Antonovsky 1987) and positive health (Mezzich 2005) under comprehensive diagnosis. Diagnostic understanding also requires a process of engagement and empowerment that recognizes the agency of patient, family, and health professionals participating in a *trialogical* partnership (Amering 2010).

In connection with the more encompassing model, one should examine the concept of the *validity* of diagnosis as it denotes its value and usefulness. Traditionally, this validity has been anchored on the faithfulness and accuracy with which a diagnosis reflects and identifies a disorder, its nature, pathophysiology, and other biomedical indicators (Robins and Guze 1978). Recently, *clinical utility* has been proposed as an additional indication of the value of diagnosis for clinical care (Kendell and Jablensky 2003). Schaffner (2009) has delineated further the epistemology of these two forms of diagnostic validity under the terms of *etiopathogenic and clinical validities*. Emphasizing the significance of the latter, experienced clinicians have suggested that treatment planning is the most important purpose of diagnosis (Adams and Grieder 2005).

Concerning the architecture of diagnostic formulations, there has been a progressive development of diagnostic schemas with increasing levels of

informational richness to support treatment planning. These schemas have ranged from a simple, typological *single-label diagnosis* denoting a symptom, problem, syndrome, or illness, to a more complex *multiple-illness formulation* listing all identified clinical conditions or disorders, including coexisting psychiatric and general medical diseases. Such schemas provide a fuller portrayal of the nosological condition, as well as other aspects of clinical interest such as disabilities, contextual factors, and quality of life, thus attempting to enhance diagnostic understanding, treatment planning, and prognostic determination (Banzato et al. 2009). Multiaxial diagnostic formulations are key components of most recent diagnostic systems including ICD-10 (World Health Organization 1996, 1997), DSM-IV (American Psychiatric Association 1994), GLADP (APAL 2004), GC-3 (Otero 1998), the French Classification for Child and Adolescent Mental Disorders (Mises et al. 2002), and the Chinese Classification of Mental Disorders (Chinese Society of Psychiatry 2001). Of note, a multiaxial schema was not included in DSM-5 (American Psychiatric Association 2013), despite that an APA Committee established to evaluate DSM multiaxial systems documented their usefulness (Mezzich et al. 2005).

Another approach to comprehensive diagnosis is that composed of both standardized and idiographic components. One such model is at the core of the International Guidelines for Diagnostic Assessment (IGDA), developed by the World Psychiatric Association (Mezzich et al. 2003). Its standardized multiaxial component includes four axes dealing respectively with clinical disorders, disabilities, contextual factors, and quality of life. Its idiographic and narrative component covers the clinician perspective, perspectives of the patient and family, and integration of the perspectives of all the above. Many of the methodological developments highlighted here have been discussed in a WPA Psychiatry for the Person volume (Salloum and Mezzich 2009).

13.3 Development and Structure of the Person-Centered Integrative Diagnosis Model

13.3.1 Design Considerations

Person-centered Integrative Diagnosis (PID), as developed under the auspices of the International College of Person-Centered Medicine, is inscribed within a paradigmatic effort to place the whole person at the center of medicine and health care (Mezzich, Snaedal, et al. 2009; Mezzich 2011b). The PID model integrates science and humanism to obtain, as previously mentioned with regard to person-centered medicine in general, a diagnosis of the person (of the totality of the person's health, both its ill and positive aspects), by the person, for the person, and with the person (Mezzich et al. 2010). This diagnostic

perspective goes beyond the restricted concepts of nosological and differential diagnoses on which conventional diagnostic systems are based—such as the WHO's International Classification of Disease and the American Psychiatric Association's Diagnostic and Statistical Manuals. The development of the PID diagnostic model was informed by the methodological considerations summarized in the preceding section.

The suitability of the prospective elements of PID were examined through surveys and consultations. Building on its long experience in developing diagnostic models, the World Psychiatric Association (WPA) Section on Classification, Diagnostic Assessment and Nomenclature conducted a survey among the members of the 43-country Global Network of National Classification and Diagnosis Groups (Salloum and Mezzich 2011). The survey was constructed in consultation with network members and aimed to identify the most important domains to consider in the development of future diagnostic classification for psychiatric disorders; 74 percent of the groups responded. Treatment planning was most frequently chosen as the key purpose of diagnosis. Communication among clinicians and diagnosis as a means to enhance illness understanding were also identified as key. The survey highlighted the areas of information judged important to be covered by psychiatric diagnosis. These included disorders (100 percent), disabilities (74 percent), risk factors (61 percent), experience of illness (58 percent), protective factors (55 percent), and experience of health (52 percent). The responses suggested that in addition to the recognized importance of nosological diagnosis, subjective explanatory narratives of illness and health are also quite valuable. The survey responses also highlighted the importance of utilizing a variety of descriptive tools including categories (81 percent), dimensions (74 percent), and narratives (45 percent). It also revealed that 80 percent of responders preferred that clinicians, patients, and caregivers work together as key players in the diagnostic evaluation process as compared to clinicians working alone (20 percent).

A number of focus and discussion groups were organized in 2009 with a variety of health stakeholders (health professionals, patients, family members, and advocates) at international events in Athens (Greece), Uppsala (Sweden), and Timisoara (Romania) (Salloum and Mezzich 2011). In an overwhelming manner, the participants in the three settings indicated that diagnosis should go beyond disease. Participants unanimously responded that diagnosis should cover dysfunctions and a great majority of them (over 83 percent) believed that it is very important to include positive aspects of health. Furthermore, there was unanimous agreement on incorporating contributing factors (including risk and protective factors), and on the use of descriptive methods, including dimensions and narratives in addition to conventional diagnostic categories.

Participants also emphasized that diagnosis is a process and not only a formulation, and highlighted the partnership between caregivers and service users as fundamental.

13.3.2 Structure of the Model

The delineation of the structure of the Person-centered Integrative Diagnosis model (PID) must take into account that diagnosis is both a formulation and a process. The presentation of the fundamental elements of the model include the following three defining conceptual pillars: a) Broad Informational Domains, b) Pluralistic Descriptive Procedures, and c) Partnership for Evaluation.

The PID framework's first pillar, *Broad Informational Domains or Levels*, is depicted in Figure 13.1. These domains cover both ill health and positive health along three structural levels: Health Status, Experience of Health, and Contributors to Health.

The broadness of the PID informational domains, including ill and positive health, is intrinsic to holistic person-centered health care. The domain level on *Health Status* includes first illnesses or disorders of both mental and physical forms, which correspond to Laín-Entralgo's (1982) nosological diagnosis. They would be assessed according to the international standard, WHO's International Classification of Diseases, or a pertinent national or regional version or adaptation. Disabilities would be assessed through procedures such as

Ill Health	**Positive Health**
I. Health Status	
Illness & its Burden	**Well Being**
Disorders	Recovery/Wellness
Disabilities	Functioning
II. Experience of Health	
Experience of Illness	**Experience of Health**
(e.g., suffering, values, perception understanding & meaning of illness)	(e.g., identity, contentment, & fulfillment)
III. Contributors to Health	
Contributors to Illness	**Contributors to Health**
(intrinsic/extrinsic: biological psychological, social	(intrinsic/extrinsic: biological psychological social)

Fig. 13.1 Broad informational domains or levels covering ill health and positive health in the Person-centered Integrative Diagnosis model

those based on the International Classification of Functioning and Health (ICF) (World Health Organization 2001). The assessment of the well-being aspect of Health Status could be conducted through standard scales such as the WHO QOL Instrument (WHO QOL Group 1994).

The domain level on *Experience of Health* would appraise the patient's illness- and health-related values and cultural experiences, possibly with a guided narrative procedure built on worldwide experience with the Cultural Formulation (Mezzich, Caracci, et al. 2009). The third domain level on *Contributors to Health* would cover a range of intrinsic and extrinsic biological, psychological, and social factors of both risk and protective types. Their assessment may involve a combination of procedures aimed at assessing healthy and unhealthy lifestyle factors and related health contributors (Seyer 2012).

The PID model's second defining pillar, *Pluralistic Descriptive Procedures*, opens up the opportunity to employ categories, dimensions, and narratives for greater flexibility and effectiveness for the evaluation task at hand (Jablensky 2005; Kirmayer 2000). The third defining pillar of the PID model is *Partnership for Evaluation*. Such partnership is a fundamental element of person-centered care, and involves the pursuit of engagement, empathy, and empowerment, as well as respect for the autonomy and dignity of the consulting person. In fact, it is crucial for achieving shared understanding for diagnosis and shared decision making for treatment planning (Adams and Grieder 2005). Additional information on the elements of the PID model can be found in Mezzich, Salloum, et al. (2009).

13.4 Practical Guides Applying the PID Model

13.4.1 The Latin American Guide for Psychiatric Diagnosis

The diagnostic model prepared and published by the Latin American Psychiatric Association (2012) at the core of the Latin American Guide of Psychiatric Diagnosis, Revised Version (GLADP-VR) (Asociación Psiquiátrica de América Latina 2012) (see Figure 13.2) was built starting with the original GLADP (Guia Latinoamericana de Diagnostico Psiquiatrico) (Asociación Psiquiátrica de América Latina 2004) and largely incorporated the basic elements of the PID. The main difference between the PID model and the GLADP-VR schema is that the former has Health Experience as the second informational domain level while the latter has Health Experience (enriched with health values and expectations) as the third level. Another major difference is of course that while the PID is a theoretical model, the GLADP-VR is a practical guide.

The key information domains or levels of the GLADP-VR diagnostic schema are now summarized.

