
Chapter 1

Issues in Diagnosis: Categorical vs. Dimensional

SCOTT O. LILIENFELD AND KRISTIN LANDFIELD

Psychiatric diagnosis is fundamental to the understanding of mental illness. Without it, the study, assessment, and treatment of psychopathology would be in disarray. In this chapter, we examine: (1) the *raison d'être* underlying psychiatric diagnosis; (2) widespread misconceptions regarding psychiatric diagnosis; (3) the present system of psychiatric diagnosis and its strengths and weaknesses; and (4) and fruitful directions for improving this system.

There are a myriad of forms of abnormality housed under the exceedingly broad umbrella of mental disorders. Indeed, the current psychiatric classification system contains well over 350 diagnoses (American Psychiatric Association [APA], (2000)). The enormous heterogeneity of psychopathology makes a formal system of organization imperative. Just as in the biological sciences where Linnaeus' hierarchical taxonomy categorizes fauna and flora and in chemistry where Mendeleev's periodic table orders the elements, a psychiatric classification system helps to organize the bewildering subforms of abnormality. Such a system, if effective, permits us to parse the variegated universe of psychological disorders into more homogeneous, and ideally more clinically meaningful, subtypes.

From the practitioner's initial inchoate impression that a patient's behavior is aberrant to later and better elaborated case conceptualization, diagnosis plays an integral role in the clinical process. Indeed, the essential reason for initiating assessment and treatment is often the observer's sense that "something is just not quite right" about that person. Meehl (1973) commented that the mental health professional's core task is to answer the question, "what does this person have, or what befell him, that makes him different

from those who have not developed clinical psychopathology” (p. 248). Therein lies the basis for psychiatric diagnosis.

General Terminological Issues

Before proceeding, a bit of terminology is in order. It's crucial at the outset to distinguish two frequently confused terms: classification and diagnosis. A system of *classification* is an overarching taxonomy of mental illness, whereas *diagnosis* is the act of placing an individual, based on a constellation of *signs* (observable indicators, like crying in a depressed patient), *symptoms* (subjective indicators, like feelings of guilt in a depressed patient), or both, into a category within that taxonomy. Classification is a prerequisite for diagnosis.

Another key set of terminological issues concerns the distinctions among *syndrome*, *disorder*, and *disease*. As Kazdin (1983) observed, we can differentiate among these three concepts based on our levels of understanding of their *pathology*—the physiological changes that may accompany the condition—and *etiology*, that is, causation (Gough, 1971; Lilienfeld, Waldman, & Israel, 1994).

At the lowest rung of the hierarchy of understanding lie *syndromes*, which are typically constellations of signs and symptoms that co-occur across individuals (syndrome means *running together* in Greek). In syndromes, neither pathology nor etiology is well understood, nor is their causal relation to other conditions established. Antisocial Personality Disorder is a relatively clear example of a syndrome because its signs (e.g., the use of alias) and symptoms (e.g., lack of remorse) tend to covary across individuals. Nevertheless, its pathology and etiology are largely unknown, and its causal relation to other conditions poorly understood (Lykken, 1995).

In rare cases, syndromes may also constitute groupings of signs and symptoms that exhibit minimal covariation across individuals but that point to an underlying etiology (Lilienfeld, Waldman, & Israel, 1994). For example, Gerstmann's syndrome in neurology (Benton, 1992) is marked by four major symptoms: agraphia (inability to write), acalculia (inability to perform mental computation), finger agnosia (inability to differentiate among fingers on the hand), and left-right disorientation. Although these symptoms are negligibly correlated across individuals in the general population, they co-occur dependably following certain instances of parietal lobe damage.

At the second rung of the hierarchy of understanding lie *disorders*, which are syndromes that cannot be readily explained by other conditions. For example, in the present diagnostic system, Obsessive-Compulsive Disorder (OCD) can be diagnosed only if its symptoms (e.g., recurrent fears of contamination) and signs (e.g., recurrent hand washing) cannot be accounted for by a specific phobia (e.g., irrational fear of dirt). Once we rule out other potential causes of OCD symptoms, such as specific phobia, anorexia nervosa, trichotillomania (compulsive hair-pulling), and hypochondriasis, we can be reasonably certain that an individual exhibiting marked obsessions, compulsions, or both, suffers from a well-defined disorder (APA, 2000, p. 463).

At the third and highest rung of the hierarchy of understanding lie *diseases*, which are disorders in which pathology and etiology are reasonably well understood (Kazdin, 1983; McHugh & Slavey, 1998). Sickle-cell anemia is a prototypical disease because its pathology (crescent-shaped erythrocytes containing hemoglobin S) and etiology (two autosomal recessive alleles) have been conclusively identified (Sutton, 1980). For

other conditions that approach the status of bona fide diseases, such as Alzheimer's disease, the primary pathology (senile plaques, neurofibrillary tangles, and granulovacuolar degeneration) has been identified, while their etiology is evolving but incomplete (Selkoe, 1992).

With the possible exception of Alzheimer's disease and a handful of other organic conditions, the diagnoses in our present system of psychiatric classifications are almost exclusively syndromes or, in rare cases, disorders (Kendell & Jablensky, 2003). This fact is a sobering reminder that the pathology in most cases of *psychopathology* is largely unknown, and their etiology poorly understood. Therefore, although we genuflect to hallowed tradition in this chapter by referring to the major entities within the current psychiatric classification system as mental disorders, readers should bear in mind that few are disorders in the strict sense of the term.

Functions of Psychiatric Diagnosis

Diagnosis serves three principal functions for practitioners and researchers alike. We discuss each in turn.

DIAGNOSIS AS COMMUNICATION

Diagnosis furnishes a convenient vehicle for communication about an individual's condition. It allows professionals to be reasonably confident that when they use a diagnosis (such as Dysthymic Disorder) to describe a patient, other professionals will recognize it as referring to the same condition. Moreover, a diagnosis (such as Borderline Personality Disorder) distills relevant information, such as frantic efforts to avoid abandonment and chronic feelings of emptiness, in a short-hand form that aids in other professionals' understanding of a case. Blashfield and Burgess (2007) described this role as "information retrieval." Just as botanists use the name of a species to summarize distinctive features of a specific plant, psychologists and psychiatrists rely on a diagnosis to summarize distinctive features of a specific mental disorder (Blashfield & Burgess, 2007). Diagnoses succinctly convey important information about a patient to clinicians, investigators, family members, managed care organizations, and others.

LINKAGES TO OTHER DIAGNOSES

Psychiatric diagnoses are organized within the overarching nosological structure of other diagnoses. *Nosology* is the branch of science that deals with the systematic classification of diseases. Within this system, most diagnostic categories are arranged in relation to other conditions; the nearer in the network two conditions are, the more closely related they ostensibly are as disorders. For example, Histrionic Personality Disorder (HPD) and Narcissistic Personality Disorder (NPD)—both classified within Cluster B, the dramatic and emotional group of personality disorders—are presumably more closely linked etiologically than are HPD and schizoid personality disorder, a condition falling into Cluster A, the odd, eccentric group of personality disorders. Thus, diagnoses help to locate the patient's presenting problems with the context of both more and less related diagnostic categories.

SURPLUS INFORMATION

Perhaps most important, a diagnosis helps us to learn new things; it affords us surplus information that we did not have previously. Among other things, a diagnosis allows us to generate predictions regarding case trajectory. As Goodwin and Guze (1996) note, perhaps hyperbolically, “diagnosis is prognosis” (Kendler, 1980). The diagnostic label of Bipolar I Disorder describes a distinctive constellation of indicators (e.g., one or more manic or mixed episodes) that discriminates the course, rate of recovery, and treatment response from such related conditions as Major Depression and Bipolar II Disorder, the latter of which is marked by one or more episodes of hypomania and disabling depression.

But a valid diagnosis does considerably more than predict prognosis. Robins and Guze’s (1970) landmark article delineated formal criteria for ascertaining whether a diagnosis is valid. *Validity* refers to the extent to which a diagnosis measures what it purports to measure. More colloquially, validity is truth in advertising: a valid diagnosis is true to its name in that it correlates in expected directions with external criteria. Specifically, Robins and Guze outlined four requirements for the validity of psychiatric diagnoses. According to them, a valid diagnosis offers information regarding:

- *Clinical Description*, including symptomatology, demographics, precipitants, and differences from seemingly related disorders. The lattermost task of distinguishing a diagnosis from similar diagnoses is called *differential diagnosis*;
- *Laboratory Research*, including data from psychological, biological, and laboratory tests;
- *Natural history*, including course and outcome; and
- *Family Studies*, especially studies examining the prevalence of a disorder in the first-degree relatives of *probands*, that is, individuals identified as having the diagnosis in question.

As a further desideratum, some authors have suggested that a valid diagnosis should ideally be able to predict the individual’s response to treatment (Waldman, Lilienfeld, & Lahey, 1995). Nevertheless, this criterion should probably not be mandatory given that the treatment of a condition bears no necessary implications for its etiology. For example, although both schizophrenia and nausea induced by food poisoning generally respond to psychopharmacological agents that block the action of the neurotransmitter dopamine, these two conditions spring from entirely distinct causal mechanisms. Some authors (e.g., Ross & Pam, 1996) have invoked the felicitous phrase *ex juvantibus* reasoning (reasoning backward from what works) to describe the error of inferring a disorder’s etiology from its treatment. Headaches, as the hoary example goes, are not caused by a deficiency of aspirin.

There’s reasonably strong evidence that many mental disorders fulfill Robins and Guze’s (1970) criteria for validity. When these criteria are met, the diagnosis offers additional information about the patient, information that was not available before this diagnosis was made. For example, if we correctly diagnose a patient with schizophrenia, we have learned that this patient:

1. Is likely to exhibit psychotic symptoms that are not solely a consequence of a severe mood disturbance;

2. Has a higher than expected likelihood of exhibiting abnormalities on several laboratory measures, including indices of sustained attention and smooth pursuit eye tracking;
3. Has a higher than average probability of having close biological relatives with schizophrenia and schizophrenia-spectrum disorders, such as schizotypal and paranoid personality disorders;
4. Is likely to exhibit a chronic course, with few or no periods of entirely normal functioning, but approximately a 30 percent chance of overall improvement; and
5. Is likely to respond positively to medications that block the action of dopamine.

Andreasen (1995) extended the Robins and Guze (1970) framework to incorporate indicators from molecular genetics, neurochemistry, and functional and structural brain imaging as additional validating indicators for psychiatric diagnoses (Kendell & Jablensky, 2003). Her friendly amendment to the Robins and Guze criteria allows us to use endophenotypic indicators to assist in the validation of a diagnosis. *Endophenotypes* are biomarkers; that is, “measurable components unseen by the unaided eye along the pathway between disease and distal genotype” (Gottesman & Gould, 2003, p. 636; Waldman, 2005). They are often contrasted with *exophenotypes*, the traditional signs and symptoms of a disorder.

We can view the process of validating psychiatric diagnoses within the overarching framework of *construct validity* (Cronbach & Meehl, 1955; Loevinger, 1957; Messick, 1995), which refers to the extent to which a measure assesses a hypothesized attribute of individuals. As Morey (1991) noted, psychiatric classification systems are collections of hypothetical constructs; thus, the process of validating psychiatric diagnoses is also a process of construct validation. More broadly, we can conceptualize most or even all psychiatric diagnoses as *open concepts* (Meehl, 1977, 1990). Open concepts are marked by (a) fuzzy boundaries, (b) a list of indicators (signs and symptoms) that are indefinitely extendable, and (c) an unclear inner nature.

Recalling that psychiatric diagnoses are open concepts helps us to avoid the perils of premature reification of diagnostic entities (Faust & Miner, 1986). For example, the present diagnostic criteria for schizophrenia are not isomorphic with the latent construct of schizophrenia; they are merely fallible, albeit somewhat valid, indicators of this construct. Yet, the past few decades have occasionally witnessed a troubling tendency to reify and deify the categories within the current classification system, with some authors regarding them as fixed Platonic essences rather than rough approximations to the true state of nature (Ghaemi, 2003; Michels, 1984). This error is manifested, for example, when journal or grant reviewers criticize researchers for examining alternative operationalizations of mental disorders that depart from those in the current diagnostic manual (see section Psychiatric Classification from *DSM-I* to the Present).

In a classic article, Cronbach and Meehl (1955) adopted from neopositivist philosophers of science the term *nomological network* to designate the system of lawful relationships conjectured to hold between theoretical entities (states, structures, events, dispositions) and observable indicators. They selected the network metaphor to emphasize the structure of such systems in which the nodes of the network, representing the postulated theoretical entities, are connected by the strands of the network, representing the lawful relationships hypothesized to hold among the entities (Garber & Strassberg, 1991).