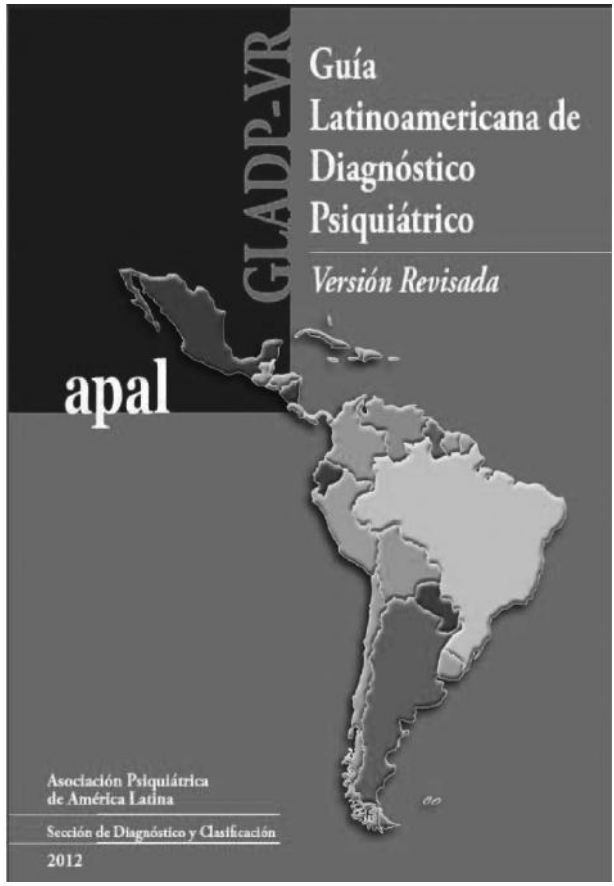

Fig. 13.2 Cover of the Latin American Guide of Psychiatric Diagnosis, Revised Version (GLADP-VR). © Asociación Psiquiátrica de América Latina (APAL).

13.4.2 Health Status

The first component of this model corresponds to Health Status. This includes standardized coverage of pathological and positive aspects of health. Utilizing a Personalized Diagnostic Formulation, this component starts with a listing of mental and general medical disorders and other significant clinical conditions. These disorders and conditions are to be coded according to the various chapters of ICD-10, including, in addition to standard disease codes, the Z codes for non-disease conditions that require clinical attention.

Next comes the evaluation of Personal Functioning in the areas of personal care, occupational, family, and social activities, each measured with a 10-point

scale marked as follows: 0: worst functioning, 2: minimal functioning, 4: marginal functioning, 6: acceptable functioning, 8: substantial functioning, and 10: optimal functioning.

Finally, the Health Status component assesses the degree of the person's well-being, from worst to excellent, by directly marking on the 10-point line displayed on the form or with the help of an appropriate standardized instrument. This assessment is principally based on the judgment of the person involved, modulated collaboratively with perceptions by the clinicians and family.

13.4.3 Health Contributing Factors

The second component of the Personalized Diagnostic Formulation corresponds to Health Contributing Factors. These include Risk Factors as well as Protective and Health Promotion Factors. Assessment in each case starts with the identification of relevant factors from the list presented on the form. These factors come from the Health Improvement Card prepared by the World Health Professions Alliance (Seyer 2012), supplemented by some factors particularly relevant to mental health. It continues with a narrative formulation of additional information about the identified factors and others that could also be elicited.

13.4.4 Health Experiences and Expectations

The third component of the GLADP-VR Personalized Diagnostic Formulation assesses Experience and Expectations on Health. This is based on the combination of elements of the experientially described Cultural Formulation (Mezzich, Caracci, et al. 2009) and of the patient's needs and preferences (Fulford et al. 2011). This assessment is obtained through the narrative presentation of the following three points: a) Personal and cultural identity (self-awareness and its potentials and limitations); b) Suffering (its recognition, idioms of distress, and beliefs on illness); and c) Experiences with and expectations for health care (Mezzich 2012).

13.4.5 Prospective Guides

A renewed Second Edition of the Latin American Guide for Psychiatric Diagnosis (GLADP-2) is in the works as a priority project of the Diagnosis and Classification Section of the Latin American Psychiatric Association (APAL). For covering mental and general medical disorders it would be based on the categories and codes of the prospective Eleventh Revision of the International Classification of Diseases (ICD-11), which is expected to be completed around 2015. Its development would be based on the ongoing experience of implementing, teaching, and studying the GLADP-VR.

There are also plans to develop under the auspices of the International College of Person-Centered Medicine PID practical guides intended for use in general medicine (including psychiatry).

13.5 Conclusions

Within the framework of a paradigmatic initiative for person-centered medicine, building on modern diagnostic methodology developments, and expanding the concept of diagnostic validity, a model for PID has been developed. It addresses the diagnosis of a person's total health through three informational levels (health status, health contributors, and health experience and expectations), the utilization of categories, dimensions, and narratives as descriptive instruments, and the interactive engagement of clinicians, patients, and families in the diagnostic process. This model is broader than ICD and DSM, which are focused on classifying and diagnosing only illness and do not have the other features mentioned earlier. The PID model has been applied recently in the latest version of the Latin American Guide for Psychiatric Diagnosis (GLADP-VR) published by the Latin American Psychiatric Association. The model is also being engaged in the preparation of other practical guides for general medical diagnosis under the auspices of the International College of Person-Centered Medicine.

References

Adams N., and Grieder D. M. (2005). *Treatment Planning for Person-Centered Care*. Amsterdam: Elsevier.

American Psychiatric Association (1994). *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (DSM-IV). Washington, DC: American Psychiatric Association.

American Psychiatric Association (2013). *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (DSM-5). Arlington, VA: American Psychiatric Association.

Amering, M. (2010). Trialog—An exercise in communication between consumers, carers, and professional mental health workers beyond role stereotype. In *Conceptual Explorations on Person-centered Medicine. International Journal of Integrated Care*, **10**, Supplement 10.

Antonovsky, A. (1987). *Unraveling the Mystery of Health*. San Francisco: Jossey-Bass.

Asociación Psiquiátrica de América Latina (2004). *Guía Latinoamericana de Diagnóstico Psiquiátrico*. Guadalajara: Asociación Psiquiátrica de América Latina, Sección de Diagnóstico y Clasificación.

Asociación Psiquiátrica de América Latina (2012). *Guía Latinoamericana de Diagnóstico Psiquiátrico, Versión revisada (GLADP-VR)*. Lima: Asociación Psiquiátrica de América Latina, Sección de Diagnóstico y Clasificación.

Banzato C. E. M., Jorge, M. R., and Kastrup, M. (2009). Multiaxial schemas for psychiatric diagnosis. In I. M. Salloum and J. E. Mezzich (eds), *Psychiatric Diagnosis: Challenges and Prospects*. Chichester, UK: Wiley-Blackwell.

Canguilhem, G. (1991). *The Normal and the Pathological*. New York: Zone Books.

Chinese Society of Psychiatry (2001). *Chinese Classification of Mental Disorders*, Version 3 (CCMD-3). Jinan: Chinese Society of Psychiatry.

Christodoulou G. N. (ed.) (1987). *Psychosomatic Medicine*. New York: Plenum.

Christodoulou G. N., Fulford, K. M. W., and Mezzich, J. E. (2008). Conceptual bases of Psychiatry for the Person. *International Psychiatry*, **5**(1), 1–3.

Fulford, K. W. M., Christodoulou, G. N., and Stein, D. J. (2011). Values and ethics: Perspectives on psychiatry for the person. *International Journal of Person Centered Medicine*, **1**, 131–3.

Garrabe, J., and Hoff, P. (2011). Historical views on psychiatry for the person. *International Journal of Person Centered Medicine*, **1**, 125–7.

Heath, I. (2005). Promotion of disease and corrosion of medicine. *Canadian Family Physician*, **51**, 1320–2.

Ierodiakonou, C. S. (2011). *The Psychology of Aristotle, The Philosopher*. London: Karnak.

Jablensky, A. (2005). Categories, dimensions and prototypes: Critical issues for psychiatric classification. *Psychopathology*, **38**, 201–5.

Jouanna, J. (1999). *Hippocrates*. Translated by M. B. Debevoise. Baltimore: Johns Hopkins University Press.

Kendell, R., and Jablensky, A. (2003). Distinguishing between the validity and the utility of psychiatric diagnoses. *American Journal of Psychiatry*, **160**, 4–12.

Kirmayer, L. J. (2000). Broken narratives: Clinical encounters and the poetics of illness experience. In C. Mattingly and L. Garro (eds), *Narrative and the Cultural Construction of Illness and Healing* (pp. 153–80). Berkeley: University of California Press.

Laín-Entralgo, P. (1982). *El Diagnóstico Médico: Historia y Teoría*. Barcelona: Salvat.

Mezzich J. E. (2005). Positive health: Conceptual place, dimensions and implications. *Psychopathology*, **38**, 177–9.

Mezzich, J. E. (2007). Psychiatry for the person: articulating medicine's science and humanism. *World Psychiatry*, **6**, 65–7.

Mezzich, J. E. (2011a). The Geneva Conferences and the emergence of the International Network of Person-centered Medicine. *Journal of Evaluation in Clinical Practice*, **17**, 333–6.

Mezzich, J. E. (2011b). The construction of person-centered medicine and the launching of an International College. *International Journal of Person Centered Medicine*, **2**, 6–10.

Mezzich, J. E. (2012). Towards a health experience formulation for person-centered integrative diagnosis. *International Journal of Person Centered Medicine*, **2**, 188–92.

Mezzich J. E., Banzato, C. E. M., Cohen P., et al. (2005). Report of the American Psychiatric Association Committee to Evaluate the DSM Multiaxial System. Presented to the APA Assembly, Atlanta, May 21, 2005.

Mezzich, J. E., Berganza, C. E., von Cranach, M., Jorge, M. R., Kastrup, M. C., Murthy, R. C., Okasha, A., Pull C., Sartorius, N., Skodol, A. E., and Zaudig, M. (2003). Essentials of the WPA International Guidelines for Diagnostic Assessment (IGDA). *British Journal of Psychiatry*, **182**, Suppl. 45.

Mezzich, J. E., Caracci, G., Fabrega, H., et al. (2009). Cultural formulation guidelines. *Transcultural Psychiatry*, **46**, 383–405.

Mezzich, J. E., Kirisci, L., and Salloum, I. M. (2013). Towards a systematic conceptualization of person-centered medicine. Paper presented at the 6th Geneva Conference of Person Centered Medicine, April 29, 2013.