For Cronbach and Meehl (1955), construct validation is a progressive and never ending process of testing the links between hypothesized strands of the nomological network, especially those that connect latent constructs—which include psychiatric diagnoses (e.g., schizophrenia and major depression)—to manifest indicators—which include the external criteria (e.g., laboratory tests and family history) laid out by Robins and Guze (1970). The more such construct-to-manifest indicator links are corroborated, the more certain we can be that our conception of the diagnosis in question is accurate. From this perspective, the approach to diagnostic validation outlined by Robins and Guze is merely one specific instantiation of construct validation.

One shortcoming of the Robins and Guze (1970) approach to construct validation is its exclusive emphasis on *external validation*, that is, the process of ascertaining the construct's associations with correlates that lie outside of the construct itself. As Skinner (1981, 1986; also Loevinger, 1957) observed, *internal validation*, ascertaining the construct's inner structure, is also a key component of construct validation. Internal validation can help investigators to test hypotheses regarding a construct's homogeneity (versus heterogeneity) and factor structure (Waldman et al., 1995). For example, if analyses suggest that a diagnosis consists of multiple and largely independent subtypes, the validity of the diagnosis would be called into question.

In summary, valid psychiatric diagnoses serve three primary functions:

1. They summarize distinctive features of a disorder and thereby allow professionals to communicate clearly with one another;
2. They place each diagnosis under the umbrella structure of other diagnoses. This nosological framework links one diagnosis to both more and less related diagnoses; and
3. They provide practitioners and researchers with surplus information regarding diagnosed patients' clinical profile, laboratory findings, natural history, family history, and possibly response to treatment; they may also offer information regarding endophenotypic indicators.

Misconceptions Regarding Psychiatric Diagnosis

Beginning psychology graduate students and much of the general public hold a plethora of misconceptions regarding psychiatric diagnosis; we examine five such misconceptions here. Doing so will also permit us to introduce a number of key principles of psychiatric diagnosis. As we will discover, refuting each misconception regarding psychiatric diagnosis affirms at least one important principle.

MISCONCEPTION # 1: “MENTAL ILLNESS” IS A MYTH

The person most closely associated with this position is Szasz (1960), who has argued famously for over 40 years that the term mental illness is a false and misleading metaphor (Schaler, 2004). For Szasz, individuals who psychologists and psychiatrists term mentally ill actually suffer from *problems in living* (that is, difficulties in adjusting their behaviors to the demands of society). Moreover, Szasz contended that mental health professionals often apply the mental illness label to nonconformists who jeopardize the

status quo (Sarbin, 1969; Szasz, 1960). This label serves as a convenient justification for forcing maladjusted, malcontented, and maverick members of society to comply with prevailing societal norms.

Specifically, Szasz maintained that medical disorders can be clearly recognized by a lesion to the anatomical structure of the body, but that the disorder concept cannot be extended to the mental realm because there is no such lesion to indicate deviation from the norm. According to him only the body can become diseased, so mentally ill people do not suffer from an illness akin to a medical disorder.

It is undeniable that psychiatric diagnoses are sometimes misapplied. Nevertheless, this legitimate pragmatic concern must be logically separated from the question of whether the mental illness concept itself exists (Wakefield, 1992). We should recall the logical principle of *abusus non tollit usum*: historical and sociological misuses of a concept do not negate its validity.

Wakefield (1992) and others (Kendell, 1975) have observed that the Szaszian argument is problematic on several fronts. Among others, it assumes that medical disorders are in every case traceable to discernible lesions in an anatomical structure, and that all lesions give rise to medical disorders. Yet identifiable lesions cannot be found in certain clear-cut medical diseases—such as trigeminal neuralgia and senile pruritis—and certain identifiable lesions, such as albinism, are not regarded as medical disorders (Kendall, 1975; Wakefield, 1992). Szasz's assertion that identifiable lesions are essentially synonymous with medical disorders is false; therefore, his corollary argument that mental disorders cannot exist because they are not invariably associated with identifiable lesions is similarly false.

MISCONCEPTION # 2: PSYCHIATRIC DIAGNOSIS IS MERELY PIGEON-HOLING

According to this criticism, when we diagnose people with a mental disorder, we deprive them of their uniqueness: We imply that all people within the same diagnostic category are alike in all important respects.

To the contrary, a psychiatric diagnosis does nothing of the sort; it implies only that all people with that diagnosis are alike in at least one important way. Psychologists and psychiatrists are well aware that even within a given diagnostic category, such as Schizophrenia or Bipolar Disorder, people differ dramatically in their race and cultural background, personality traits, interests, and cognitive skills (APA, 2000, p. xxxi).

MISCONCEPTION # 3: PSYCHIATRIC DIAGNOSES ARE UNRELIABLE

Reliability refers to the consistency of a diagnosis. As many textbooks in psychometrics remind us, reliability is a prerequisite for validity but not vice versa. Just as a bathroom scale cannot validly measure weight if it yields dramatically different weight estimates for the same person over brief periods of time, a diagnosis cannot validly measure a mental disorder if it yields dramatically different scores on measures of psychopathology across times, situations, and raters.

Because validity is not a prerequisite for reliability, extremely high reliability can exist without validity. A researcher who based his diagnoses of schizophrenia on patients' heights would end up with extremely reliable but entirely invalid diagnoses of schizophrenia.

There are three major subtypes of reliability. Contrary to popular (mis)conception, these subtypes are frequently discrepant with one another, so high levels of reliability for one metric do not necessarily imply high levels for the others.

Test-retest reliability refers to the stability of a diagnosis following a relatively brief time interval, typically about a month. In other words, after a short time lapse, will patients receive the same diagnoses? Note that we wrote *brief* and *short* in the previous sentences; marked changes following lengthy time lapses, such as several years, may reflect genuine changes in patient status rather than the measurement error associated with test-retest unreliability.

In general, we assess test-retest reliability using either a Pearson correlation coefficient or, more rigorously, an *intraclass correlation* coefficient. Intraclass correlations tend to provide the most stringent estimates of test-retest reliability because, in contrast to Pearson correlations, they are influenced not merely by the rank ordering and differences among people's scores, but by their absolute magnitude.

Our evaluation of a diagnosis' test-retest reliability hinges on our conceptualization of the disorder. We should anticipate high test-reliability only for diagnoses that are trait-like, such as personality disorders, or that tend to be *chronic* (long-lasting), such as schizophrenia. In contrast, we should not necessarily anticipate high levels of test-test reliabilities for diagnoses that tend to be *episodic* (intermittent), such as major depression.

Internal consistency refers to the extent to which the signs and symptoms comprising a diagnosis "hang together," that is, correlate highly with one another. We generally assess internal consistency using such metrics as coefficient alpha (Cronbach, 1951) or the mean interitem correlation. Cronbach's alpha can overestimate the homogeneity of a diagnosis, however, if this diagnosis contains numerous signs and symptoms because this statistic is affected by test length (Schmidt, Le, & Ilies, 2003). We should anticipate high levels of internal consistency for most conditions in the current classification system given that most are syndromes, which are typically constellations of signs and symptoms that covary across people.

Inter-rater reliability is the degree to which two or more observers, such as different psychologists or psychiatrists, agree on the diagnosis of a set of individuals. High inter-rater reliability is a prerequisite for all psychiatric diagnoses, because different observers must agree on the presence or absence of a condition before valid research on that condition can proceed.

Many early studies of psychiatric diagnosis operationalized inter-rater reliability in terms of percentage agreement, that is, the proportion of cases on which two or more raters agree on the presence or absence of a given diagnosis. Nevertheless, measures of percentage agreement tend to overestimate inter-rater reliability. Here's why: imagine two diagnosticians working in a setting (e.g., an outpatient phobia clinic) in which the *base rate* (prevalence) of the diagnosis of specific phobia is 95%. The finding that they agree with each other on the diagnosis of specific phobia 95% of the time would hardly be impressive and could readily be attributed to chance. As a consequence, most investigators today operationalize inter-rater reliability in terms of the *kappa coefficient*, which assesses the degree to which raters agree on a diagnosis after correcting for chance, with chance being the base rate of the disorder in question. Nevertheless, the kappa coefficient often provides a conservative estimate of inter-rater reliability, as the correction for chance sometimes penalizes raters for their independent expertise (Meyer, 1997).

Many laypersons and even political pundits believe that psychiatric diagnoses possess low levels of reliability, especially inter-rater reliability. This perception is probably fueled by high-profile media coverage of dueling expert witnesses in criminal trials in which one expert diagnoses a defendant as schizophrenic, for example, and another diagnoses him as normal. After the widely publicized 1982 trial of John Hinckley, who was acquitted on the basis of insanity for his attempted assassination of then-president Ronald Reagan, political commentator George Will maintained (on national television) that the disagreements among expert witnesses regarding Hinckley's diagnosis merely bore out what most people already knew: psychiatric diagnosis is wildly unreliable (Lilienfeld, 1995).

Yet there is a straightforward explanation for such disagreement: Given the adversarial nature of our legal system, the prosecution and defense typically go out of their way to find expert witnesses who will present their point of view. This inherently antagonistic arrangement virtually guarantees that the inter-rater reliabilities of experts in criminal trials will be modest at best.

Certainly, the inter-rater reliability of psychiatric diagnoses is far from perfect. Yet for most major mental disorders, such as schizophrenia, mood disorders, anxiety disorders, and alcohol dependence (alcoholism), inter-rater reliabilities are typically about as high—intraclass correlations between raters of 0.8 or above, out of a maximum of 1.0—as those for most well established medical disorders (Matarazzo, 1983). Still, the picture is not entirely rosy. For most personality disorders in particular, inter-rater reliabilities tend to be considerably lower than for other conditions (Zimmerman, 1994), probably because most of these disorders comprise highly inferential constructs (e.g., lack of empathy) that raters find difficult to assess during the course of brief interviews.

MISCONCEPTION # 4: PSYCHIATRIC DIAGNOSES ARE INVALID

From the standpoint of Szasz (1960) and other critics of psychiatric diagnosis (Eysenck, Wakefield, & Friedman, 1983), psychiatric diagnoses are largely useless because they do not provide us with new information. According to them, diagnoses are merely descriptive labels for behaviors we do not like. Millon (1975) proposed a helpful distinction between psychiatric *labels* and *diagnoses*; a label simply describes behaviors, whereas a diagnosis helps to explain them.

When it comes to a host of informal pop psychology labels, like sexual addiction, Peter Pan syndrome, co-dependency, shopping disorder, and road rage disorder, Szasz and his fellow critics probably have a point. Most of these labels merely describe collections of socially problematic behavior and do not provide us with much, if any, new information (McCann, Shindler, & Hammond, 2003). The same may hold for some personality disorders in the current classification system; for example, the diagnosis of Dependent Personality Disorder appears to do little more than describe ways in which people are pathologically dependent on others, such as relying excessively on others for reassurance and expecting others to make everyday life decisions for them.

Yet, as we have already seen, many psychiatric diagnoses, such as Schizophrenia, Bipolar Disorder, and Panic Disorder, do yield surplus information (Robins & Guze, 1970; Waldman et al., 1995) and, therefore, possess adequate levels of validity. Nevertheless, because construct validation, like all forms of theory testing in science, is a never ending process, the validity of these diagnoses is likely to improve over time with subsequent revisions to the present classification system.

MISCONCEPTION # 5: PSYCHIATRIC DIAGNOSES STIGMATIZE PEOPLE, AND OFTEN RESULT IN SELF-FULFILLING PROPHECIES

According to advocates of labeling theory, including Szasz (1960), Sarbin (1969), and Scheff (1975), psychiatric diagnoses produce adverse effects on labeled individuals. They argue that diagnostic labels not only stigmatize patients, but also frequently become self-fulfilling prophecies, leading observers to interpret ambiguous and relatively mild behaviors (e.g., occasional outbursts of anger) as reflecting serious mental illness.

A sensational 1973 study by Rosenhan appeared to offer impressive support for labeling theory. Rosenhan, along with seven other normal individuals, posed as pseudopatients (fake patients) in 12 U.S. psychiatric hospitals (some of the pseudopatients presented at more than one hospital). They informed the admitting psychiatrist only that they were hearing a voice saying “empty, hollow, and thud.” All were promptly admitted to the hospital and remained there for an average of three weeks, despite displaying no further symptoms or signs of psychopathology. In 11 of 12 cases, they were discharged with diagnoses of schizophrenia in remission (the 12th pseudopatient was discharged with a diagnosis of manic depression in remission).

Rosenhan (1973) noted that the hospital staff frequently interpreted pseudopatients’ innocuous behaviors, such as note taking, as indicative of abnormality. In case summaries, these staff also construed entirely run of the mill details of pseudopatients’ life histories, such as emotional conflicts with parents during adolescence, as consistent with their present illness. These striking results led Rosenhan to conclude that psychiatric labels color observers’ perceptions of behavior, often to the point that they can no longer distinguish mental illness from normality.

Even today, some writers interpret Rosenhan’s findings as a resounding affirmation of labeling theory (e.g., Slater, 2004). Yet, the evidence for labeling theory is less impressive than it appears. As Spitzer (1975) observed, the fact that all 12 of Rosenhan’s pseudopatients were released with diagnoses in remission (meaning showing no indications of illness) demonstrates that the psychiatrists who treated them were in all cases able to distinguish mental illness from normality. Spitzer went further, demonstrating in a survey of psychiatric hospitals that in remission diagnoses of previously psychotic patients are exceedingly infrequent, showing that the psychiatrists in Rosenhan’s study successfully made an extremely rare judgment with perfect consensus.

Although incorrect psychiatric diagnoses can engender stigma, at least in the short run (Harris, Milich, Corbett, Hoover, & Brady, 1992; Milich, McAninch, & Harris, 1992) there is scant evidence to support the popular claim that correctly applied psychiatric diagnoses do so. The lion’s share of the research suggests that stigma is a consequence not of diagnostic labels, but of disturbed and sometimes disturbing behavior that precedes labeling (Link & Cullen, 1990; Ruscio, 2004). For example, within 30 minutes or less, children begin to react negatively to children with Attention-Deficit/Hyperactivity Disorder (ADHD) who have joined their peer group (Milich et al., 1992; Pelham & Bender, 1982).

Contrary to the tenets of labeling theory, there is evidence that accurate psychiatric diagnoses sometimes reduce stigma, because they provide observers with at least a partial explanation for otherwise inexplicable behaviors (Ruscio, 2004). For example, adults tend to evaluate mentally retarded children more positively when these children are labeled as mentally retarded than when they are not (Seitz & Geske, 1976), and peers rate the essays of children with ADHD more positively when these children are labeled with ADHD than when they are not (Cornez-Ruiz & Hendricks, 1993).

What Is Mental Disorder?

Our discussion up to this point presupposes that the boundaries of the higher-order concept of “disorder,” including mental disorder, are clear-cut or at least reasonably well-delineated.¹ To develop a classification system of disorders, one must first be able to ascertain whether a given condition is or is not a disorder. Yet the answer to the question of how best to define disorder, including mental disorder, remains elusive (Gorenstein, 1992). The issues here are of more than academic interest, because each revision of psychiatry’s diagnostic manual has been marked by contentious disputes regarding whether such conditions as Attention-Deficit/Hyperactivity Disorder, Posttraumatic Stress Disorder, and Premenstrual Dysphoric Disorder are *really* disorders (Wakefield, 1992). The fact that homosexuality was removed from the formal psychiatric classification system in 1974 by a majority vote of the membership of the American Psychiatric Association (Bayer & Spitzer, 1982) further demonstrates that these debates are frequently resolved more by group consensus than by scientific research.

Here we evaluate several influential attempts to delineate the boundaries of disorder. As we will discover, each approach has its limitations but each captures something important about the concept of disorder. As we will also discover, these approaches differ in the extent to which they embrace an *essentialist* as opposed to a *nominalist* view of disorder (Ghaemi, 2003; Scadding, 1996). Advocates of an essentialist view (Widiger & Trull, 1985) believe that all disorders share some essence or underlying property, whereas advocates of a nominalist view (Lilienfeld & Marino, 1999) believe that the higher-order concept of disorder is a social construction that groups together a variety of largely unrelated conditions for the purposes of social or semantic convenience.

STATISTICAL MODEL

Advocates of a *statistical model*, such as Cohen (1981), equate disorder with statistical rarity. According to this view, disorders are abnormal because they are infrequent in the general population. This definition accords with findings that many mental disorders are indeed rare; schizophrenia, for example, is found in about 1% of the population across much of the world (APA, 2000).

Yet, a purely statistical model falls short on at least three grounds. First, it offers no guidance for where to draw cut-offs between normality and abnormality. In many cases, these cut-offs are scientifically arbitrary. Second, it is silent on the crucial question of which dimensions are relevant to abnormality. As a consequence, a statistical model misclassifies high scores on certain adaptive dimensions (like intelligence, creativity, and altruism) as inherently abnormal. Moreover, it does not explain why high scores on certain dimensions (e.g., anxiety) but not others (e.g., hair length) are pertinent to psychopathology. Third, by definition a statistical model assumes that all common conditions are normal (Wakefield, 1992). Yet the common cold is still an illness despite its essentially 100% lifetime prevalence in the population, and the Black Death (bubonic plague) was still an illness in the mid-1300s despite wiping out approximately one-third of the European population.

¹ In our discussion of the definition of disorder, we use the term disorder, including mental disorder, generically to refer to all medical and psychological conditions and do not distinguish disorder from disease (Wakefield, 1992).

SUBJECTIVE DISTRESS MODEL

Proponents of a *subjective distress* model maintain that the core feature distinguishing disorder from nondisorder is psychological pain. This model unquestionably contains a large kernel of truth; many serious mental illnesses (such as Major Depression, Obsessive-Compulsive Disorder, Generalized Anxiety Disorder, and Gender Identity Disorder) are marked by considerable distress, even anguish.

The subjective distress model also falls short of an adequate definition of mental illness, because it fails to distinguish *ego-dystonic* conditions—those that conflict with one's self-concept—from *ego-syntonic* conditions—those that are consistent with one's self-concept. Although most mental disorders are ego-dystonic, some (like Antisocial Personality Disorder) are largely or entirely ego-syntonic, because individuals with these conditions frequently see nothing wrong with their behavior. They experience little or no distress in conjunction with their condition, and frequently seek treatment only when demanded by courts or significant others, or when their condition is complicated by a secondary condition that generates interpersonal difficulties (e.g., alcoholism). Moreover, approximately half of patients with schizophrenia and other severe psychotic conditions are afflicted with *anosognosia*, meaning that they are not aware of the fact that they are ill (Amador & Paul-Ouduard, 2000).

BIOLOGICAL MODEL

Proponents of a *biological model* (Kendell, 1975) contend that disorder can be defined in terms of a biological or evolutionary disadvantage to the organism, such as reduced lifespan or fitness (i.e., the ability to pass on genes to subsequent generations). Indeed, some mental disorders are associated with biological disadvantages; for example, major depression is associated with a dramatically increased risk for completed suicide (Joiner, 2006), and between 5 and 10% of patients with anorexia nervosa eventually die from complications due to starvation (Goodwin & Guze, 1996).

A biological model, however, also falls prey to numerous counterexamples. For example, being a soldier in front-line combat is not a disorder despite its average adverse effect on longevity and fitness. Conversely, some relatively mild psychological conditions, such as most specific phobias, are probably not associated with decreased longevity or fitness, yet are still mental disorders.

NEED FOR TREATMENT

One parsimonious definition is simply that disorders are a heterogeneous class of conditions all characterized by a perceived need for medical intervention on the part of health (including mental health) professionals (Kraupl Taylor, 1971). Like other definitions, this definition captures an important truth: Many or most mental disorders, such as Schizophrenia, Bipolar Disorder, and Obsessive-Compulsive Disorder, are indeed viewed by society as necessitating treatment. Nevertheless, this definition too falls victim to counterexamples. For example, pregnancy clearly is associated with a perceived need for medical intervention, yet it is not regarded as a disorder.

HARMFUL DYSFUNCTION

In an effort to remedy the shortcomings of extant models of disorder, Wakefield (1992) proposed a hybrid definition that incorporates both essentialist and nominalist features.

According to Wakefield, all disorders, including all mental disorders, are harmful dysfunctions: socially devalued (harmful) breakdowns of evolutionarily selected systems (dysfunctions). For example, according to Wakefield, panic disorder is a mental disorder because it (a) is negatively valued by society and often by the individual afflicted with it, and (b) reflects the activation of the fight-flight system in situations for which it was not evolutionary selected, namely those in which objective danger is absent. In other words, panic attacks are false alarms (Barlow, 2001). Wakefield's operationalization of disorder has its strengths; for example, it acknowledges (correctly) that most and perhaps all disorders are viewed negatively by others. The concept of disorder, including mental disorder, is clearly associated with social values. As Wakefield (1992) noted, however, social devaluation is not sufficient to demarcate disorder from nondisorder, claims by Szasz (1960) to the contrary. For example, rudeness, laziness, slovenliness, and even racism are viewed negatively by society, but are not disorders (for a dissenting view regarding racism, see Poussaint, 2002). Therefore, Wakefield contends something else is necessary to distinguish disorder from nondisorder, namely evolutionary dysfunction.

Nevertheless, the dysfunction component of Wakefield's analysis appears to fall prey to counterexamples. In particular, many medical disorders appear to be adaptive defenses against threat or insult. For instance, the symptoms of influenza (flu), such as vomiting, coughing, sneezing, and fever, are all adaptive efforts to expel an infectious agent rather than failures or breakdowns in an evolutionarily selected system (Lilienfeld & Marino, 1999; Nesse & Williams, 1994). Such counterexamples appear to falsify the harmful dysfunction analysis. Similarly, many psychological conditions appear to be adaptive reactions to perceived threat. For example, in contrast to other specific phobias, blood phobia is marked by a coordinated set of dramatic parasympathetic reactions—especially rapid decreases in heart rate and blood pressure—that were almost surely evolutionarily selected to minimize blood loss (Barlow, 2001). Although these responses may not be especially adaptive in the early 21st century, they were adaptive prior to the advent of Band-Aids, tourniquets, and anticoagulants (Lilienfeld & Marino, 1995).

ROSCHIAN ANALYSIS

An alternative approach to defining disorder is radically different. According to a *Roschian analysis*, the attempt to define disorder explicitly is sure to fail because disorder is intrinsically undefinable (Gorenstein, 1992). Drawing on the work of cognitive psychologist Eleanor Rosch (Rosch, 1973; Rosch & Mervis, 1975), advocates of a Roschian analysis contend that the concept of mental disorder lacks defining (i.e., singly necessary and jointly sufficient) features and possesses intrinsically fuzzy boundaries. In this respect, mental disorder is similar to many other concepts. For example, the concept of a chair lacks strictly defining features (e.g., a human-made object with four legs that someone one can sit on does not succeed as a defining feature, because one can sit on a table and many chairs do not have four legs) and displays unclear boundaries. In addition, the concept of mental disorder, like many other concepts, is organized around a prototype that shares all of the features of the category. Just as certain chairs (e.g., a typical office chair) are more chair-like than others (e.g., a bean-bag), certain mental disorders (e.g., Schizophrenia) are more disorder-like than others (e.g., Hypoactive Sexual Desire Disorder). Not surprisingly, it is at the fuzzy boundaries of disorder

where controversies concerning whether a psychological condition is really a disorder most frequently arise. According to the Roschian analysis, these controversies are not only inevitable, but also not resolvable by scientific data.

Even if the Roschian analysis is correct (see Wakefield, 1999, and Widiger, 1997, for criticisms of this approach), it would not imply that specific mental disorders themselves are not amenable to scientific inquiry. As Gorenstein (1992) noted, the concept of a “drug” is inherently undefinable; there are no scientific criteria for deciding whether caffeine, nicotine, or some other widely used but addictive substances are drugs. Yet, this problem has not stopped psychopharmacologists from studying specific drugs’ properties, modes of action, or behavioral effects. Nor should the absence of an explicit definition of mental disorder preclude psychopathology researchers from investigating the diagnosis, etiology, treatment, and prevention of Schizophrenia, Major Depression, Panic Disorder, and other conditions.

The recent controversy regarding whether Pluto is a planet is another telling case in point. Following weeks of heated discussion, the International Astronomical Union caused a furor in 2006 by ignominiously demoting Pluto from its lofty planetary status. Yet, as most witnesses to this acrimonious debate acknowledged, the question of whether Pluto is genuinely a planet is largely or entirely arbitrary from a scientific standpoint. One prominent astronomer, Michael Brown (2006), wrote in the *New York Times* that:

The term “planet” is similar to “continent.” The word helps us organize our world, but the division between continents and subcontinents is thoroughly arbitrary. Yet no union of geologists has tried to vote on a definition of “continent,” and no one is concerned that letting culture determine the difference between Australia, the smallest continent, and Greenland, the largest island, somehow erodes science. (p. 17)

Just as the question of Pluto’s planet-hood has had no discernable impact on planetary astronomers’ daily activities, the question of whether controversial psychological conditions are mental disorders should have no effect on the day-to-day activities of practitioners or psychopathology researchers.

Psychiatric Classification from *DSM-I* to the Present

Prior to the 1950s, the state of psychiatric classification in the United States was largely disorganized, as no standard system was in place for operationalizing specific mental disorders. Indeed, prior to World War I, there was scant interest in developing a systematic classification of mental disorders (Grob, 1991), and even after World War I no consensual system of classification was in place for over three decades. As a consequence, what one diagnostician meant by major depression might bear minimal correspondence to what another diagnostician meant by the same term.