Mezzich, J. E., Salloum, I. M., Cloninger, C. R., et al. (2010). Person-centered integrative diagnosis: conceptual bases and structural model. *Canadian Journal of Psychiatry*, **55**, 701–8.

Mezzich, J. E., Snaedal, J., van Weel, C., et al. (2009). The international network for person-centered medicine: background and first steps. *World Medical Journal*, **55**, 104–7.

Mezzich J. E., Snaedal, J., van Weel, C., et al. (2012). Introduction to conceptual explorations on person-centered medicine. In J. E. Mezzich, J. Snaedal, C. van Weel, et al. (eds), Conceptual Explorations on Person-centered Medicine. *International Journal of Integrated Care*, Supplement, Vol. 10.

Miles A., and Mezzich, J. E. (2011). Advancing the global communication of scholarship and research for personalized healthcare. *The International Journal of Person Centered Medicine*, **1**, 1–5.

Mises, R., Quemada, N., Botbol, M., et al. (2002). French Classification for Child and Adolescent Mental Disorders (CFTMEA). *Psychopathology*, **35**, 176–80.

Otero, A. A. (1998). *Tercer Glosario Cubano de Psiquiatría (GC-3)*. La Habana: Hospital Psiquiátrico de La Habana.

Patwardhan, B., Warude, D., Pushpangadan, P., et al. (2005). Ayurveda and traditional Chinese medicine: a comparative overview. *Evidence-based Complementary and Alternative Medicine*, **2**, 465–73.

Robins E., and Guze, S. (1970). Establishment of diagnostic validity in psychiatric illness: its application to schizophrenia. *American Journal of Psychiatry*, **126**(7), 983–7.

Salloum I. M., and Mezzich, J. E. (2009). *Psychiatric Diagnosis: Challenges and Prospects*. Chichester, UK: Wiley-Blackwell.

Salloum, I. M., and Mezzich, J. E. (2011). Conceptual appraisal of the person-centered integrative diagnosis model. *International Journal of Person Centered Medicine*, **1**, 39–42.

Schaffner, K. F. (2009) The validity of psychiatric diagnosis: Etiopathogenic and clinical approaches. In I. M. Salloum and J. E. Mezzich (eds), *Psychiatric Diagnosis: Challenges and Prospects*. Chichester, UK: Wiley-Blackwell.

Seyer, J. (2012). Development of the Health Improvement Card developed by the World Health Professions Alliance. Paper presented at the 5th Geneva Conference on Person-centered Medicine. International College of Person Centered Medicine, May 2012.

WHO QOL Group (1994). The development of the World Health Organization quality of life assessment instrument (the WHO QOL). In J. Orly and W. Kuyken (eds), *Quality of Life Assessment: International Perspectives*. Heidelberg: Springer Verlag.

World Health Organization (1946). *Constitution of the World Health Organization*. Geneva: World Health Organization.

World Health Organization (1996). *Multiaxial Classification of Child and Adolescent Psychiatric Disorders*. Cambridge, UK: Cambridge University Press.

World Health Organization (1997). *Multiaxial Presentation of the ICD-10 for Use in Adult Psychiatry*. Cambridge, UK: Cambridge University Press.

World Health Organization (2001). *International Classification of Functioning and Health (ICF)*. Geneva: World Health Organization.

Chapter 14

The four domains of mental illness (FDMI): An alternative to the DSM-5

René J. Muller

14.1 Introduction

Between 1994 and 2004, I evaluated over three thousand psychiatric patients in the emergency room at three hospitals in Baltimore (Muller 2003). About halfway through this decade, I began to realize that at least half of my patients who had previous psychiatric treatment carried a wrong diagnosis. Patient after patient, describing the crisis that brought them to the ER, came clean with me about what was going on in their lives. Many actually knew they did not have the mental illness they had been diagnosed with and didn't need the medication they had been prescribed, and, in many cases, were still taking. Not only were these patients being treated for a mental illness they didn't have, their real problem was going unaddressed.

Eventually, I came to see that the method fostered by the American Psychiatric Association for diagnosing mental illness—the *Diagnostic and Statistical Manual of Mental Disorders*, at that time the DSM-IV (1994)—was a virtual invitation for clinicians to get a diagnosis wrong. In 2008 I published *Doing Psychiatry Wrong: A Critical and Prescriptive Look at a Faltering Profession* (Muller 2008), where I spelled out the damage that I saw done to patients who were misdiagnosed. Though many clinicians had hoped that the DSM-5, which derives from the same paradigm as the third and fourth editions, would be a better guide to making valid psychiatric diagnoses, it became clear to me that this was not the case (Muller 2013).

14.2 The DSM-5

The official rollout of the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5) at the May 2013 meeting of the American Psychiatric Association included the acknowledgment by the Task Force that their primary goal—producing a guide that would permit the diagnosis of mental illnesses

to be based on a detectable biological cause—had not been met. Days before the official unveiling of the DSM-5, a number of psychiatrists who were closely associated with the project scrambled to do some preemptory damage control, mostly by lowering expectations for what was to come.

Michael B. First, professor of psychiatry at Columbia University, acknowledged on National Public Radio that there was still no empirical method to confirm or rule out any mental illness: "We were hoping and imagining that research would advance at a pace that laboratory tests would have come out. And here we are 20 years later and we still unfortunately rely primarily on symptoms to make our diagnoses" (Hamilton 2013).

Speaking to the *New York Times*, Thomas R. Insel, director of the National Institutes of Mental Health, insisted that this failure had not been for lack of effort: "We've tried. You know we've actually looked—using brain imaging, using various endocrine tests, looking at a range of other kinds of biomarkers. So far that has been found wanting" (Belluck and Carey 2013).

In the same *Times* article, David J. Kupfer, chairman of the DSM-5 Task Force, admitted "a failure of our neuroscience and biology to give us the level of diagnostic criteria, a level of sensitivity and specificity that we would be able to introduce into the diagnostic manual." Kupfer, Insel, and First agree that the new paradigm envisioned for psychiatry—the reason the new edition was undertaken—remains elusive.

Nonetheless, four months after publication, following many lukewarm and negative reviews, the DSM-5 had sold 382,000 copies and brought in $33.7 million. Over the previous ten years, the fifth edition had cost the APA about $22 million to produce.

14.3 Adolf Meyer's Psychobiology and the Johns Hopkins Perspectives of Psychiatry

Largely eschewing the DSM's paradigm, which rests on the notion that all mental illnesses are brain diseases, the department of psychiatry at the Johns Hopkins University School of Medicine continues to follow Adolf Meyer's psychobiological approach to diagnosing and treating mental illness that was instituted by Meyer while he was chief of psychiatry there from 1910 to 1941. In 2013 Johns Hopkins celebrated the centennial of the Henry Phipps Clinic, created by Meyer to foster his vision for mental health.

In Meyer's psychobiology—the psychological study of the person in the context of biology—every facet of life is simultaneously and inseparably both psychological and biological. The Johns Hopkins (and later Harvard) psychiatrist Leon Eisenberg famously remarked that asking how much of what people do is psychological and how much is biological is as unproductive as asking how

much of the area of a rectangle derives from its width and how much from its height (Eisenberg 1995). To psychobiologists, the terms *psychological* and *biological* do not signify a separation of mind and brain, but specify two polarities of a single unit, the person. To existentialists, this entity is *the self as active agent*, a designation that Meyer would have surely endorsed.

Meyer insisted that most mental illnesses emerge out of lives dynamically rather than impinge on them biologically. He did not believe that the majority of these illnesses were brain diseases, or were passed along genetically. In his psychobiological synthesis, Meyer saw the mind as the organ of personality. He had already grasped the modern notion of epigenesis—that a person's experience in the world, with others, shapes a life, simultaneously altering brain structure and function. This "biological" alteration may then influence "psychological" development, opening a "two-way street" that remains open through the life course. In this psychobiological view of self-creation, the "man-made"—and ultimately invalid—distinction between psychology and biology disappears.

In 1998 Paul R. McHugh and Phillip R. Slavney, professors of psychiatry at Johns Hopkins, published the second edition of their Meyerian treatise *The Perspectives of Psychiatry* (McHugh and Slavney 1998). Their intention was to systematize Meyer's often vague, unstructured notions about mental illness so as to make his psychobiological approach more accessible to clinicians and clinical researchers. They began by distinguishing four different types of psychopathology, based on etiology and the meaning of symptoms, distinctions that were purposely omitted in the DSM-III (1980), DSM-IV (1994), and DSM-5 (2013).

If not all mental illnesses are brain diseases, the question then becomes how do these illnesses come to be? Who is at risk, and why? The Johns Hopkins perspectives offer four different *directions* from which to view a patient's pathological predicament based on etiology, as far as this can be discerned given our present state of knowledge. Clinicians are prompted to look down four different axes, so to speak, to see how a person's mental life has been pathologically altered.

14.4 The Four Domains of Mental Illness (FDMI)

Since 2011, in consultation with Paul McHugh, now university distinguished professor of psychiatry at Johns Hopkins, I have been working on a guide for classifying and diagnosing mental illness—*The Four Domains of Mental Illness* (FDMI)—that is rooted in *The Perspectives of Psychiatry* and Meyer's psychobiology. The FDMI does away with the DSM's checklists of mostly behavioral symptoms, which were originally justified by the promise of finding a biological cause

for every mental illness, in favor of identifying the altered emotions, thoughts, and acts that constitute the *phenomenon* of each illness. The heart of this process is establishing, as far as possible, the psychobiological origin of the phenomenon, along with its meaning. Meyer implicitly took this phenomenological approach to diagnosis, as did McHugh and Slavney in *The Perspectives of Psychiatry*.

The DSM-III, DSM-IV, and DSM-5 were modeled on the idea of Emil Kraepelin (1856–1926) that mental illnesses are *categorical* disease entities, syndromes comprised of *symptoms* that demarcate one illness from another. (Hence the pie charts and Venn diagrams that show the "overlap" of symptoms and the "comorbidity" for different illnesses.) In contrast, the perspectives and the FDMI are based on a *dimensional* model of mental illness, which sees thought, emotion, and behavior as falling along a continuum from normal to aberrant, where pathology is defined as a phenomenon that occurs beyond a certain point in that continuum.