DSM-I AND *DSM-II*

This situation changed in 1952, when the American Psychiatric Association released the first edition of its *Diagnostic and Statistical Manual of Mental Disorders*, abbreviated as *DSM-I* (APA, 1952). Although *DSM-I* was a slim 132 pages in length, it was a landmark. For the first time, it offered reasonably clear, albeit brief, descriptions of major

psychiatric diagnoses, thereby facilitating inter-rater reliability among clinicians and researchers. Here, for example, was the description for “manic depressive action, depressed type” (later to become “major depression”) in *DSM-I*:

Here will be classified those cases with outstanding depression of mood and with mental and motor retardation and inhibition; in some cases there is much uneasiness and apprehension. Perplexity, stupor or agitation may be prominent symptoms, and may be added to the diagnosis as manifestations. (APA, 1952, p. 25)

DSM-II appeared 16 years later (APA, 1968) and was similar in approach and scope to *DSM-I*, although it provided somewhat greater detail concerning the signs and symptoms of many diagnoses. Despite their strengths, *DSM-I* and *DSM-II* suffered from several notable weaknesses, three of which we discuss here:

1. The inter-rater reliabilities of many of their diagnoses were still problematic, probably because these manuals consisted of global and often vague descriptions of mental illnesses that necessitated considerable subjective judgment on the part of diagnosticians. For example, returning to the description of manic depressive reaction, depressed type, *DSM-I* is silent on what qualifies as “outstanding depression,” and how much motor retardation and inhibition are necessary for the diagnosis.
2. *DSM-I* and *DSM-II* were not theoretically agnostic. In particular, they were influenced by psychoanalytic concepts of mental disorders and often made references to defense mechanisms and other concepts derived from Freudian theory. As a consequence, diagnosticians whose orientation was not psychoanalytic, such as behaviorists, cognitive-behaviorists, or humanistic-existential psychologists, found these classification systems difficult to use. *DSM-I* and *DSM-II* also conceptualized mental disorders largely from the perspective of psychiatrist Adolph Meyer (1866–1950), who regarded most forms of psychopathology as aberrant reactions to life events (Lief, 1948). Hence, the use of the term reaction in the diagnosis of manic depressive reaction, depressed type and many other *DSM-I* and *DSM-II* diagnoses. Nevertheless, this assumption was based more on plausible theoretical conjecture than on evidence.
3. Despite their Meyerian emphasis, *DSM-I* and *DSM-II* focused almost exclusively on patients’ mental disorders per se, and largely neglected to consider contextual factors, such as co-occurring medical conditions, life stressors, and adaptive functioning, which can play key roles in the etiology and maintenance of psychopathology.

***DSM-III* AND BEYOND**

Largely in response to these criticisms, the American Psychiatric Association, with psychiatrist Robert Spitzer at the helm, released *DSM-III* in 1980 (APA, 1980). As most historians of psychiatric classification and diagnosis now recognize, *DSM-III* was an important revision of the diagnostic manual; it represented a radical change in thinking and approach from all that came before, and has provided the template for all that has come since (Klerman, 1984; Mayes & Horwitz, 2005). In this respect, it was every bit as much a landmark, if not more, than *DSM-I* was. Coming in at a hefty 494 pages, a nearly four-fold increase from *DSM-II*, *DSM-III* not only dramatically increased the

coverage of mental disorders—from 163 to 224—but also presented far more detailed guidelines than its predecessors for establishing diagnoses. The operational and philosophical approach of *DSM-III* is often termed *neo-Kraepelinian* (Compton & Guze, 1995) because it followed in the footsteps of the great German psychiatrist Emil Kraepelin (1856–1926), who grouped and differentiated psychological conditions on the basis on their signs, symptoms, and natural history.

Diagnostic Criteria, Algorithms, and Hierarchical Exclusion Rules

In accord with its neo-Kraepelinian emphasis, *DSM-III* instituted several major changes in psychiatric classification and diagnosis. First and foremost, it standardized: (a) *diagnostic criteria*, and (b) *algorithms*, or decision rules, for each diagnosis. Rather than merely describing each diagnosis as *DSM-I* and *DSM-II* had done, *DSM-III* explicitly delineated the signs and symptoms comprising each diagnosis and the method by which these signs and symptoms needed to be combined to establish each diagnosis. In these respects, it was influenced heavily by the pioneering efforts of the St. Louis group at Washington University (including Robins, Guze, Winokur, and other giants of descriptive psychopathology), who had introduced preliminary diagnostic criteria and algorithms for 14 major mental disorders in the early 1970s (Feighner et al., 1972).

For example, to meet criteria for the diagnosis of major depressive episode, *DSM-III* required that clients: (1) experience “dysphoric mood or loss of interest or pleasure in all or almost all activities” (p. 213; with dysphoric mood described in terms of seven symptoms, including depression, hopelessness, and irritability), and (2) experience at least four of eight symptoms, such as poor appetite, insomnia, loss of energy, difficulty thinking and concentrating, nearly every day for at least a two week period. Compare the specificity of these criteria with the skimpy and highly impressionistic description in *DSM-I* presented earlier.

DSM-III also outlined *hierarchical exclusion rules* for many diagnoses; such rules prevent clinicians and researchers from making these diagnoses if other diagnoses can account for their clinical picture. For example, *DSM-III* forbade clinicians and researchers from making a diagnosis of major depressive episode if the episode was superimposed on Schizophrenia, Schizophreniform Disorder, or a Paranoid Disorder, or if it appeared to be due to either an organic mental disorder (e.g., hypothyroidism) or uncomplicated bereavement (a prolonged grief reaction). Among other things, hierarchical exclusion rules remind diagnosticians to think organic: that is, to rule out potential physical causes of mental disorders before diagnosing them (Morrison, 1997).

DSM-III's use of diagnostic criteria, algorithms, and hierarchical exclusion rules has been criticized by many commentators as the Chinese menu approach to diagnosis (choose three from column A, two from column B, four from column C). Despite these criticisms, there is evidence that this approach has markedly decreased the subjectivity of diagnostic decision-making and increased the inter-rater reliabilities of many diagnoses (Spitzer, Forman, & Nee, 1979). However, some authors argue that these increases are exaggerated by *DSM-III*'s proponents (Kirk & Kutchins, 1992).

The inter-rater reliability of *DSM* diagnoses has also been enhanced by the development of *structured* and *semi-structured diagnostic interviews*, such as the Structured Clinical Interview for *DSM* (SCID; First, Spitzer, Gibbon, & Williams, 2002), which are coordinated explicitly around *DSM* criteria. These interviews consist of standardized questions—to be read verbatim by interviewers—and required and suggested follow-up probes with which to assess specific diagnostic criteria. For example, the SCID provides

the following question to assess the criterion of current, unexpected panic attacks in the current *DSM* diagnosis of Panic Disorder: “Have you ever had a panic attack, when you suddenly felt frightened or anxious or suddenly developed a lot of physical symptoms?” If the respondent replies yes, the SCID instructs the interviewer to ask, “Have these attacks ever come on completely out of the blue—in situations where you didn’t expect to be nervous or uncomfortable?” (First et al., 2002).

Theoretical Agnosticism

In sharp contrast to its predecessors, *DSM-III* was agnostic with respect to etiology (with the exception of one diagnosis, Posttraumatic Stress Disorder, which required the presence of a traumatic event ostensibly tied to the symptoms of the disorder). In particular, *DSM-III* assiduously shunned concepts, such as defense mechanisms, that were tied to psychoanalysis or other specific theoretical orientations. By doing so, it permitted practitioners and researchers of varying persuasions to use the manual with equal ease and comfort. It also facilitated scientific progress by allowing researchers to pit differing theoretical orientations against each other to determine which offered the most scientifically supported etiological explanations for specific disorders (Wakefield, 1998).

Multiaxial Approach

DSM-III adopted a *multiaxial* approach to diagnosis, in which each client is described along a series of axes (that is, dimensions). A multiaxial approach forces clinicians to adopt a more *holistic* approach to diagnosis by considering variables in addition to the individuals’ mental disorders. In *DSM-III* (and its revision), the first two axes are restricted to mental illnesses, and the last three axes assess other dimensions often relevant to psychological functioning.

On Axis I most of the major mental disorders are found, including Schizophrenia and other psychotic disorders, mood disorders, anxiety disorders, impulse control disorders, eating disorders, sleep disorders, and substance-related disorders. On Axis II mental retardation and the *personality disorders* are found, believed to be extremes of personality traits that are inflexible, maladaptive, or both. The Axis I–Axis II distinction, although at times fuzzy, ostensibly reflects the difference between conditions (e.g., Major Depression and Panic Disorder) that tend to be superimposed on the individuals’ pre-existing functioning (Axis I) and conditions (e.g., Borderline Personality Disorder), which tend to capture the person’s longstanding ways of viewing and relating to the world (Axis II). More colloquially and perhaps less precisely, Axis I is intended to assess what the person *has*, whereas Axis II is intended to assess what the person *is*.

Axis III assesses medical disorders, which again reminds clinicians to consider physical conditions that can mimic or complicate the course of psychological disorders. Axis III is especially important given estimates that 50% of psychiatric patients suffer from at least one major medical condition (Cooper, 2007). Axis IV assesses psychosocial stressors, including recent major life events, and Axis V assesses the individual’s overall level of adaptive functioning on a 1–100 Global Assessment of Functioning (GAF) scale, with 100 representing optimal functioning.

DSM-III-R and DSM-IV

DSM-III-Revised (*DSM-III-R*), which appeared in 1987, and *DSM-IV*, which appeared in 1994 (and in a more expanded text revision in 2000), retained all of the major features and innovations of *DSM-III* (APA, 1987, 1994, 2000). Nevertheless, they

continued to increase their coverage of psychopathology; *DSM-IV*, now 943 pages long, contains 374 diagnoses (APA, 2000).

Both *DSM-III-R* and *DSM-IV* gradually moved away from a *monothetic approach* to diagnosis, emphasized in much of *DSM-III*, toward a *polythetic approach*. In a monothetic approach, the signs and symptoms are singly necessary and jointly sufficient for a diagnosis. In contrast, in a polythetic approach the signs and symptoms are neither necessary nor sufficient for a diagnosis.

The potential disadvantage of a polythetic approach is extensive heterogeneity at the symptom and (perhaps) etiological levels. In *DSM-IV*, for example, 256 different symptom combinations are compatible with a diagnosis of Borderline Personality Disorder. It is implausible that the etiologies of all of these combinations are similar, let alone identical. It is even possible for two people to meet *DSM-IV* criteria for Obsessive-Compulsive Disorder yet share no diagnostic criteria in common (Widiger, 2007). Nevertheless, most scholars agree that the potential disadvantage of symptomatic heterogeneity is outweighed by the higher inter-reliability of the polythetic approach (Widiger, Frances, Spitzer, & Williams, 1991). In a monothetic approach, a disagreement about the presence or absence of only one criterion necessarily leads to a disagreement about the presence or absence of the diagnosis. In contrast, in a polythetic approach, such disagreement often has no impact on levels of agreement about the presence or absence of the diagnosis, because raters can still agree on the presence or absence of the diagnosis even if they disagree on one or more specific criteria.

The shift toward a polythetic approach is also an implicit nod to the fact that few, if any, signs and symptoms of psychopathology are *pathognomonic*. A pathognomonic indicator is characteristic of a disorder that can be used by itself to establish its diagnosis. For example, Koplik's spots—tiny spots in the mouth that look much like grains of sand surrounded by red rings—are essentially pathognomonic for measles. A sign or symptom can in principle be one-way pathognomonic, meaning that it is a perfect *inclusion test* (the sign or symptom's presence always indicates the presence of the disorder) or two-way pathognomonic, meaning that it is both a perfect inclusion test and *exclusion test* (the sign or symptom's presence always indicates the presence of the disorder, and the sign or symptom's absence always indicates the absence of the disorder). With the possible exception of organic brain disorders, no *DSM* diagnoses boast a one-way pathognomonic indicator.

DSM-III-R and *DSM-IV* also witnessed a relaxation of many, though not all, of *DSM-III*'s hierarchical exclusion rules (Pincus, Tew, & First, 2004). This change largely reflected the paucity of research evidence concerning the causal primacy of certain disorders above others. In addition, many of these exclusion rules proved difficult to apply in practice, because they required subjective and highly inferential judgments of causal primacy on the part of diagnosticians.

Finally, *DSM-IV* added an appendix for *culture-bound syndromes*, recognizing the fact that some conditions vary, or at least vary markedly in their expression, across cultures (Draguns & Tanaka-Matsumi, 2003). Most of these culture-bound syndromes are widely known in non-Western cultures, although their etiology and relation to conditions diagnosed in Western cultures are poorly understood. For example, *koro*, an epidemic condition observed in parts of China and Malaysia, is marked by abrupt and intense fears that the penis (in men) or vulva or breasts (in women) are receding into the body. Still other culture-bound syndromes appear to be variants of diagnoses that we readily recognize in Western culture. For example, *taijin kyofusho*, common in

Japan, refers to a fear of offending others by one's appearance, body odor, nonverbal behavior, and so on. It may be a subspecies of social phobia that is especially prevalent in cultures, especially in Asia, that stress group harmony above individual autonomy (Kleinknecht, Dinnel, Tanouye-Wilson, & Lonner, 1994).

Criticisms of the Current Classification System

Recent versions of the diagnostic manual have helped to place the field of psychopathology on firmer scientific ground, largely because they have established reasonably reliable operationalizations for most mental disorders and furthered the development of standardized instruments, such as structured psychiatric interviews, to assess these disorders. The theoretical agnosticism of recent *DSMs* has also facilitated research comparing the scientific support for competing theoretical conceptualizations of psychopathology (Wakefield, 1998). Despite the undeniable advances of *DSM-III* and its progeny, many critics have charged that these manuals are scientifically problematic in several respects. Here we examine five key criticisms of the current classification system: comorbidity, proliferation of diagnoses, neglect of the attenuation paradox, adoption of a categorical model, and a scientifically unsupported distinction between Axis I and Axis II.²

COMORBIDITY

DSM-III and its revisions are marked by high levels of co-occurrence and covariation among many of its diagnostic categories, a phenomenon known, perhaps misleadingly, as *comorbidity* (Caron & Rutter, 1991; Lilienfeld et al., 1994; Pincus et al., 2004). We say misleadingly because it is premature in most cases to assume that comorbidity reflects the overlap among etiologically distinct conditions, as opposed to slightly different variants of the same underlying condition (Drake & Wallach, 2007). Although comorbidity is frequent among Axis I conditions, it is especially rampant among Axis II conditions (Widiger & Rogers, 1989). In one analysis based on multiple sites, patients who met criteria for one personality disorder met criteria for approximately two additional personality disorders, on average—with 10% meeting criteria for four or more personality disorders (Stuart et al., 1998). One patient in a research study met criteria for all ten *DSM* personality disorders (Widiger et al., 1998).

The extent of comorbidity among both Axis I and II disorders is often underestimated in routine clinical practice (Zimmerman & Mattia, 2000), in part because of a phenomenon known as *diagnostic overshadowing*. Diagnostic overshadowing refers to the tendency for a more florid disorder to draw attention away from less florid co-occurring

² One frequent criticism of the *DSM* revision process (for example, Caplan, 1995) that we do not discuss at length here is the reliance on committee consensus in settling on both the: (a) inclusion and exclusion of specific disorders from the manual, and (b) the diagnostic criteria for specific disorders, largely because we find this criticism to be without substantial merit. Although expert consensus inevitably introduces subjective and political considerations into the diagnostic revision process (Ghaemi, 2003; Kirk & Kutchins, 1992) and has almost certainly resulted in flawed decisions, it is almost surely superior to a system in which one appointed expert adjudicates scientific complex disputes without the benefit of input from other experts. As Widiger and Clark (2000) observed, “no diagnostic manual can be constructed without a group of fallible persons interpreting the results of existing research” (p. 948). To paraphrase Winston Churchill’s famous wisecrack about democracy, the *DSM* revision process is probably the worst system possible except for every other system.

disorders, thereby leading diagnosticians to either overlook them or attribute them to the more florid disorder. For example, the dramatic symptoms of Borderline Personality Disorder frequently lead clinicians to under diagnose commonly co-occurring but less salient conditions, such as Narcissistic and Dependent Personality Disorders (Garb, 1998). The genuine extent of comorbidity among personality disorders typically becomes evident only when structured and semistructured diagnostic interviews, which force assessors to inquire about all diagnostic criteria, are administered (Zimmerman & Mattia, 2000).

There are multiple potential explanations for comorbidity—some primarily substantive, others primarily methodological (see Klein & Riso, 1993, and Lilienfeld, 2003, for reviews). On the substantive front, one disorder (e.g., Generalized Anxiety Disorder) may predispose to another disorder (e.g., Dysthymic Disorder), the two disorders may mutually influence each other, or both disorders may be slightly different expressions of the same latent liability (e.g., neuroticism or negative emotionality). On the methodological front, comorbidity may result from overlapping diagnostic criteria, *clinical selection bias* (du Fort, Newman, & Bland, 1993), that is, the tendency for psychiatric patients with one disorder to seek treatment only when they develop a co-occurring disorder. In addition, comorbidity can be produced by *logical errors* (Guilford, 1936), mistakes stemming from the tendency of diagnosticians to assume that two largely unrelated conditions are correlated.

Whatever its causes, extensive comorbidity is potentially problematic for the *DSM*, because an ideal classification system yields largely mutually exclusive categories with few overlapping cases (Lilienfeld, VanValkenberg, Larntz, & Akiskal, 1986; Sullivan & Kendler, 1998). As a consequence, such comorbidity may suggest that the current classification system is attaching multiple labels to differing manifestations of the same underlying condition. Defenders of the current classification system are quick to point out that high levels of comorbidity are also prevalent in organic medicine, and often indicate that certain conditions (e.g., diabetes) increase individuals' risk for other conditions (e.g., blindness), a phenomenon that Kaplan and Feinstein (1974) termed *pathogenetic comorbidity*. Nevertheless, in stark contrast to organic medicine, in which the causal pathways contributing to pathogenetic comorbidity are often well understood, the causal pathways contributing to pathogenetic comorbidity in the domain of psychopathology remain unknown.

PROLIFERATION OF DIAGNOSES

One dramatic change from *DSM-I* to *DSM-IV* has been the massive increase in the sheer number of diagnoses. Some critics have argued that this increase reflects the tendency for successive editions of the *DSM* to expand their range of coverage into new and largely uncharted waters (Houts, 2001). Many of these novel diagnoses may be of dubious validity, often reflecting a tendency to medicalize behaviors previously thought to be merely odd or unusual (Sommers & Satel, 2005). For example, the relatively recent *DSM* diagnosis of Asperger's syndrome, often believed to be a mild form of autism, appears to be applied increasingly to children who are withdrawn, shy, or awkward (Gernsbacher, Dawson, & Goldsmith, 2005).

Nevertheless, as Wakefield (2001) noted, there is little or no evidence that *DSM* has expanded its range of coverage. Instead, the increase in the number of diagnoses across *DSMs* reflects an increased splitting of broader diagnoses into progressively narrower

subtypes. The distinction between *splitting* and *lumping* derives from biological taxonomy (Mayr, 1982) and refers to the difference between two classificatory styles: the tendency to subdivide broad and potentially heterogeneous categories into narrower and presumably more homogeneous categories (splitting) or the tendency to combine narrow and presumably more homogeneous categories into broad and potentially heterogeneous categories (lumping). For example, given evidence that Bipolar I Disorder and Bipolar II Disorder are related (although by no means identical) conditions with relatively similar family histories, laboratory correlates, prognoses, and treatment response, should we keep these diagnoses separate or combine them into a more encompassing, albeit more heterogeneous, category?

The splitting preferences of the architects of recent *DSMs* have been widely maligned (Houts, 2001). Herman van Praag (2000) even humorously diagnosed the *DSM's* predilection for splitting as the disorder of Nosologomania (also see Ghaemi, 2003). A preference for splitting is entirely defensible from the standpoint of research and nosological revision. A key point is that the relation between splitting and lumping is asymmetrical: If we begin by splitting diagnostic categories, we can always lump them later if research demonstrates that they are essentially identical according to the Robins and Guze (1970) criteria for validity. Yet, if we begin by lumping it would be more problematic to split later. As a consequence, we may overlook potentially crucial distinctions among etiologically separable subtypes that bear differing implications for treatment and prevention.

NEGLECT OF THE ATTENUATION PARADOX

Much of the impetus behind *DSM-III* was the laudable attempt to increase the reliability of psychiatric diagnosis and, thereby, place the fields of psychiatry and clinical psychology on firmer scientific footing. Nevertheless, reliability is only a means to an end, namely validity; moreover, as noted earlier, validity is limited not by reliability per se, but by its square root (Meehl, 1986). Therefore, diagnoses of even modest reliability can, in principle, achieve high levels of validity.

Ironically, efforts to achieve higher reliability, especially internal consistency, can sometimes produce decreases in validity, a phenomenon that Loewinger (1957) referred to as the *attenuation paradox* (also see Clark & Watson, 1995). This paradox can result when an investigator uses a narrowly circumscribed pool of items to capture a broad and multifaceted construct. In such a case, the measure of the construct may exhibit high internal consistency yet low validity, because it does not adequately tap the full breadth and richness of the construct.

Some authors have argued that this state of affairs occurred with several *DSM* diagnoses. Putting it a bit differently, they have suggested that *DSM-III* and its descendants sacrificed validity at the altar of reliability (Vaillant, 1984). For example, the current *DSM* diagnosis of Antisocial Personality Disorder (ASPD) is intended to assess the core interpersonal and affective features of psychopathic personality (psychopathy) delineated by Cleckley (1941), Karpman (1948), and others. Indeed, the accompanying text of *DSM-IV* even refers misleadingly to ASPD as synonymous with psychopathy (APA, 2000, p. 702). Because the developers of *DSM-III* (APA, 1980) were concerned that the personality features of psychopathy—such as guiltlessness, callousness, and self-centeredness—were difficult to assess reliably, they opted for a diagnosis emphasizing overt and easily agreed on antisocial behaviors—such as vandalism, stealing, and

physical aggression (Lilienfeld, 1994). These changes may have resulted in a diagnosis with greater internal consistency and inter-rater reliability than the more traditional construct of psychopathy (although evidence for this possibility is lacking). Nevertheless, they may have also resulted in a diagnosis with lower validity, because the *DSM* diagnosis of ASPD largely fails to assess the personality features central to psychopathy (Lykken, 1995). Indeed, accumulating evidence suggests that measures of ASPD are less valid for predicting a number of theoretically meaningful variables—including laboratory indicators—than are measures of psychopathy (Hare, 2003; also see Vaillant, 1984, for a discussion of the reliability-tradeoff in the case of the *DSM-III* diagnosis of schizophrenia).

ADOPTION OF A CATEGORICAL MODEL

Technically, the *DSM* is agnostic on the question of whether psychiatric diagnoses are truly categories in nature, or what Meehl (Meehl & Golden, 1982) termed *taxa*, as opposed to continua or dimensions. Taxa differ from normality in kind, whereas dimensions differ in degree. Pregnancy is a taxon, as a woman cannot be slightly pregnant; in contrast, height is a dimension (although certain taxonic conditions, like hormonal abnormalities, can lead to heights that differ qualitatively from the general population). The opening pages of *DSM-IV* state: “There is no assumption that each category of mental disorder is a completely discrete entity with absolute boundaries dividing it from other mental disorders or from no mental disorder” (p. xxxi). Yet at the measurement level, the *DSM* embraces an exclusively categorical model, classifying individuals as either meeting criteria for a disorder or not.

This categorical model is problematic for at least two reasons. First, there is growing evidence from *taxometric analyses* (Meehl & Golden, 1982); namely, those that allow researchers to ascertain whether a single observed distribution is decomposable into multiple independent distributions, that many or even most *DSM* diagnoses are underpinned by dimensions rather than taxa (Kendell & Jablensky, 2003), with schizophrenia and schizophrenia-spectrum disorders being notable probable exceptions (Lenzenweger & Korfine, 1992). This is particularly true for most personality disorders (Cloninger, in press; Trull & Durrett, 2005), including Antisocial Personality Disorder (Marcus, Edens, Lilienfeld, & Poythress, 2006). Even many or most Axis I disorders, such as Major Depression (Slade & Andrews, 2005) and Social Phobia (Kollman, Brown, Liverant, & Hoffman, 2006), appear to be dimensional as opposed to taxonic in structure.

Second, setting aside the ontological issue of taxonicity versus dimensionality, there is good evidence that measuring most disorders (especially personality disorders) dimensionally by using the full range of scores typically results in higher correlations with external validating variables than does measuring them categorically in an all-or-none fashion (Ullrich, Borkenau, & Marneros, 2001). Such findings are not surprising given that artificial dichotomization of variables almost always results in a loss of information and, hence, statistical power (Cohen, 1983; MacCallum, Zhang, Preacher, & Rucker, 2002).

AXIS I–AXIS II DISTINCTION

The rationale for the Axis I–Axis II distinction has never been grounded in high quality scientific evidence (Harkness & Lilienfeld, 1997). As already noted, there is increasing

evidence that some Axis I conditions, including mood and anxiety disorders, are underpinned by dimensions that (e.g., high levels of negative emotionality) may be the same that underpin many Axis II conditions. Moreover, there is no compelling evidence for a qualitative difference between Axis I and Axis II conditions.