Each of the four perspectives identified by McHugh and Slavney implies a corresponding pathological *domain*, a lived space created and inhabited by someone with a mental illness. Meyer, who was an existentialist in all but name, would have surely agreed that a psychiatric diagnosis should access and assess a patient's *lived experience*—colloquially, where the patient lives. (*Domain* derives from the Latin *territory*.) The FDMI also allows the clinician who is evaluating a patient for an abnormality in one domain to simultaneously assess deviances in the other domains, which may be contributing to the patient's altered mental life.

Viewing mental illness from four perspectives and defining the corresponding domains—a domain is a part of the patient's disturbed world that a perspective invites us to enter—permits a "first cut" to be made in parsing the varieties of psychopathological phenomena (Table 14.1). This four-part division is a considerable refinement over the divisions made by Meyer, and in the DSM-I (1952), which is largely based on Meyer's psychobiology. It is also a different kind of sorting. Each perspective in *The Perspectives of Psychiatry*, and each domain in the FDMI, derives from a psychobiological deviation from normalcy that can be distinguished from deviations in the other three domains. Not infrequently, deviations in multiple domains contribute to a person's illness.

Because, for the FDMI, it seemed sensible to consider the most prevalent mental illnesses first, the order of the domains that derive from the first and fourth perspectives were interchanged. This change also permits the discussion of mental illness to begin with anxiety, rather than with brain diseases as is the case in *The Perspectives of Psychiatry*—a desirable strategy since pathological reactions to anxiety are generally seen as the root of all psychogenic illness. Chisolm and Lyketsos had previously made this switch in their book *Systematic Psychiatric Evaluation: A Step-by-Step Guide to Applying "The Perspectives of Psychiatry"* (2012).

The designation *disease* (first perspective, fourth domain) is conserved in the FDMI. However, we believe that parsing the other three domains as *dimension*, *behavior*, and *story*, as is done in *The Perspectives*, would not adequately differentiate or characterize the illnesses in these domains. Consider: first, whatever can be called human, whether normal or aberrant, is eventually manifested as behavior; second, all behavior is dimensional, which is to say that all behavior falls along a continuum from normal to aberrant; and third, all behavior can be parsed as a series of stories that ultimately allow us to see the shape of our lives. In the FDMI, what constitutes the *essence* of the dimension, behavior, and story perspectives for each of these domains is recast as brief *descriptions* of three different, though sometimes interdependent, *types* of defensive psychobiological *reaction*—Meyer's term, signifying both the active participation of the person undergoing the reaction and the pathological change incurred.

14.4.1 The First Domain

Meyerian reactions of the first domain originate in maladaptive reactions to life's challenges, stresses, losses, and failures.

The most common illnesses deriving from these reactions are the multiple clinical expressions of anxiety; panic (the body's manifestation of severe anxiety); demoralization and depression; pathological anger; dissociation; psychobiogenic psychosis (delusions, hallucinations, paranoia); and severe obsession–compulsion, which exceeds anything that could follow from a second-domain obsessive–compulsive style, and can include psychosis.

While all mental illnesses have biological correlates, brain pathology is not the primary cause of first domain illnesses, though it is generally agreed that some people are more psychobiologically vulnerable to developing these illnesses. (Biological correlates of many mental illnesses have been identified. Reduced brain glucose metabolism revealed in PET scans of depressed persons is widely, and wrongly, cited as proof that depression is a brain disease.)

14.4.2 The Second Domain

Second domain reactions come about in the context of aberrant personality development and temperament. Biological factors contribute, but are not primary causes.

Not everyone receives the minimum parental and social affirmation required to develop a degree of selfhood necessary to withstand the challenges of life and succeed as a person. What we are calling *pathological personality styles* (paranoid, borderline, narcissistic, histrionic, schizoid, antisocial, and obsessive–compulsive) are thought to take root in the

developmental—really, psychobiological—deficit incurred in these situations. Many people with pathological personality styles have trouble controlling their expansive and destructive impulses, leading them to have highly conflicted and painful lives, while often causing hardship for those they interact with (Shapiro 1965). Those with pathology in the second domain are more likely to succumb to defensive reactions of the first and third domains because of the brittleness of self associated with these deficits. Psychosomatic illness also falls in the second domain.

14.4.3 The Third Domain

Third domain reactions are willed, self-gratifying, and ultimately self-destructive acts that exceed the limit of what most people consider safe, sensible, and authentic behavior.

These reactions include alcohol abuse; drug abuse; pathological gambling; sexual paraphilia; anorexia; bulimia; kleptomania; pyromania; trichotillomania; and, paradoxically, self-injury (cutting, burning). In most third domain reactions, people deceive themselves as they deny the price they and others are paying for their actions. Many third domain behaviors damage the body biologically, and this fact is often used to buttress the false argument that these reactions, especially alcohol and drug dependence, and eating disorders, are autonomous brain diseases.

14.4.4 The Fourth Domain

Citing Meyer's distinction, mental disturbances of the fourth domain are those that "impinge" autonomously on a life as if from the outside rather than "emerge" dynamically from life choices as happens with reactions of the first three domains.

This kind of alteration in mental state can be due to a generally correctable physiological change known as delirium—a clouding of consciousness that accompanies metabolic and electrolyte imbalances, drug toxicity, and some medical conditions (Slavney 1998).

Autism is a condition of arrested psychobiological development, making this disorder a candidate for the second domain. But deviant biology seems to be more of a factor here than in other second domain illnesses, impinging on rather than emerging from the lives of its young victims, to cite Meyer's distinction. Until there is evidence to the contrary, autism is probably best assigned to the fourth domain—though when doing clinical work with patients, what we have learned about second domain pathology should influence our approach (Levin 2014).

Neurodegenerative, dementing diseases such as tertiary syphilis and Alzheimer's irreversibly damage the brain's neural substrate causing numerous psychiatric symptoms, including delusions and hallucinations.

The primary psychiatric illnesses of the fourth domain, schizophrenia and manic depression, are putative brain diseases: putative because, unlike tertiary syphilis or Alzheimer's, no brain abnormality has yet been identified that explains either illness, even after 150 years of intense research. Some psychotic experiences diagnosed as schizophrenia and some disturbances of mood diagnosed as manic depression are most likely autonomous brain diseases that, in Meyer's words, biologically impinge on a person. But many of these alterations in emotion, cognition, and behavior are first domain or second domain dynamic reactions accompanied by psychobiologically altered brain substrates.

The DSM-5, which intentionally disregards the etiology and meaning of psychiatric symptoms (beyond ruling out a medical condition as the cause), does not offer clinicians sufficient guidance to make the kind of diagnostic distinctions that are allowed by the FDMI. Instead, following the medical model for diagnosis, the DSM designates sets of mostly behavioral symptoms as signifying single mental illnesses, and takes all mental illnesses, including all psychotic illnesses, to be brain diseases. The FDMI and the DSM originate in, and are defined by, two essentially incompatible paradigms.

During several decades of clinical work, I saw many patients who had been previously misdiagnosed and wrongly treated using the DSM's approach. Some patients had psychotic symptoms and were incorrectly diagnosed with schizophrenia. Especially disturbing were the overzealous attempts by clinicians to identify and treat, usually with "antipsychotic" drugs, what they called "early schizophrenia" in children and adolescents. Again, these clinicians followed the medical model: diseases are best diagnosed early and treated early. There are many reasons why young people, or, for that matter, people of any age, have delusions and hallucinations. Most do not have schizophrenia.

Just how different the FDMI is from the DSM-5 can be appreciated when we recall that schizophrenia, alcoholism, and PTSD are all considered to be brain diseases in the DSM, while in FDMI schizophrenia falls in the fourth domain (putative altered brain substrate leading to a disease), alcoholism in the third domain (willed choice of self-destructive behavior), and PTSD in the first domain (failing to meet life's challenges and recover from setbacks) (McHugh 2012). It should be obvious that patients who are diagnosed with the guidance of the FDMI would think differently about the predicament they find themselves in than if they were diagnosed with the DSM-5. And, of course, they would receive a different kind of treatment.

14.5 Eliciting the Phenomena of Mental Illnesses from the Life-Stories of Exemplary Patients

The two life-stories that follow should help to hone the distinction made in the FDMI between first and fourth domain psychotic experiences—with allowances, of course, for the ambiguity that is inevitable when phenomena of only partly understood etiology and meaning are parsed and compared. The point here is not simply to hold up these individuals as exemplars of one mental illness or another but, using these examples, to *designate the phenomenon* which constitutes that illness. To do this requires going beyond the specifics of any case, no matter how compelling, to universal essences. Though the life-stories of Mrs. K and John Nash, which led to diagnoses of a first domain paranoid reaction and fourth domain schizophrenia respectively, are just two of the pathological phenomena elucidated in the FDMI (Table 14.1), it is hoped that the richness of these stories of altered mental life will give the reader a sense of what it means to uncover the truth about someone who is mentally ill by identifying the phenomenon, or phenomena, behind the illness. This chapter is a synopsis of a text, in progress, that aims to characterize the phenomena of all the mental illnesses represented in Table 14.1.

14.5.1 Mrs. K: A First Domain Paranoid Reaction

Mrs. K, who is 95, lives alone in a ranch-style house on half an acre of land in a rural suburb. On most days during the spring, summer, and fall when the weather is good, she works outdoors in the garden. During the fall of her 95th year, she raked 40 bags of leaves. During the winter, when the snow is six inches or less, Mrs. K shovels the driveway out to the road. After heavier accumulations, she calls in someone with a plow. She never complains about having to cope with the long, cold winters.

Mrs. K pays her own bills and never overdraws her checking account. She prefers to spend most of her time alone and encourages only occasional short visits from family members. She has no friends and wants none, even though neighbors occasionally make overtures to her. She keeps up with the outside world by watching the news on cable TV. In 1986 Mrs. K's husband died suddenly from heart failure. She has never shown any sign of mourning and, in fact, appeared to be rejuvenated by her husband's death. Though Mrs. K values life in her advancing years and takes good care of herself, she has made it clear that she is not afraid to die.