DSM-III and its revisions have not been consistent in their handling of some Axis I and Axis II disorders (Frances, 1980). Certain Axis I conditions—such as Schizophrenia, Dysthymic Disorder, or Cyclothymic Disorder—are at least as chronic as most Axis II disorders and arguably belong on Axis II given its emphasis on trait-like stability. The *DSM*'s placement of Axis II disorders has also been inconsistent in many cases. For example, Cyclothymic Disorder appears to be a subsyndromal form of Bipolar Disorder and is placed on Axis I, yet Schizotypal Personality Disorder appears to be a subsyndromal form of Schizophrenia and is placed on Axis II. The reasons for this differential treatment of Cyclothymic Disorder and Schizotypal Personality Disorder are unclear, and they do not appear to be based primarily on scientific considerations.

The DSM: *Quo Vadis?*

In some respects, *DSM-III-R* and *DSM-IV* have been disappointments, as they have not resolved many of serious problems endemic to *DSM-III* (Ghaemi, 2003). Comorbidity in *DSM-III-R* and *DSM-IV* has, if anything, mushroomed since the dismantling of many hierarchical exclusion rules (Lilienfeld & Waldman, 2004). Some diagnostic categories (e.g., Antisocial Personality Disorder) of questionable validity remain, and the Axis I-Axis II distinction remains in place despite a conspicuous lack of compelling scientific evidence. The planning for the next edition of the American Psychiatric Association's diagnostic manual, *DSM-V*, began in 1999, with its projected publication date in 2011. *DSM-V*, to be spearheaded by David Kupfer, presents both challenges and opportunities: challenges because many conceptual and methodological quandaries regarding psychiatric diagnosis remain unresolved, and opportunities because a new manual opens the door for novel approaches to the classification of psychopathology.

With these considerations in mind, we sketch out two promising future directions for *DSM-V*: adoption of a dimensional approach and the incorporation of endophenotypic markers into psychiatric diagnosis (see Widiger & Clark, 2000, for other proposals for *DSM-V*).

A DIMENSIONAL APPROACH

The accumulating evidence for the dimensionality of many psychiatric conditions, particularly personality disorders, has led many authors to suggest replacing or at least supplementing the *DSM*—Axis-II in particular—with a set of dimensions derived from the science of personality (Widiger & Clark, 2000). The leading candidate for a dimensional model is the Five Factor Model (FFM; Goldberg, 1993), which consists of five major dimensions that have emerged repeatedly in factor analyses of omnibus (broad) measures of personality: extraversion, neuroticism, agreeableness, conscientiousness, and openness to experience (the FFM, incidentally, can easily be recalled using the water-logged mnemonics of OCEAN or CANOE). These five dimensions also contain lower-order facets that provide a fine-grained description of personality; for example,

the FFM dimension of extraversion contains facets of warmth, gregariousness, assertiveness, excitement seeking, and so on (Costa & McCrae, 1992).

The FFM may be able to accommodate variations not only in normal but also in abnormal personality (Costa & Widiger, 2001). For example, within the FFM the prototypical individual with Antisocial Personality Disorder might be described as low in most facets of agreeableness and conscientiousness, low in some facets of neuroticism (especially those relevant to anxiety), high in other facets of neuroticism (especially those relevant to hostility), and high in some facets of extraversion (especially those relevant to assertiveness and excitement seeking). The FFM has the distinct advantages of being consistent with emerging data on the dimensionality of most personality disorders, and of being widely replicated in studies using diverse methodologies. In addition, research suggests that much of the comorbidity among *DSM-IV* personality disorders can be reproduced by the patterns of correlations among FFM dimensions (Lynam & Widiger, 2001).

There are, however, at least two major obstacles confronting the implementation of a dimensional system within *DSM-V*. First, the FFM—although influential and widely used—is far from universally accepted (Block, 1995). Moreover, a number of authors have offered plausible and reasonably well supported competing models of the structure of personality (Cloninger, in press; Eysenck & Eysenck, 1975; Livesley, 2003; Tellegen, 1982). For example, Tellegen (1982) has proposed a three-dimensional model of personality encompassing *positive emotionality* (the enduring propensity to experience pleasant affects of many kinds, including cheerfulness, social intimacy, and achievement striving), *negative emotionality* (the enduring propensity to experience unpleasant affects of many kinds, including anxiety, hostility, and mistrust), and *constraint* (response inhibition and impulse control). The differences across dimensional systems may not be an insurmountable problem, however, because some authors have noted significant correspondences among competing models of personality structure (Watson, Clark, & Harkness, 1994). For example, Tellegen's "Big Three" Model maps nicely onto much of the FFM, with positive emotionality largely subsuming FFM extraversion, negative emotionality largely subsuming FFM neuroticism and some features of reversed agreeableness, and constraint largely subsuming FFM conscientiousness and reversed openness to experience (Church, 1994).

A second and perhaps more serious objection to a purely dimensional approach derives from the often neglected distinction between *basic tendencies* and *characteristic adaptations* in personality psychology (Harkness & Lilienfeld, 1997; McCrae & Costa, 1995). Basic tendencies are core personality traits, whereas characteristic adaptations are the behavioral manifestations of these traits. A large body of personality research suggests that basic tendencies can often be expressed in a wide variety of different characteristic adaptations depending on the upbringing, interests, cognitive skills, and other personality traits of the individual. For example, the scores of firemen on a well-validated measure of the personality trait of sensation seeking (a construct closely related to, although broader than, risk-taking) are significantly higher than those of college students, but comparable to those of incarcerated prisoners (Zuckerman, 1994). This finding dovetails with the notion that the same basic tendency, in this case sensation seeking, can be expressed in either socially constructive or destructive outlets depending on yet unidentified moderating influences.

The distinction between basic tendencies versus characteristic adaptations implies that personality dimensions, such as those from the FFM, may never be sufficient to

capture the full variance in personality disorders, because these dimensions (basic tendencies) do not adequately assess many key aspects of psychopathological functioning, many of which can be viewed as maladaptive characteristic adaptations (Sheets & Craighead, 2007). This theoretical conjecture is corroborated by findings that the FFM dimensions do not account for a sizeable chunk of variance in many *DSM-IV* personality disorders. For example, in one study the correlations between FFM prototype scores of *DSM-IV* personality disorders (derived from expert ratings of the FFM facets most closely associated with each disorder) and structured interview-based measures of these disorders were high for some disorders (e.g., Avoidant Personality Disorder; $r = .67$) and modest and even negligible for others (e.g., Obsessive-Compulsive Disorder; $r = .13$; Miller, Reynolds, & Plikonis, 2004). The lattermost finding may reflect the fact that some obsessive-compulsive traits—such as perfectionism—may be adaptive in certain settings and, therefore, may not lead inevitably to personality pathology. Moreover, Skodol et al. (2005) reported that the dimensions of the Schedule for Nonadaptive and Adaptive Personality (SNAP; Clark, 1993)—a measure that assesses many pathological behaviors associated with personality disorders—displayed incremental validity above and beyond the FFM dimensions in distinguishing among *DSM-IV* personality disorders (also see Reynolds & Clark, 2001). This finding suggests that the FFM overlooks crucial distinctions captured by the SNAP, perhaps in part because the SNAP assesses not only basic tendencies but also the maladaptive characteristic adaptations of many personality disorders (Lilienfeld, 2005).

The findings reviewed here imply that a dimensional model may be useful in capturing core features of many *DSM* personality disorders. Nevertheless, they raise the possibility that personality dimensions, including the FFM, may not be sufficient by themselves to capture personality pathology, because they cannot tell us whether individuals' behavioral adaptations to these dimensions were adaptive or maladaptive, nor the phenotypic (behavioral) manifestations these adaptations have assumed.

ENDOPHENOTYPIC MARKERS

As noted earlier, considerable recent interest has focused on the use of endophenotypes in the validation of psychiatric diagnoses (Andreasen, 1995; Waldman, 2005). Nevertheless, endophenotypic markers have thus far been excluded from *DSM* diagnostic criterion sets, which consist entirely of the classical signs and symptoms of disorders (exophenotypes). This omission is noteworthy, because endophenotypes may lie closer to the etiology of many disorders than exophenotypes.

This situation may change in coming years with accumulating evidence from studies of biochemistry, brain imaging, and performance on laboratory tasks, which hold the promise of identifying more valid markers of certain mental disorders (Widiger & Clark, 2000). To take just two examples, many impulse control disorders (e.g., Pathological Gambling, Intermittent Explosive Disorder) appear to be associated with low levels of serotonin metabolites (Moeller, Barratt, Dougherty, Schmitz, & Swann, 2001) and major depression is frequently associated with left frontal hypoactivation (Henriques & Davidson, 1991).

Nevertheless, at least two potential obstacles confront the use of endophenotypic markers in psychiatric diagnosis, the first conceptual and the second empirical. First, the widespread assumption, that endophenotypic markers are more closely linked to underlying etiological processes than exophenotypic markers (Kihlstrom, 2002), is just

that, an assumption. For example, the well replicated finding that diminished amplitude of the P300 (an brain event-related potential appearing approximately 300 milliseconds following stimulus onset) is dependably associated with externalizing disorders—such as Conduct Disorder and drug dependence (Patrick et al., 2006)—could reflect the fact that P300 is merely a sensitive indicator of attention. As a consequence, diminished P300 amplitude could be a downstream consequence of the inattention and low levels of motivation often associated with externalizing disorders. This possibility would not necessarily negate the incorporation of P300 amplitude into diagnostic criterion sets, although it could raise questions concerning its specificity for externalizing disorders, let alone specific externalizing disorders.

Second, no endophenotypic markers, yet, identified are close to serving as inclusion tests for their respective disorders. Even smooth pursuit eye movement dysfunction, which is perhaps the most dependable biological marker of schizophrenia, is only present in anywhere from 40% to 80% of patients with schizophrenia, so it would miss many individuals with the disorder. It may come closer, however, to serving as a good exclusion test, as it is present in only about 10% of normal individuals (Clementz & Sweeney, 1990; Keri & Janka, 2004). Thus, although endophenotypic markers may eventually add to the predictive efficiency of some diagnostic criteria assets, they are likely to be fallible indicators, just like traditional signs and symptoms. These markers also sustain the hope of assisting in the identification of more etiologically pure subtypes of disorders; for example, schizophrenia patients with abnormal smooth pursuit eye movements may prove to be separable in important ways from other schizophrenia patients.

Summary and Future Directions

We conclude the chapter with ten take-home messages:

1. A systematic system of psychiatric classification is a prerequisite for psychiatric diagnosis.
2. Psychiatric diagnoses serve important, even essential, communicative functions.
3. A valid psychiatric diagnosis gives us new information—for example, it tells about the diagnosed individuals' probable family history, performance on laboratory tests, natural history, and perhaps response to treatment—and it also distinguishes that person's diagnosis from other, related diagnoses.
4. The claim that mental illness is a myth rests on a misunderstanding of the role of lesions in medical disorders.
5. Prevalent claims to the contrary, psychiatric diagnoses often achieve adequate levels of reliability and validity, and do not typically pigeon-hole or stigmatize individuals when correctly applied.
6. There is no clear consensus on the correct definition of mental disorder, and some authors have suggested that the higher-order concept of mental disorder is intrinsically undefinable. Even if true, this should have no effect on the scientific investigation, assessment, or treatment of specific mental disorders (e.g., Schizophrenia, Panic Disorder), which undeniably exist.

7. Early versions of the diagnostic manual (*DSM-I* and *DSM-II*) were problematic because they provided clinicians and researchers with minimal guidance for establishing diagnoses and required high levels of subjective judgment and clinical inference.
8. *DSM-III*, which appeared in 1980, helped to alleviate this problem by providing diagnosticians with explicit diagnostic criteria, algorithms (decision-rules), and hierarchical exclusion criteria, leading to increases in the reliability of many psychiatric diagnoses.
9. The current classification system, *DSM-IV*, is a clear advance over *DSM-I* and *DSM-II*. Nevertheless, *DSM-IV* continues to be plagued by a variety of problems, especially extensive comorbidity, reliable diagnoses that are of questionable validity, adoption of a categorical model in the absence of compelling evidence, and a largely arbitrary distinction between Axis I (major mental disorders) and Axis II (mental retardation and personality disorders).
10. Fruitful potential directions for *DSM-V* include a dimensional model of personality to replace or supplement the existing categorical system of Axis II, and the incorporation of endophenotypic markers into the diagnostic criteria for some disorders.