Mrs. K has a good quality of life and can still do many of the things that were always important to her. Her sense of the world is largely intact. She appears thin and frail, but for a nonagenarian her health is good. Her close vision has

deteriorated because of macular degeneration, and she can no longer sew, but beyond six feet she sees well. She takes 81 mg of aspirin every other day, and receives monthly subcutaneous injections of vitamin B_{12} and folic acid. Mrs. K has had occasional chest pains since her mid-eighties, which her doctor attributes to angina. Sometime after that she was found to have atrial fibrillation. Her only prescription medications are Cardizem and Plavix.

Mrs. K has a son and a daughter, both in their sixties. The daughter and her four adult children live nearby. The son lives in a distant city. The daughter, who is divorced, does Mrs. K's grocery shopping and drives her to doctors' appointments.

Mrs. K's mental life is intact, except for one glitch: she claims to believe that her grandchildren come in the middle of the night, or when she is away during the day, to steal her possessions and that her daughter knows and approves of this. The "stolen" items include sheets, towels, pots and pans, milk, and orange juice. According to Mrs. K, her sterling silver and antiques are being sold and replaced with cheaper items by her grandchildren so they can pocket the difference. These accusations have been made time and again, over a period of many years. Mrs. K also claims that her phone is being tapped. She puts all the blame for this intrusion on her grandchildren and does not feel that either the phone company or the government is involved. According to Mrs. K, the grandchildren listen in on her phone calls because they want to know when she is going to sell her house and when they will get their inheritance.

Mrs. K alleges that her grandchildren steal from her and covet her money because things are not going well for them. Being reminded that three of the grandchildren have good jobs and that the fourth has a husband who makes a respectable living does not sway Mrs. K from this belief. She has been able to convince herself that her grandchildren need the money they steal from her to survive, and that she is their savior. Mrs. K's apparent hatred of her family, manifested in many ways over many years, seems to be transformed through this self-deception into an act of *their* betrayal. The ultimate reason for this woman's hatred is opaque, but there has always been something about her family being successful and happy that has threatened her and tweaked her envy.

Mrs. K clearly meets criteria for what the DSM-5 designates as delusional disorder, persecutory type. Though she has often directed outbursts of anger tinged with paranoia at family members, she has never shown any indication of being clinically depressed, or of having had a sustained period of low mood. No case can be made for psychotic depression or for schizophrenia. Mrs. K has never been manic or hypomanic, or ever abused alcohol or any other drug. Neither she nor any of her blood relatives have been diagnosed with or treated for a mental illness.

The FDMI places Mrs. K's pathology in the first domain, and, following Meyer, names her behavior a paranoid psychotic reaction.

14.5.2 John Nash: A Noted Mathematician Diagnosed with Schizophrenia, a Putative Fourth Domain Brain Disease

John Nash is considered to be one of the great mathematical minds of the twentieth century. A Princeton Ph.D. at age 21, he is best known for developing the mathematics of game theory that was later used to plan military and economic strategy.

During the 1950s, while he was still doing what was considered brilliant work, his thinking, feeling, and behavior became unaccountably bizarre, and he was eventually diagnosed with paranoid schizophrenia. Nash angrily resigned his positions at Princeton and M.I.T., making bitter accusations against his stunned colleagues. His wife was not spared from his paranoid rage. After standing by him for many years, she divorced him (they later remarried). He floundered badly, could not do any sustained mathematical work, or teach, and lost the ability to function socially.

Nash had paranoid and grandiose delusions and auditory hallucinations. He came to believe that extraterrestrials were sending him messages and that the course of his life was being determined by certain sequences and patterns of numbers. He showed a partial response to Thorazine and Stelazine, but refused to take antipsychotic drugs after 1970. In the mid-1980s, after struggling for three decades with a serious mental illness that required many hospitalizations, Nash mysteriously got over the worst of this illness and reclaimed a part of his life and his career. He was awarded the Nobel Prize in economics in 1994 for work he had done before he became ill.

In 1998 Sylvia Nasar published a widely praised biography of Nash, *A Beautiful Mind* (Nasar 1998). She carefully parsed every phase of Nash's life, showcasing his brilliance, nobility, and tragedy. It is clear from Nasar's book that, during the prodromal phase of his illness, Nash was anxious and overwhelmed by the pressures of work and marriage, as well as by the conflict he seems to have felt about his homoerotic attachments.

Nash had always been considered aloof and eccentric. These personality traits are not unusual in persons who breathe the rarefied air of mathematics and theoretical physics where the abstract is prized over the concrete and the hard edges of everyday reality can be avoided as a matter of course. Leonard Mlodinow, a physicist, acknowledged that "one of the advantages of theoretical physics is that you can wander in different realities, and yet you're not considered mentally ill" (Mlodinow 2012). Under pressure, Nash may have found

it relatively easy to blur the boundary between a world that was becoming increasingly difficult for him and a world of fantasy that was more palatable.

In the spring of 2002, the Public Broadcasting Service (PBS) aired a TV program on Nash titled *A Brilliant Madness*. Nash provided some insightful sound bytes, including this one: "To some extent sanity is a form of conformity. People are always selling the idea that people with mental illness are suffering. I think madness can be an escape. If things are not so good, you maybe want to imagine something better. In madness, I thought I was the most important person in the world" (Kennedy 2002). Nash seems to be saying that he rejected the rational world after it became too painful for him to live there. The delusional ideas he developed seemed real, he said, because they came to him in the same way that his mathematical ideas came to him.

Nash claims that he partially willed his illness into being and then willed his recovery as well. Of the voices that directed his life while he was seriously ill he said, "I began rejecting them and deciding not to listen" (James 2002). His language is direct and strong here, and his words are those of someone who feels that he is in control—*he* rejected, *he* decided. In 1996 Nash recalled, "I emerged from irrational thinking ultimately, without medicine other than the natural hormonal changes of aging" (Nasar 1998: 353). To hear John Nash tell it, what the existentialists call the self as active agent appears to have been calling the shots all along.

What may ultimately distinguish the phenomenon of a first domain psychotic reaction from the phenomenon of schizophrenia, a putative brain disease of the fourth domain, is the degree of psychobiological disintegration—the "hit" taken by the organism—that occurs when a constitutionally vulnerable person is psychically traumatized to the point of opting out of reality, to follow one escape route or another to an "inner" world. Meyer believed that a dynamically driven psychotic experience can alter brain structure and function. "Mind," he said, "like every other function, can demoralize and undermine itself and its organ," the brain (Meyer 1906). Seen in this way, psychoses of the first and fourth domains are psychobiological phenomena of different pathological power within a continuum of psychotic experience.

Mrs. K limited her paranoid psychotic reaction to her family. Whatever the origin and nature of the anxiety that spawned her pathological thinking and behavior, it did not require her to be any crazier than that. In his schizophrenic reaction, as Meyer would have called it, John Nash needed to psychotically transform his entire world and everyone in it. At its peak, his psychic terror, an emotion that was an order of magnitude more intense and crippling than any anxiety Mrs. K may have felt, demanded an escape from reality itself. If we believe Meyer, Nash's greater psychological transformation caused a greater

correlative biological upset as well, putting his illness well into the fourth domain, and leaving us to wonder just what kind of reversible "brain disease" Nash actually had!

Which is why, when speaking of schizophrenia, we say "putative brain disease" to distinguish this illness from fourth domain neurological diseases such as tertiary syphilis and Alzheimer's that also produce psychotic symptoms, but have known organic causes. Again, following Meyer, we can picture a schizophrenic reaction epigenetically altering a brain's structure and function, sometimes in a way that is reversible, as Nash's illness was at least partly reversible. Schizophrenia has long been recognized as a heterogeneous illness. There are undoubtedly other schizophrenias that are more biological than Nash's, more genetic, more disease-like.

14.6 The DSM-5 Ignores Freedom and Responsibility in Assessing the Contents of Mental Life

Our ultimate goal as clinicians is to get our patients right, and persuade them to exchange a life that does not work well for them for one that works better (Frank and Frank 1991). To do that, we must grasp the essence of what it means to be a person, in health and in sickness. The DSM-III, DSM-IV, and DSM-5 ultimately fail because these systems endorse a way of diagnosing mental illness that denies the radical essence of every person's life: *freedom*.

To deny freedom is to deny responsibility. Grounded in these denials, the DSM is a veritable invitation for clinicians to get a diagnosis wrong. Purposely ignoring etiology, and taking symptoms at face value, the DSM makes no provision for holding patients (or anyone else) accountable for a mental illness they develop.

What most differentiates the FDMI and the DSM is the notion that, to a considerable degree, the person we become emerges from the sum of the choices we make in the process of exercising our freedom. Originating in what is surely one of the great philosophical insights of the twentieth century, Jean-Paul Sartre declared that "we are condemned to be free" (Sartre 2007: 29). We have no choice in the matter, only in how we *use or misuse* our freedom. Being condemned to freedom is the ur-paradox of human existence!

Even while we are denying our freedom, which we all do at times, especially under pressure and when things go badly, we are using that freedom to construct this denial: refusing to choose *is* a choice. In the Meyerian and existential formulations of psychopathology, most mental illnesses are thought to originate in a person's self-deceiving misuse of freedom. In the FDMI, freedom and

its misuse play out across all four domains, though in different ways in each domain, and to different degrees.

While acknowledging the limits of what we know about how and why people become mentally ill, the FDMI offers clinicians an approach to parsing the varieties of mental illness that affords both freedom and biology their due, so that patients can receive *their* due. To do justice to the growing number of patients who are seeking our help, the mental health profession must create a valid paradigm based on a valid anthropology—the notion of what it means to be a human being—and jettison the current paradigm that falsely informs the DSM-5 (Muller 2013).