References

- Amador, X. F., & Paul-Ouduard, R. (2000). Defending the unabomber: Anosognosia in schizophrenia. *Psychiatric Quarterly*, 71, 363–371.
- American Psychiatric Association. (1952). *Diagnostic and statistical manual of mental disorders*. Washington, DC: Author.
- American Psychiatric Association. (1968). *Diagnostic and statistical manual of mental disorders* (2nd ed.). Washington, DC: Author.
- American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.
- American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., rev). Washington, DC: Author.
- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (Text rev.). Washington, DC: Author.
- Andreasen, N. C. (1995). The validation of psychiatric diagnosis: New models and approaches. *American Journal of Psychiatry*, 152, 161–162.
- Barlow, D. H. (2001). *Anxiety and its disorders: The nature and treatment of anxiety and panic* (2nd ed.). New York: Guilford.
- Bayer R., & Spitzer, R. L. (1982). Edited correspondence on the status of homosexuality in *DSM-III*. *Journal of the History of the Behavioral Sciences*, 18, 32–52.
- Benton, A. L. (1992). Gerstmann's syndrome. *Archives of Neurology*, 49, 445–447.
- Blashfield, R., & Burgess, D. (2007). Classification provides an essential basis for organizing mental disorders. In S. O. Lilienfeld & W. T. O'Donohue (Eds.), *The great ideas of clinical science: 17 principles that every mental professional should understand* (pp. 93–118). New York: Routledge.
- Block, J. (1995). A contrarian view of the five-factor approach to personality description. *Psychological Bulletin*, 117, 187–215.
- Brown, M. (2006, August 16). War of the worlds. *New York Times*, p. 17.
- Caron, C., & Rutter, M. (1991). Comorbidity in child psychopathology: Concepts, issues and research strategies. *Journal of Child Psychology and Psychiatry*, 32, 1063–1080.
- Clark, L. A. (1993). *Manual for the schedule for non-adaptive and adaptive personality (SNAP)*. Minneapolis: University of Minnesota Press.
- Clark, L. A., & Watson, D. (1995). Constructing validity: Basic issues in objective scale development. *Psychological Assessment*, 7, 309–319.
- Cleckley, H. (1941). *The mask of sanity*. St. Louis, MO: Mosby.
- Clementz, B. A., & Sweeney, J. A. (1990). Is eye movement dysfunction a biological marker for schizophrenia? A methodological review. *Psychological Bulletin*, 108, 77–92.
- Cloninger, C. R. (2007). Foreward. In W.O'Donohue, K. A. Fowler, & S. O. Lilienfeld, (Eds.), *Personality disorders: Toward the DSM-V* (pp. vii–xvi). Thousand Oaks, CA: Sage.
- Cohen, H. (1981). The evolution of the concept of disease. In A. L. Caplan, H. T. Engelhardt, Jr., & J. J. McCartney (Eds.), *Concepts of health and disease: Interdisciplinary perspectives* (pp. 209–220). Reading, MA: Addison-Wesley.
- Cohen, J. (1983). The cost of dichotomization. *Applied Psychological Measurement*, 7, 249–253.
- Compton, W. M. & Guze, S. B. (1995). The neoKraepelinian revolution in psychiatric diagnosis. *European Archives of Psychiatry and Clinical Neuroscience*, 245, 196–201.
- Cooper, G. (2007). Medical errors with psychiatric patients. *Psychotherapy Networker*, 31(3), 17–18.
- Cornez-Ruiz, S., & Hendricks, B. (1993). Effects of labeling and ADHD behaviors on peer and teacher judgments. *Journal of Educational Research*, 86, 349–355.
- Costa, P. T., Jr., & McCrae, R. R. (1992). Normal personality assessment in clinical practice: The NEO personality inventory. *Psychological Assessment*, 4, 5–13.
- Costa, P. T., & Widiger, T. A. (2001). *Personality disorders and the five-factor model of personality* (2nd ed.). Washington, DC: American Psychological Association.
- Cronbach, L. J. (1951). Coefficient alpha and the internal structure of tests. *Psychometrika*, 16, 297–335.

- Cronbach, L. J., & Meehl, P. E. (1955). Construct validity in psychological tests. *Psychological Bulletin*, 52, 281–302.
- Draguns, J. G., & Tanaka-Matsumi, J. (2003). Assessment of psychopathology across and within cultures: Issues and findings. *Behaviour Research and Therapy*, 41, 755–776.
- Drake, R. E., & Wallach, M. A. (2007). Is comorbidity a psychological science? *Clinical Psychology: Science and Practice*, 14, 20–22.
- Du Fort, G. G., Newman, S. C., & Bland, R. C. (1993). Psychiatric comorbidity and treatment seeking: Sources of selection bias in the study of clinical populations. *Journal of Nervous and Mental Disease*, 181, 467–474.
- Eysenck, H. J., & Eysenck, S. B. G. (1975). *Manual of the Eysenck personality questionnaire* (adult and junior). London: Hodder and Stoughton.
- Eysenck, H. J., Wakefield, J., & Friedman, A. (1983). Diagnosis and clinical assessment: The *DSM-III*. *Annual Review of Psychology*, 34, 167–193.
- Faust, D., & Miner, R. A. (1986). The empiricist in his new clothes: *DSM-III* in perspective. *American Journal of Psychiatry*, 143, 962–967.
- Feighner, J., Robins, E., Guze, S., Woodruff, R., Winkler, G., & Munoz, R. (1972). Diagnostic criteria for use in psychiatric research. *Archives of General Psychiatry*, 26, 57–63.
- First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (2002). *Structured clinical interview for DSM-IV-TR axis I disorders, research version, patient edition*. New York: Biometrics Research, New York State Psychiatric Institute.
- Frances, A. J. (1980). The *DSM-III* personality disorders section: A commentary. *American Journal of Psychiatry*, 137, 1050–1054.
- Garb, H. N. (1998). *Studying the clinician: Judgment research and psychological assessment*. Washington, DC: American Psychological Association.
- Garber, J., & Strassberg, Z. (1991). Construct validity: History and application to developmental psychopathology. In W. M. Grove & D. Cicchetti (Eds.), *Personality and psychopathology* (pp. 218–258). Minneapolis: University of Minnesota Press.
- Gernsbacher, M. A., Dawson, M., & Goldsmith, H. H. (2005). Three reasons not to believe in an autism epidemic. *Current Directions in Psychological Science*, 14, 55–58.
- Ghaemi, N. (2003). *The concepts of psychiatry: A pluralistic approach to the mind and mental illness*. Baltimore, MD: Johns Hopkins University Press.
- Goldberg, L. R. (1993). The structure of phenotypic personality traits. *American Psychologist*, 48, 266–275.
- Goodwin, D. W., & Guze, S. B. (1996). *Psychiatric diagnosis* (5th ed.) New York: Oxford University Press.
- Gorenstein, E. E. (1992). *The science of mental illness*. San Diego, CA: Academic Press.
- Gottesman, I. I., & Gould, T. D. (2003). The endophenotype concept in psychiatry: Etymology and strategic intentions. *American Journal of Psychiatry*, 160, 636–645.
- Gough, H. (1971). Some reflections on the meaning of psychodiagnosis. *American Psychologist*, 26, 160–167.
- Grob, G. N. (1991). Origins of *DSM-I*: A study in appearance and reality. *American Journal of Psychiatry*, 148, 421–431.
- Guilford, J. P. (1936). *Psychometric methods*. New York: McGraw-Hill.
- Harkness, A. R., & Lilienfeld, S. O. (1997). Individual differences science for treatment planning: Personality traits. *Psychological Assessment*, 9, 349–360.
- Harris, M. J., Milich, R., Corbitt, E. M., Hoover, D. W., & Brady, M. (1992). Self-fulfilling effects of stigmatizing information on children's social interactions. *Journal of Personality and Social Psychology*, 63, 41–50.
- Henriques, J. B., & Davidson, R. J. (1991). Left frontal hypoactivation in depression. *Journal of Abnormal Psychology*, 100, 535–545.
- Houts, A. C. (2001). The diagnostic and statistical manual's new white coat and circularity of plausible dysfunctions: Response to Wakefield, part 1. *Behavior Research and Therapy*, 39, 315–345.
- Joiner, T. (2006). *Why people die by suicide*. Cambridge, MA: Harvard University Press.
- Kaplan, M. H., & Feinstein, A. R. (1974). The importance of classifying initial co-morbidity in evaluating the outcome of diabetes mellitus. *Journal of Chronic Diseases*, 27, 387–404.
- Karpman, B. (1948). The myth of the psychopathic personality. *American Journal of Psychiatry*, 104, 523–524.
- Kazdin, A. E. (1983). Psychiatric diagnosis, dimensions of dysfunction, and child behavior therapy. *Behavior Therapy*, 14, 73–99.
- Kendell, R. E. (1975). The concept of disease and its implications for psychiatry. *British Journal of Psychiatry*, 127, 305–315.
- Kendell, R., & Jablensky, A. (2003). Distinguishing between the validity and utility of psychiatric diagnoses. *American Journal of Psychiatry*, 160, 4–12.

- Kendler, K. S. (1980). The nosologic validity of paranoia (simple delusional disorder): A review. *Archives of General Psychiatry*, 37, 699–706.
- Keri, S., & Janka, Z. (2004). Critical evaluation of cognitive dysfunctions as endophenotypes of schizophrenia. *Acta Psychiatrica Scandinavica*, 110, 83–91.
- Kihlstrom, J. F. (2002). To honor Kraepelin . . . : From symptoms to pathology in the diagnosis of mental illness. In L. Beutler & M. Malik (Eds.), *Rethinking the DSM: A psychological perspective* (pp. 279–303). Washington, DC: American Psychological Association.
- Kirk, S. A., & Kutchins, H. (1992). *The selling of DSM: The rhetoric of science in psychiatry*. New York: Aldine de Gruy.
- Klein, D., & Riso, L. P. (1993). Psychiatric disorders: Problems of boundaries and comorbidity. In C. G. Costello (Ed.), *Basic issues in psychopathology* (pp. 19–66). New York: Guilford.
- Kleinknecht, R. A., Dinnel, D. L., Tanouye-Wilson, S., & Lonner, W. J. (1994). Cultural variation in social anxiety and phobia: A study of *Taijin Kyofusho*. *The Behavioral Therapist*, 17(8), 175–178.
- Klerman, G. (1984). The advantages of DSM-III. *American Journal of Psychiatry*, 141, 539–542.
- Kollman, D. M., Brown, T. A., Liverant, G. I., & Hofmann, S. G. (2006). A taxometric investigation of the latent structure of social anxiety disorder in outpatients with anxiety and mood disorders. *Depression and Anxiety*, 23, 190–199.
- Kraupl Taylor, F. (1971). A logical analysis of medico-physiological concept of disease. *Psychological Medicine*, 1, 356–364.
- Lenzenweger, M. F., & Korfine, L. (1992). Confirming the latent structure and base rate of schizotypy: A taxometric analysis. *Journal of Abnormal Psychology*, 101, 567–571.
- Lief, A. A. (Ed.). (1948). *The commonsense psychiatry of Dr. Adolf Meyer: Fifty-two selected papers*. New York: McGraw-Hill.
- Lilienfeld, S. O. (1994). Conceptual problems in the assessment of psychopathy. *Clinical Psychology Review*, 14, 17–38.
- Lilienfeld, S. O. (1995). *Seeing both sides: Classic controversies in abnormal psychology*. Pacific Grove, CA: Brooks/Cole.
- Lilienfeld, S. O. (2003). Comorbidity between and within childhood externalizing and internalizing disorders: Reflections and directions. *Journal of Abnormal Child Psychology*, 31, 285–291.
- Lilienfeld, S. O. (2005). Longitudinal studies of personality disorders: Four lessons from personality psychology. *Journal of Personality Disorders*, 19, 547–556.
- Lilienfeld, S. O., & Marino, L. (1995). Mental disorder as a Rochian concept: A critique of Wakefield's "harmful dysfunction" analysis. *Journal of Abnormal Psychology*, 104, 411–420.
- Lilienfeld, S. O., & Marino, L. (1999). Essentialism revisited: Evolutionary theory and the concept of a mental disorder. *Journal of Abnormal Psychology*, 108, 400–411.
- Lilienfeld, S. O., VanValkenburg, C., Larntz, K., & Akiskal, H. S. (1986). The relationship of histrionic personality to antisocial personality and somatization disorders. *American Journal of Psychiatry*, 143, 718–722.
- Lilienfeld, S. O., Waldman, I. D., & Israel, A. C. (1994). A critical note on the use of the term and concept of "comorbidity" in psychopathology research. *Clinical Psychology: Science and Practice*, 1, 71–83.
- Link, B. G., & Cullen, F. T. (1990). The labeling theory of mental disorder: A review of the evidence. *Research in Community and Mental Health*, 6, 75–105.
- Livesley, W. J. (2003). Diagnostic dilemmas in the classification of personality disorder. In K. A. Phillips, M. B. First, & H. A. Pincus (Eds.), *Advancing the DSM: Dilemmas in psychiatric diagnosis* (pp. 153–189). Washington, DC: American Psychiatric Association.
- Loevinger, J. (1957). Objective tests as instruments of psychological theory. *Psychological Reports*, 3, 635–694.
- Lykken, D. T. (1995). *The antisocial personalities*. Hillsdale, NJ: Erlbaum.
- Lynam, D. R., & Widiger, T. A. (2001). Using the five-factor model to represent the *DSM-IV* personality disorders: An expert consensus approach. *Journal of Abnormal Psychology*, 110, 401–412.
- MacCallum, R. C., Zhang, S., Preacher, K. J., & Rucker, D. D. (2002). On the practice of dichotomization of quantitative variables. *Psychological Methods*, 7, 19–40.
- Marcus, D. K., Lilienfeld, S. O., Edens, J. F., & Poythress, N. G. (2006). Is antisocial personality disorder continuous or categorical? A taxometric analysis. *Psychological Medicine*, 36, 1571–1581.
- Matarazzo, J. D. (1983). The reliability of psychiatric and psychological diagnosis. *Clinical Psychology Review*, 3, 103–145.
- Mayes, R., & Horwitz, A. V. (2005). *DSM-III* and the revolution in the classification of mental illness.

- Journal of the History of the Behavioral Sciences*, 41, 249–267.
- Mayr, E. (1982). *The growth of biological thought: Diversity, evolution, and inheritance*. Cambridge, MA: Belknap Press.
- McCann, J. T., Shindler, K. L., & Hammond, T. R. (2003). The science and pseudoscience of expert testimony. In S. O. Lilienfeld, J. M. Lohr, & S. J. Lynn (Eds.), *Science and pseudoscience in contemporary clinical psychology* (pp. 77–108). New York: Guilford.
- McCrae, R. R., & Costa, P. T. (1995). Trait explanations in personality psychology. *European Journal of Personality*, 9, 231–252.
- McHugh, P. R., & Slavney, P. R. (1998). *The perspectives of psychiatry* (2nd ed.). Baltimore: Johns Hopkins University Press.
- Meehl, P. E. (1973). Why I do not attend case conferences. In P. E. Meehl (Ed.), *Psychodiagnosis: Selected papers* (pp. 225–302). Minneapolis: University of Minnesota Press.
- Meehl, P. E. (1977). Specific etiology and other forms of strong influence: Some quantitative meanings. *Journal of Medicine and Philosophy*, 2, 33–53.
- Meehl, P. E. (1986). Diagnostic taxa as open concepts: Metatheoretical and statistical questions about reliability and construct validity in the grand strategy of nosological revision. In T. Millon & G. L. Klerman (Eds.), *Contemporary directions in psychopathology: Toward the DSM-IV* (pp. 215–231). New York: Guilford.
- Meehl, P. E. (1990). Schizotaxia as an open concept. In A. I. Rabin, R. Zucker, R. Emmons, & S. Frank (Eds.), *Studying persons and lives* (pp. 248–303). New York: Springer.
- Meehl, P. E., & Golden, R. (1982). Taxometric methods. In P. C. Kendall & J. N. Butcher (Eds.), *Handbook of research methods in clinical psychology* (pp. 127–181). New York: Wiley.
- Messick, S. (1995). Validity of psychological assessment: Validation of inferences from persons' responses and performances as scientific inquiry into score meaning. *American Psychologist*, 50, 741–749.
- Meyer, G. J. (1997). Assessing reliability: Critical corrections for a critical examination of the Rorschach Comprehensive System. *Psychological Assessment*, 9, 480–489.
- Michels, R. (1984). A debate on *DSM-III*: First rebuttal. *American Journal of Psychiatry*, 141, 548–553.
- Milich, R., McAninich, C. B., & Harris, M. J. (1992). Effects of stigmatizing information on children's peer relations: Believing is seeing *School Psychology Review*, 21, 399–408.
- Miller, J. D., Reynolds, S. K., & Pilkonis, P. A. (2004). The validity of the five-factor model prototypes for personality disorders in two clinical samples. *Psychological Assessment*, 16, 310–322.
- Millon, T. (1975). Reflections on Rosenhan's "On being sane in insane places." *Journal of Abnormal Psychology*, 84, 456–461.
- Moeller, F. G., Barratt, E. S., Dougherty, D. M., Schmitz, J. M., & Swann, A. C. (2001). Psychiatric aspects of impulsivity. *American Journal of Psychiatry*, 158, 1783–1793.
- Morey, L. C. (1991). Classification of mental disorders as a collection of hypothetical constructs. *Journal of Abnormal Psychology*, 100, 289–293.
- Morrison, J. (1997). *When psychological problems mask medical disorders: A guide for psychotherapists*. New York: Guilford.
- Neese, R., & Williams, G. (1994). *Why we get sick*. New York: Vintage.
- Patrick, C. J., Bernat, E., Malone, S. M., Iacono, W. G., Krueger, R. F., & McGue, M. K. (2006). P300 amplitude as an indicator of externalizing in adolescent males. *Psychophysiology*, 43, 84–92.
- Pelham, W. E., & Bender, M. E. (1982). Peer relationships in hyperactive children: Description and treatment. In K. D. Gadow & I. Bialer (Eds.), *Advances in learning and behavioral disabilities* (Vol. 1, pp. 365–436). Greenwich, CT: JAI Press.
- Pincus, H. A., Tew, J. D., & First, M. B. (2004). Psychiatric comorbidity: Is more less? *World Psychiatry*, 3, 18–23.
- Pouissant, A. F. (2002). Is extreme racism a mental illness? Point-counterpoint. *Western Journal of Medicine*, 176, 4.
- Reynolds, S. K., & Clark, L. A. (2001). Predicting personality disorder dimensions from domains and facets of the five-factor model. *Journal of Personality*, 69, 199–222.
- Robins, E., & Guze, S. B. (1970). Establishment of diagnostic validity in psychiatric illness: Its application to schizophrenia. *American Journal of Psychiatry*, 126, 983–987.
- Rosch, E. R. (1973). Natural categories. *Cognitive Psychology*, 4, 328–350.
- Rosch, E. R., & Mervis, C. B. (1975). Family resemblances: Studies in the internal structure of categories. *Cognitive Psychology*, 7, 573–605.
- Rosenhan, D. L. (1973). On being sane in insane places. *Science*, 179, 250–258.
- Ross, C., & Pam, A. (1996). *Pseudoscience in biological psychiatry: Blaming the body*. New York: Wiley.

- Ruscio, J. (2004). Diagnoses and the behaviors they denote: A critical evaluation of the labeling theory of mental illness. *Scientific Review of Mental Health Practice*, 3(1), 5–22.
- Sarbin, T. R. (1969). On the distinction between social roles and social types, with special reference to the hippie. *American Journal of Psychiatry*, 125, 1024–1031.
- Scadding, J. G. (1996). Essentialism and nominalism in medicine: Logic of diagnosis in disease terminology. *Lancet*, 348, 594–596.
- Schaler, J. A. (Ed.). (2004). *Szasz under fire: The psychiatric abolitionist faces his critics*. Chicago: Open Court.
- Scheff, T. (Ed.). (1975). *Labeling madness*. New Jersey: Prentice Hall.
- Schmidt, F. L., Lee, H., & Ilies, R. (2003). Beyond Alpha: An empirical examination of the effects of different sources of measurement error on reliability estimates for measures of individual differences constructs. *Psychological Methods*, 8, 206–224.
- Seitz, S., & Geske, D. (1976). Mothers' and graduate trainees' judgments of children: Some effects of labeling. *American Journal of Mental Deficiency*, 81, 362–370.
- Selkoe, D. J. (1992). Aging brain, aging mind. *Scientific American*, 267, 134–142.
- Sheets, E., & Craighhead, W. E. (2007). Toward an empirically based classification of personality pathology. *Clinical Psychology: Science and Practice*, 14, 77–93.
- Skinner, H. A. (1981). Toward the integration of classification theory and methods. *Journal of Abnormal Psychology*, 90, 68–87.
- Skinner, H. A. (1986). Construct validation approach to psychiatric classification. In T. Millon & G. L. Klerman (Eds.), *Contemporary directions in psychopathology: Toward the DSM-IV* (pp. 307–330). New York: Guilford.
- Slade, T., & Andrews, G. (2005). Latent structure of depression in a community sample: A taxometric analysis. *Psychological Medicine*, 35, 489–497.
- Slater, L. (2004). *Opening Skinner's box: Great psychological experiments of the 20th century*. New York: W.W. Norton.
- Sommers, C. H., & Satel, S. (2005). *One nation under therapy: How the helping culture is eroding self-reliance*. New York: St. Martin's.
- Spitzer, R. L. (1975). On pseudoscience, logic in remission, and psychiatric diagnosis: A critique of Rosenhan's "On being sane in insane places." *Journal of Abnormal Psychology*, 84, 442–452.
- Spitzer, R. L., Foreman, J. B. W., and Nee, J. (1979). DSM-III field trials. *American Journal of Psychiatry*, 136, 815–820.
- Stuart, S., Pfohl, B., Battaglia, M., Bellodi, L., Grove, W., & Cadoret, R. (1998). The cooccurrence of DSM-III-R personality disorders. *Journal of Personality Disorders*, 12, 302–315.
- Sullivan, P. F., & Kendler, K. S. (1998). The genetic epidemiology of smoking. *Nicotine and Tobacco Research*, 1, S51–S57.
- Sutton, E. H. (1980). *An introduction to human genetics*. Philadelphia: Saunders College.
- Szasz, T. (1960). The myth of mental illness. *American Psychologist*, 15, 113–118.
- Tellegen, A. (1982). *Manual for the Multidimensional Personality Questionnaire*. Unpublished manuscript, University of Minnesota.
- Trull, T. J., & Durett, C. A. (2005). Categorical and dimensional models of personality disorder. *Annual Review of Clinical Psychology*, 1, 355–380.
- Ullrich, S., Borkenau, P., & Marneros, A. (2001). Personality disorders in offenders: Categorical versus dimensional approaches. *Journal of Personality Disorders*, 15, 442–449.
- VanPraag, H. M. (2000). Nosologomania: A disorder of psychiatry. *World of Biological Psychiatry*, 1, 151–158.
- Wakefield, J. C. (1992). The concept of mental disorder: On the boundary between biological facts and social values. *American Psychologist*, 47, 373–388.
- Wakefield, J. C. (1998). The DSM's theory-neutral nosology is scientifically progressive: Response to Follette and Houts. *Journal of Consulting and Clinical Psychology*, 66, 846–852.
- Wakefield, J. C. (1999). Evolutionary versus prototype analyses of the concept of disorder. *Journal of Abnormal Psychology*, 108, 374–399.
- Wakefield, J. C. (2001). The myth of DSM's invention of new categories of disorder: Houts's diagnostic discontinuity thesis disconfirmed. *Behaviour Research and Therapy*, 39, 575–624.
- Waldman, I. D. (2005). Statistical approaches to complex phenotypes: Evaluating neuropsychological endophenotypes of attention-deficit/hyperactivity disorder. *Biological Psychiatry*, 57, 1347–1356.
- Waldman, I. D., Lilienfeld, S. O., & Lahey, B. B. (1995). Toward construct validity in the childhood disruptive behavior disorders: Classification and diagnosis in DSM-IV and beyond. In T. H. Ollendick & R. J. Prinz (Eds.), *Advances in clinical child*

- psychology* (Vol. 17, pp. 323–363). New York: Plenum.
- Watson, D., Clark, L. A., & Harkness, A. R. (1994). Structures of personality and their relevance to psychopathology. *Journal of Abnormal Psychology, 103*, 18–31.
- Widiger, T. A. (1997). The construct of mental disorder. *Clinical Psychology: Science and Practice, 4*, 262–266.
- Widiger, T. A. (2007). Alternatives to DSM-IV: Axis II. In W. O'Donohue, K. A. Fowler, & S. O. Lilienfeld (Eds.), *Personality disorders: Toward the DSM-V* (pp. 21–40). Thousand Oaks, CA: Sage.
- Widiger, T. A., & Clark, L. A. (2000). Toward *DSM-V* and the classification of psychopathology. *Psychological Bulletin, 126*, 946–963.
- Widiger, T. A., Frances, A. J., Pincus, H. A., Ross, R., First, M. B., & Davis, W. W. (Eds.). (1998). *DSM-IV sourcebook* (Vol. 4). Washington, DC: American Psychiatric Press.
- Widiger, T. A., & Rogers, J. H. (1989). Prevalence and comorbidity of personality disorders. *Psychiatric Annals, 19*, 132–136.
- Widiger, T. A., & Trull, T. (1985). The empty debate over the existence of mental illness: Comments on Gorenstein. *American Psychologist, 40*, 468–470.
- Widiger, T. A., Frances, A. J., Spitzer, R. L., & Williams, J. B. W. (1991). The *DSM-III-R* personality disorders: An overview. *American Journal of Psychiatry, 145*, 786–795.
- Zimmerman, M. (1994). Diagnosing personality disorders. *Archives of General Psychiatry, 51*, 225–245.
- Zimmerman, M., & Mattia, J. I. (2000). Principal and additional *DSM-IV* disorders for which outpatients seek treatment. *Psychiatric Services, 51*, 1299–1304.
- Zuckerman, M. (1994). *Behavioral expressions and biosocial bases of sensation seeking*. New York: Cambridge University Press.