14.7 Knowing What is True and Valid When Diagnosing the Altered States of Mental Life

Truth stipulates that an entity is represented as it is before preconceptions and bias transform it into something else. Ultimately, we need to think about the world in such a way that our own "bias"—no one is without a point of view—tends toward a perspective that can be considered valid because it does not distort what is there.

William James (1842–1910) was an American philosopher and psychologist whose New-World existential thinking—known as pragmatism—significantly influenced Meyer, and united him in spirit with the European existentialists. James and Meyer shared the existentialists' view that the truth about others could be intuitively known, a notion that the logical positivists vigorously deny. This denial is a major component of the anthropology that underlies the DSM-III, DSM-IV, and DSM-5, and allows the etiology and meaning of symptoms to be ignored. To James, Meyer, and the existentialists, such a view of what a person is would preclude a valid understanding of the person's behavior, normal or otherwise.

In his superb rendering of James's life and work, *A Stroll with William James*, Jacques Barzun begins the discussion of pragmatism by citing the need for what he calls a "test of truth." How, he asks, can we know anything substantial about a life that is flux at its core?

> If Experience as natively given in consciousness is fleeting, variable, helter-skelter, and if we have the power to work upon it, by attention, by making concepts, by combining images, then we need a way of making sure that the account we give of any experience, the bearing of any connections we discover, is solid, reliable, permanent because [it is] rooted in the nature of things (Barzun 1983: 83).

A stick is placed in a transparent container filled with water, one long enough to extend several inches above the surface of the water. Barzun asks us to consider the conundrum of what is observed. At the water line,

> we see certain lines and colors that makes us think [the stick is] broken—we know from past experience that that is how a broken stick looks. But if we have any doubt, we slide our hand along the stick in the water and feel no break: the idea "broken stick" is not true; it disagrees with a subsequent, relevant experience. The pragmatic test is repeated when we pull the stick out of the water, see it whole and lean on it—*not* broken (Barzun 1983: 86).

Ever the concrete thinker, James locates truth close to the ground: "Truth is simply a collective name for verification processes, just as health, wealth, strength, etc. are names for other processes connected with life. Truth is *made*, just as health, wealth, and strength are made, in the course of experience" (James 1907: 218).

Pragmatically speaking, truth is what works, and what makes sense in the context of everything we know about the world. Clearly, James's "truth" aphorism does not cover all we encounter. James, like Meyer, realized that theories are not absolute transcripts of reality. "Their great use is to summarize old facts and to lead to new ones. They are only a man-made language, a conceptual shorthand, in which we write our reports of nature" (James 1907: 212–13, 57).

Let us apply James's concept of truth to the claims made by the creators of the third, fourth, and fifth editions of the DSM that the mostly behavioral criteria in these texts can be used to diagnose mental illnesses—in spite of their acknowledgment that what was done to increase reliability compromised validity in the process (Bernstein 2011). This trade-off ignores the fact that even if reliability were improved—but how could it be with all the diagnostic "noise" generated by ignoring the etiology and meaning of symptoms?—the sacrifice of validity would render this "reliability" worthless. The DSM-5 fails the pragmatists' test of validity: it does not work. It leads, ineluctably, to many wrong diagnoses. It cannot be fixed because it is not broken—it is just *wrong*. As a result, countless patients have been compromised, and the results of a good deal of psychiatric research based on diagnoses made using the DSM's last three editions fall wide of the mark.

Every beginning course in natural science belabors the distinction between validity and reliability, and the importance of this distinction in doing research and interpreting data. But the creators and users of the third, fourth, and fifth editions of the DSM appear to have forgotten their early science training when they created the implicit subtext that as long as clinicians agree, to some extent anyway, on a wrong diagnosis, their agreement trumps the need to get a correct diagnosis. It is hard to imagine what standard of truth and validity is met here. Had Adolf Meyer lived to see the DSM-5, he undoubtedly would have felt that this approach to classifying and diagnosing mental illness lacked "common-sense" (Lief 1948)—the signature of his psychobiological approach—and that the DSM-5 was invalid.

Table 14.1 The Four Domains of Mental Illness

1st Domain	2nd Domain	3rd Domain	4th Domain*
Pathological Reactions of the First Domain Originating in Failures to Meet Life's Challenges and Recover from Setbacks. Biological Factors Contribute, but Are Not Primary Causes.	*Second Domain Reactions Come About in the Context of Aberrant Personality Development and Temperament. Biological Factors Contribute, but Are Not Primary Causes.*	*Third Domain Reactions Are Willed, Self-gratifying and Ultimately Self-destructive Acts that Exceed the Limit of What Most People Consider Safe, Sensible, and Authentic Behavior. Biological Factors Contribute, but Are Not Primary Causes.*	*Altered Mental States of the Fourth Domain Due to Physiological Imbalance, Medical Disease, and Primary Mental Illness Associated with a Putative Altered Brain Substrate.*
The Anxiety Spectrum Anxiety Reaction Pervasive Anxiety Reaction Separation Anxiety Reaction Pathological Stress Reaction Agoraphobic Reaction Phobic Reactions Panic Reaction/Panic Attack/ Panic Disorder Post-Traumatic Stress Reaction Obsessive-Compulsive Reaction **The Depression Spectrum** Depressive Reaction Adjustment Reaction Grief Reaction Dysthymic Reaction (Pathological Anger Reaction) **The Dissociative Spectrum** Dissociative Reaction Depersonalization Reaction	**Pathological Personality Styles** Paranoid Personality Style Borderline Personality Style Narcissistic Personality Style Histrionic Personality Style Schizoid Personality Style Antisocial Personality Style Obsessive–Compulsive Style **Psychosomatic Illness** **ADHD: A Non-phenomenon**	**Self-Destructive Choices** Alcohol abuse Drug abuse Pathological gambling Sexual paraphilia Anorexia Bulimia Kleptomania Pyromania Trichotillomania Self-injury (cutting, burning)	**Delirium** *Metabolic Causes* Hypoglycemia and hyperglycemia Hyperthyroidism and hypothyroidism Vitamin deficiencies (D, B_{12}, folate, nicotinic acid, thiamine) Excess of vitamins A and D Addison's disease and Cushing's syndrome *Electrolyte Causes* Dehydration Hyponatremic water intoxication Acidosis Alkalosis Deficiency or excess of ionic calcium, potassium, sodium and magnesium. *Toxological Causes* CO poisoning Lead and other heavy metal poisoning Organic solvents Insecticides Inhalants (ether, gasoline, glue) Plant and fungal poisons

Dissociative Amnesia Reaction
Dissociative Fugue Reaction
Dissociative Identity Reaction
The Psychosis Spectrum
Depressive Psychotic Reaction
Paranoid Psychotic Reaction
Other Dynamic Psychotic Reactions
Brief Psychotic Reaction

Medical Causes
Infectious disease
Neoplastic disease
Paraneoplastic syndrome
Intracranial space-occupying lesions
Head trauma
Traumatic brain injury (TBI)
Epilepsy
Vascular and blood disorders
Post-surgical complications
Vascular stroke
Heat stroke
Prescribed and prohibited drugs
Withdrawal from these drugs
Arrested Psychobiological Development of the Brain
Autism
Neurodegenerative Brain Diseases with Psychiatric Symptoms
Alzheimer's and other dementing diseases—Pick's, Huntington's, and Parkinson's
Tertiary syphilis
Tardive dyskinesia
Physiologically Induced Mood Disorders
Anxiety
Depression
Primary Mental Illnesses Emerging from a Putative Altered Brain Substrate
Some Phenotypes of Schizophrenia
Some Phenotypes of Manic-depression

* The entries for delirium and neurodegenerative diseases in the fourth domain were adapted from Slavney, P. R. (1998). *Psychiatric Dimensions of Medical Practice*. Baltimore: The Johns Hopkins University Press.

References

Barzun, J. (1983). *A Stroll with William James*. New York: Harper & Row.

Belluck, P., and Carey, B. (2013). Psychiatry's new guide falls short, experts say. *The New York Times*, May 7, A13–15.

Bernstein, C. A. (2011). Meta-structure in DSM-5 process. *Psychiatric News*, March 4, 7–29.

Chisolm, M. S., and Lyketsos, C. G. (2012). *Systematic Psychiatric Evaluation: A Step-by-Step Guide to Applying "The Perspectives of Psychiatry"*. Baltimore: The Johns Hopkins University Press.

Eisenberg, L. (1995). The social construction of the human brain. *American Journal of Psychiatry*, **152**, 1563–75.

Frank, J. D., and Frank, J. B. (1991). *Persuasion and Healing: A Comparative Study of Healing*, Third edition. Baltimore: The Johns Hopkins University Press.

Hamilton, J. (2013). Why is psychiatry's new manual so much like the old one? Available at: <http://www.npr.org/blogs/health/2013/05/16/184454931> [accessed February 7, 2014].

James, C. (2002). The man, not a legend, of "A Beautiful Mind" [Article on PBS's American Experience: "A Brilliant Madness"]. *The New York Times*, April 26, 2002, B28.

James, W. (1907). *Pragmatism: A New Name for Some Old Ways of Thinking*. New York: Longmans.

James, W. (1920). *Collected Essays and Reviews*. New York: Longmans.

Kennedy, D. (2002). The mind inside the madness [Article on PBS's American Experience: "A Brilliant Madness"]. *The New York Times*, April 21, 2002, AR (Sect 2.), 35.

Levin, A. (2014). When autism entered the lexicon. *Psychiatric News*, May 16, 2014, **7**, 33.

Lief, A. (ed.) (1948). *The Commonsense Psychiatry of Dr. Adolf Meyer: Fifty-Two Selected Papers*. New York: McGraw-Hill.

McHugh, P. R. (2012). Rendering mental disorders intelligible: Addressing psychiatry's urgent challenge. In K. S. Kendler and J. Parnas (eds), *Philosophical Issues in Psychiatry II: Nosology*. New York: Oxford University Press, 269–79.

McHugh, P. R., and Slavney, P. R. (1998). *The Perspectives of Psychiatry*, Second edition. Baltimore: The Johns Hopkins University Press.

Meyer, A. (1906). Fundamental conceptions of dementia praecox. *British Medical Journal*, **2**, 757–9.

Mlodinow, L. (2012). Physicist: I studied theories of infinite dimensions—in the university and in Hollywood. *The New York Times*, June 3, 2012, SR7.

Muller, R. J. (2003). *Psych ER: Psychiatric Patients Come to the Emergency Room*. New York: The Analytic Press/Taylor & Francis Group.

Muller, R. J. (2008). *Doing Psychiatry Wrong: A Critical and Prescriptive Look at a Faltering Profession*. New York: The Analytic Press/Taylor & Francis Group.

Muller, R. J. (2013). Biological psychiatry's false paradigm. Baltimore *Sun*, June 19, 2013, 19.

Nasar, S. (1998). *A Beautiful Mind: The Life of Mathematical Genius and Nobel Laureate John Nash*. New York: Simon & Schuster.

Sartre, J.-P. (2007). *Existentialism Is a Humanism*, tr. C. Macomber. New Haven: Yale University Press.

Shapiro, D. (1965). *Neurotic Styles*. New York: Basic Books.

Slavney, P. R. (1998). *Psychiatric Dimensions of Medical Practice*. Baltimore: The Johns Hopkins University Press.

Part 4

Epilogue

Chapter 15

United in diversity: Are there convergent models of psychiatric validity?

Drozdstoj St. Stoyanov and
Massimiliano Aragona

15.1 The Divergent Convergent Method for Problem Solving

One of the most significant contributions of the twentieth-century philosopher of science Azarya Polikarov (1974) was his *Divergent–Convergent Method* (DCM). Published in the Boston Studies in the Philosophy of Science, it was presented as a "heuristic approach to problem-solving" (p. 211). According to Polikarov, scientific problems can be classified into problems of (i) existence, (ii) explanation, and (iii) elimination of contradiction (Entscheidbarkeit). In his scheme, *classification and validity* belong to the problem of explanation, which includes under its auspices the activities of construction and substantiation.

According to Polikarov, scientific problems are explored in two stages. In the first stage, an initial *field of possible or design-solutions* is formulated, some of them only hypothetical, others better supported with available evidence. Very often those initial design-solutions are contradictory and mutually exclusive. This divergent stage is developed further as new variant-solutions interact. In the case of psychiatric nosology the initial field is composed of such classical approaches as the categorical model adopted from medicine and the dimensional model adopted from psychology. The new variant solutions which have emerged over the past decades include the prototype, cluster, structural, RDoC, and narrative/person-centered models.

Additionally, Polikarov describes three variations of design-solutions in the divergent stage: radical, moderate, and combined. By radical he means solutions which take an opposite and incommensurable stand with respect to prevailing models. By moderate he means solutions with an intermediate position between the radical and combined. Most modern scientific problems are

usually penetrated at the level of combined design-solutions. The latter are subdivided into three kinds:

(i) alternative—without constraint, including radically alternative;

(ii) with weak constraint of diversity, i.e., produced by removal of some variants;

(iii) with strong constraint of diversity, which can entail formulation of unique combinations of solutions.

In Polikarov's second stage the field is "reduced" (or converged) to a more restricted area of *predominant group of project-solutions*. This convergence is performed via logical and content considerations, emerging empirical data, and methodological principles. Those principles include: representativeness, simplicity, originality, explanatory and predictive power, coherence, and congruity. The reduction of the field of possible design-solutions eventually results in a dichotomous situation:

(i) there either exists one true solution and all others are false; or

(ii) the true decision is the combined (synthetic) one.

In the case of psychiatric validity, two contrasting possible design-solutions are the *realist* model and the *pluralist–perspectival* model. In the realist model, the concepts of validity are unified under one single biologically sound concept and others eliminated as irrelevant. In the pluralist model the concepts of validity can be seen as partially true and context dependent.

Historically, diagnostic systems have pursued a realist concept of validity, i.e., to validate a mental disorder is to show that it is a real entity in nature, and this is best done when we are able to validate it with external criteria such as neurobiological dysfunctions. Those proponents of biological psychiatry that had adopted a reductive approach to the mind–brain relationship expected that technological progress in neuroscience would have offered ultimate explanations and therefore be robust validators for mental disorders. This expectation unfortunately has not been fulfilled. Despite the many encouraging advances in neuroscience, neurobiological markers are still far from being discovered for mental disorders.

The result has led to a crisis of confidence regarding the validity of psychiatric classification. With this crisis in mind, the fundamental queries raised in conceptualizing our monograph were:

1. Is a "Big V" (single, unified) form of validity the only option for psychiatry?
2. Are there alternative models of validity?
3. Does it make sense to talk about validity at all?

As is evident in the preceding chapters, an exciting field of divergent solutions to the problem of psychiatric validity has emerged.

15.2 A Divergent Field of Possible Solutions to the Problem of Psychiatric Validity

According to Loughlin and Miles, questions about the scientific validity of psychiatric diagnosis derive their meaning from specific conceptions of science, value, and reality. In their chapter they contest the dilemma created by the anti-psychiatry movement in which mental disorders are either real diseases or moral entities based on subjective values. They claim that such a rigid dichotomy is the result of a "scientistic" ideal of objectivity, arguing that the assertion that mental disorders are not real diseases does not lead to moral subjectivism if the value-ladenness of psychiatric diagnosis is based upon value-realism. According to them, validity is not a matter of objective scientific evidence, but of ethical judgment about what is (and should be) the human good.

Other authors focus on validity within science while also examining foundational distinctions. For example, making a strong ontological claim, Sabbarton-Leary et al. assert that not all syndromes listed in the DSM-5 are natural kinds. Only those entities that have a biological cause deserve to be considered "mental disorders," and only to them does the traditional debate on validity apply. But there are also "mental harms," and they too are of interest to psychiatry. Mental harms are defined ontologically as "para-natural kinds," which are states harmful to the agent, without a clear and distinct biological etiology. In such cases validity cannot be a matter of discovering an underlying biological etiology, and other factors such as intensity, duration, or distress may help to determine their clinical relevance.

Thus, while Loughlin and Miles tend to translate the debate about validity into the field of ethics, Sabbarton-Leary et al. accept the traditional realist account for their "mental disorders" *strictu sensu*, but deny its application to their "mental harms."

Marková and Berrios examine a more specific question: whether neuroimaging techniques are adequate tools for validating mental disorders. According to their analysis, there is a basic ontological question which is prior to any consideration of validity, namely, what kind of objects are those *mental symptoms* that we should investigate by means of neuroimaging tools? They argue that mental symptoms are hybrid objects with a physical kernel (the neurobiological signal) enveloped by semantic wrappers (personal, familial, and sociocultural meanings that shape the original experience). Those symptoms where the neurobiological part is prevalent are more apt for neuroimaging exploration, while in those cases in which the semantic construction is more important, imaging will be an inadequate validator.

One question that arises from these reflections is: if validity is a matter of finding a correlation between the index mental phenomena and the neurobiological substratum, is current neuroscience able to validate disorders? This is not an easy question to address. The limitations that serve as barriers to answering this

question include limitations in the techniques (e.g. insufficient spatial and/or temporal resolution), in the phenomena to be explored (the discussed ontological differences between mental disorders and between mental symptoms), in the proxy variables reporting such phenomena (e.g., is a score in a rating scale a good substitute for the index phenomenon?), and also in the timing in the assessment procedures.

Stoyanov et al. directly address the last two limitations. They stress that the outputs of rating scales and standardized interviews represent decontextualized excerpts drawn from patients' narratives, which can hardly correlate with neuroscientific examinations that are performed at different times and within a different domain of knowledge. They show that the findings from psychiatric tools tend to be validated within their own disciplinary field and the findings from neurobiological tools tend to be validated within their own disciplinary field. Stoyanov and colleagues propose to manage such an explanatory gap by implementing a program of cross-disciplinary validation. As an example, mental phenomena should be cross-validated through measurement of the brain activation detected by fMRI and simultaneous administration of state-dependent clinical measures.

The relationship between validity and measurement practices in psychological assessment and psychiatric diagnosis is insightfully explored by Keeley. Validity in psychiatry reflects both the validity of the diagnostic construct (the category, or the dimensional profile), and the validity of the diagnostic process (i.e., the procedure we use to arrive at that diagnostic construct). Keeley argues that the assumptions underlying the measurement process have a role in shaping the structure of the resulting diagnostic entity. In other words, the way measures are constructed, as well as the way they are practically used, both influence the resulting diagnostic entity. For example, if a clinician believes that psychiatric objects are categorical, he/she will adopt assessment instruments (such as yes–no checklists) which are more likely to provide categorical diagnoses. In contrast, a clinician who believes that psychiatric symptoms vary continuously in the population without a clear distinction between normality and pathology will probably use inventories that provide dimensional scores. One important conclusion that can be drawn is that there is no single way to estimate the validity of psychiatric diagnosis. Diagnostic validity is pluralistic and impossible to disentangle from the measurements and pragmatic context in which it is used.

Similar pragmatic considerations are expressed by Aragona, who conceives mental disorders as more or less useful concepts for practical needs, constructed in specific places and times to meet practical needs, and in need of recalibration depending on socio-cultural circumstances and scientific priorities. In this context validation is no longer absolute but relative to the diagnostic system(s) in which validity questions make sense.

In their chapter, Rodrigues and Banzato focus on the pragmatic concept of utility. They distinguish between diagnostic validity and nosologic validity. Diagnostic validity refers to how diagnostic criteria lead to the proper identification of clinical instances of a psychiatric construct. Here they place the notions of face, content, criterion-oriented, and construct validity. Nosologic validity refers to the justification for including a diagnostic category in a system of classification. They distinguish between two different conceptions of nosologic validity. According to the realist conception a diagnostic category is valid if it represents a real entity. According to the pragmatic conception a diagnostic category is valid if it is useful. They stress that reality and utility cannot be reduced to one or the other, and thus a "dual-track" program of validation is needed.

Pragmatic accounts of validity are further addressed by Murphy, who critically discusses Schaffner's pragmatism. Schaffner opposes making a firm distinction between realist validity and utility on the grounds that utility is constitutive of reality (thus, a position which is opposite to the "dual-tack" validation proposed by Rodrigues and Banzato). Against Schaffner, Murphy argues that a disorder's dependence upon being recognized under some concept does not mean that what is perceived via concepts does not exist. Despite this conceptual influence, he argues that we have a "relatively direct access to the world" which is sufficient for a realist account, provided that the realist accepts auxiliary hypotheses and can discard plausible alternatives.

Murphy's realism holds that psychiatric constructs are related to the causal structure of the world. Drawing on Cummins' concept of dysfunction, which he calls "the systemic view," Murphy argues that although mental disorders can be the effects of biological dysfunctions, the concept of dysfunction itself is not a mere matter of fact. Rather, dysfunctions are intrinsically normative concepts. We judge something to be dysfunctional because it does not conform to our ideas about how a good-functioning system should be. Like weeds, mental disorders are something that we don't like because of certain interests we have. We know scientific facts about them, but there is nothing intrinsically disordered independently from our negative evaluations. Normative judgment comes first, and normative issues being open, we cannot validate a diagnosis; we can just correlate it with part of the world's structure.

Other interesting remarks about the intrinsic limitations of validity intended as the discovering of the neurobiological etiology are offered by James Phillips. After distinguishing the validity of the diagnostic constructs from the validity of the diagnostic criteria and the validity of the external measures used to confirm a diagnosis, Phillips considers three different kinds of diagnostic validity: "Strong Syndromal Validity," "Weak Syndromal Validity," and the validity of the RDoC project. According to Phillips, all three kinds of validity have the same limitation: they depict psychiatric disorders as clocks whose parts can

be studied independently and mechanically disassembled. The only difference would be that the Strong and the Weak Syndromal Validities focus on syndromes, while the RDoC project focuses on symptoms.

Against this common mechanistic view, Phillips argues that mental disorders are complex systems, meaning that the actual function of their parts depends upon the total organization of the system. Complex, interactive etiology can produce heterogeneous presentations and high levels of comorbidity, which influences our efforts to classify mental disorders. Additionally, Phillips introduces another important theme, i.e., the role of psychological factors. While the debate on validity is usually shaped in neurobiological terms, Phillips argues that a complete biomedical model would take the complexity of psychiatric disorders into account, and in doing so have to include psychological domains among its validators.

The theme of psychological and psychosocial factors leads us to a related issue, that of "personal" features. In their chapter, Mezzich and Salloum propose a person-centered integrative diagnosis as an alternative to the reductionism which is implicit in the concept of diagnostic validity. They argue that good diagnosis must go beyond the nosological diagnosis (i.e., identifying a diagnosis as a general entity pertaining to several patients) to also involve understanding the person who is presenting for care. They propose an integrative model in which the nosographical level (the categorical attribution of a formal ICD diagnosis) is only one part of a larger assessment procedure including whole health (both ill and positive health), dimensional profiles, and narrative accounts of the patient's values and of cultural experiences of both illness and health.

That personal and transnosographic factors have to be considered, beyond the categorical diagnosis, is exemplified by two chapters that utilize personality as exemplar mental disorders. Jacobs and Krueger contrast the current top-down approach to psychiatric diagnosis to what they call "structural validity." The top-down approach is criticized because it starts from expert opinions instead of starting from scientific evidence. According to them, the top-down approach, focusing on reliability and external validity, has led to artificial distinctions between disorders that are actually empirically linked. This has resulted in mixed cases, overlapping boundaries, and high comorbidity. Moreover, they argued that external validity is too broad and unspecific to be the sole source of validation of a diagnostic system. The structural validity alternative is a bottom-up data-driven procedure. In this approach the categorization of mental disorders would reflect the way that disorders are organized in nature. For example, the DSM-IV personality disorders have been discredited because their comorbidity is excessively high, thus undermining the categorical assumption that they are distinct disorders. In a structural validity approach the focus would be on a "trait-based" system because such a system is better able to comprehensively model the symptom space and to correspond to the patterns that exist in patient populations.

Another personality-oriented alternative is presented by Cloninger, who criticizes both the reification of current psychiatric categories (considered by many as discrete disease-entities) and those approaches that start from personality traits considered as separate units that can be added together. In his psychobiological approach, which shows similarities with the Person-centered Integrative Diagnosis described by Mezzich and Salloum, Cloninger proposes to integrate category-like prototypes, dimensions, configural profiles, and person-centered narrative accounts. In doing so, he considers the person's self-organizing goals and values and their role in health promotion, as well as in the vulnerability to personality disorders and psychopathology. In this context, validation is not a purely logical or empirical process. Rather, it requires an integration of intuition, reasoning, and observations to test, correct, and refine the initial insights. From this perspective, validation is an open process building on insights coming from different disciplines (biological, psychological, social, cultural, philosophical, and spiritual).

Finally, Muller criticizes the operationalized, neo-Kraepelinian DSMs for being not valid because they ignore what is essential in psychiatric diagnosis: namely, that a valid paradigm should be based on a valid anthropology, acknowledging the role of freedom, responsibility, and self-deception in the genesis of mental illness. Having renounced the search for the essence of mental phenomena in favor of an agreement on superficial descriptions, the DSM, he asserts, has failed. Muller's alternative model, called the "Four Domains of Mental Illness" (FDMI), is based on recent perspectivist theories of psychiatric illness and is deeply influenced by Meyer's psychobiology, existentialism, and pragmatism. In this model truth is made in the course of experience, and the validity of a psychiatric classification largely depends on its ability to provide an understanding of the person's experience and behavior that both makes sense and helps psychiatrists achieve their professional goals.

15.3 A Convergent Field of Predominant Project Solutions

The extensive field of the divergent perspectives on psychiatric validity is produced by interactions with novel variant solutions (e.g., structural validation versus person-centered diagnosis). Divergent design solutions without any constraint of diversity have practically led to the *status quo* of psychiatric validation. The clearly understood concept of reliability has been emphasized instead of validity, in part, because of the incommensurability of the alternative validity concepts. Polikarov's strong constraint of diversity in design-solutions and eventually one single realist model are still far out of reach and most probably impossible. Therefore, the most likely scenario would be to limit psychiatric validity to a weak constraint of diversity in the divergent stage of problem solving.

Further, a reduced field of predominant project-solutions to the problem of validity in mental health should be pluralistic, therefore a combined (synthetic) solution in terms of the divergent convergent approach. This combined project should be based on the following considerations, drawing from the following agreements shared by two or more contributors:

1. Current classification systems such as the DSM and ICD seem to be unsuccessful attempts to establish validity by convention (Aragona, Cloninger, Jacobs, and Krueger). Current classification systems were unsuccessful because they define mental disorders on the basis of superficial symptoms without considering their role in the person's mental structure and their meaning for the person. This led to heterogeneous categories that are not easy to correlate to external validators, with too many interfering variables contemporarily at work.
2. The categorical and semi-dimensional (criterion) approaches to psychiatric nosology have neither scientific validity nor clinical utility since they miss important facets of knowledge about mental disorder (Sabbarton-Leary et al.; Rodriguez and Banzato; Phillips).
3. The person of the patient-in-context should remain the focus of clinical interest, instead of any form of presumed formal criteria for diagnosis (Cloninger et al.; Mezzich and Salloum).
4. Broader, "high umbrella" approaches to validation should be employed in the first place, such as prototype, dimensional, and person-centered (Mezzich and Salloum; Jacobs and Krueger; Cloninger).
5. Diagnostic entities should be underpinned with sound evidence from neuroscience. However, the current status of the connections between neuroscience and psychiatry is insufficient due to essential methodological gaps (Markova and Berrios; Stoyanov et al.). Therefore, the framework of joint neuroscience–psychiatry scientific inquiry should be revisited to provide stronger inter-disciplinary research findings.

In conclusion, validity is a manifold concept. The different options presented in this book can be arranged along a dimensional logical space having two extremes. One is a radical pluralistic approach where every perspective stipulates a unique concept of validity that is to some extent incommensurable with other concepts of validity. In this relativistic case, there is no common ground to decide between alternative validation procedures. Any concept of validity would be fine in its own domain, but any domain could learn nothing from the debates taking place in other disciplinary fields.

The other extreme is a radical unifying approach aiming at finding *the only right* concept of validity, which should be used by everyone. Although this

approach does not need to be a realist one (the unique acceptable definition might also be ethical, or logical, etc.), it is always reductionist, i.e., alternative concepts have to converge on the correct concept. There are three problems with this approach. The first problem is about the preferred level of analysis: should we base the concept of validity on neurobiology and at what level of neurobiology? The second problem is about feasibility: are we ready to decide what concept of validity is the right one? The third problem is desirability: are we sure that we would not lose important phenomena by converging all levels into one preferred level?

Several possibilities occupy the middle ground between these extremes. These include what we might call "moderate perspectivism" and "moderate convergentism." According to moderate perspectivism, every domain of knowledge has its own methodologies and core assumptions, and thus its own concept of validity, but nevertheless concepts can be contrasted across domains. As a result, different disciplines can interact and reciprocally influence each other through dialogue. According to moderate convergentism, although a unique validity concept is probably unattainable, nevertheless the plethora of validity proposals might be reduced, focusing on shared similarities and cross-validation procedures.

One contribution of this book is that it provides resources for (i) those seeking a unified realistic model, as well as (ii) those that want to maintain a pluralistic approach to knowledge domains that they consider separate but interacting. Hopefully we have managed to provide a cognitive framework that may inform future efforts toward the reappraisal of validity and validation standards in the mental health disciplines.

References

Polikarov, A. (1974). The Divergent–Convergent Method. In R. S. Cohen and M. Wartofsky (eds), *Methodological and Historical Essays in the Natural and Social Sciences*, Boston Studies in the Philosophy of Science, Vol. **XIV**, Dordrecht, 213–33.

Stoyanov, D. S. (2010). On the epistemological legacy of A. Polikarov. *Epistemologia*, **XXXIII** (2010), 135–8.

Index

Note: The letter 'f' following a page number indicates a figure and 't' a table.