Diagnosis and Classification

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Glossary

Comorbidity Overlap among different psychiatric diagnoses.
Diagnosis The act of placing an individual within a category.
Disease A disorder whose pathology and etiology is well understood.
Disorder A syndrome that cannot be readily explained by other conditions.
Endophenotypes Markers of psychiatric disorders that cannot be observed using the naked eye.
Sign Observable manifestation of psychopathology.
Symptom Subjectively reported manifestations of psychopathology.
Syndrome Constellations of signs and symptoms that covary across individuals.

Introduction

A half century ago, the psychoanalyst, Menninger (1963), declared that there is no need for a system of psychiatric classification, because there is essentially only one mental illness. For Menninger, all mental disorders, despite their surface differences, were fundamentally similar, differing only in severity. Yet, despite recent factor-analytic evidence that a higher-order dimension of general psychopathology may underpin variation across a plethora of mental disorders (Caspi *et al.*, 2013), few researchers today would concur with Menninger's 'single domain' hypothesis. Among other things, Menninger's assertion seems implausible on *a priori* grounds. With its approximately 85 billion neurons and 100 trillion neuronal connections, the human brain is far and away the most complex structure in the known universe. It would be utterly remarkable if there were only one way in which human psychological systems, which are enabled by the brain, to go awry.

Indeed, most evidence suggests that the domain of psychopathology is enormously variegated and heterogeneous. For example, the current psychiatric classification system used in the United States and much of the rest of the world, the *Diagnostic and Statistical Manual of Mental Disorders*, fifth edition (DSM-5), contains over 300 diagnoses (American Psychiatric Association, 2013). Although some of these diagnoses may reflect dubious distinctions that do not capture the state of nature (Greenberg, 2013), others almost certainly reflect genuine differences within the broad domain of psychopathology. Hence, the need for a classification system to bring order to the disorder, pun intended.

Why Classify?

Psychiatric classification systems serve several crucial purposes (Blashfield and Draguns, 1976; Lilienfeld *et al.*, 2013). We examine four of these purposes here.

Enhancing Communication

Classification systems enhance accurate communication among mental health professionals, boosting the chances that when one psychologist or psychiatrist uses a term for a given condition (say, 'schizophrenia' or 'panic disorder'), this term will refer to approximately the same condition used by another psychologist or psychiatrist. A related role is what Blashfield and Burgess (2007) termed 'information retrieval.' Just as zoologists use the name of a species to summarize distinctive features of a specific animal, psychologists and psychiatrists rely on a diagnosis to summarize distinctive features of a specific mental disorder. Diagnoses succinctly convey important information about patients to clinicians, researchers, family members, managed care organizations, and patients themselves.

Relations to Other Conditions

Within classification systems, diagnostic categories are typically arranged in relation to other conditions. The more adjacent in the network two conditions are, the more closely related they ostensibly are in their etiology (causation). For example, antisocial personality disorder (ASPD) and narcissistic personality disorder (NPD) – both classified within Cluster B, the 'dramatic, emotional' group of personality disorders – are presumably more closely linked etiologically than are ASPD and dependent personality disorder, a condition that falls into Cluster C, the 'anxious, fearful' group of personality disorders. Hence, diagnoses help to place the patient's presenting problems in the context of both more and less related diagnoses.

Providing Additional Information

A valid diagnosis within a classification system helps us to learn new things; it affords us additional information that we did not previously have at our disposal. In a classic article, psychiatrists Robins and Guze (1970) outlined four criteria for ascertaining whether a diagnosis is valid – that is, whether the diagnosis measures what it purports to measure. Specifically, Robins and Guze argued that a valid psychiatric diagnosis offers information regarding:

(1) Clinical description, including the condition's presenting picture, demographics, precipitants, and differences from

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seemingly related disorders. The lattermost task of distinguishing a diagnosis from similar diagnoses is called differential diagnosis;
(2) Laboratory findings, including data from psychological, biological, and laboratory tests;
(3) Natural history, including course (the condition's pattern over time) and outcome (the condition's long-term aftermath); and
(4) Family history, especially the extent to which the condition 'runs' (aggregates) within biological families.

Some authors have proposed that a valid diagnosis should also be able to predict individuals' response to treatment (see Waldman *et al.*, 1995). Nevertheless, this criterion should not be mandatory given that the treatment of a condition bears no necessary implications for its etiology. For example, although both schizophrenia and nausea caused by food poisoning respond to psychopharmacological agents that inhibit the action of the neurotransmitter dopamine, these two conditions spring from distinct causal mechanisms (the phrase *ex juvantibus reasoning*, or reasoning backward from what works, describes the error of inferring a disorder's etiology from its treatment; see Ross and Pam, 1996).

Andreasen (1995) extended the Robins and Guze framework to incorporate findings derived from molecular genetics, neurotransmitter metabolites, and brain imaging as further validating indicators for psychiatric diagnoses (see also Kendell and Jablensky, 2003). Her amendment to the Robins and Guze criteria allows researchers to use endophenotypes to assist in the validation of diagnoses. Endophenotypes are presumably intermediate phenotypes, that is, 'measurable components unseen by the unaided eye along the pathway between disease and distal genotype' (Gottesman and Gould, 2003, p. 636; Waldman, 2005). For example, a potential endophenotype for schizophrenia might be smooth pursuit eye movement dysfunction, which characterizes a large proportion of patients with the disorder (Nkam *et al.*, 2010). Endophenotypes differ from exophenotypes, the traditional diagnostic features of a disorder, such as delusions and hallucinations in schizophrenia.

We can view the process of validating psychiatric diagnoses within the overarching framework of 'construct validity' (Cronbach and 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 constructs; thus, the process of validating psychiatric diagnoses is a matter of construct validation. More broadly, we can conceptualize most or all psychiatric diagnoses as open concepts (Meehl, 1977, 1989). Open concepts are marked by (1) fuzzy boundaries, (2) a list of indicators (e.g., diagnostic criteria) that are indefinitely extendable, and (3) an unclear inner nature.

Understanding that psychiatric diagnoses are open concepts helps us to avoid the perils of premature reification of diagnostic entities (e.g., Faust and Miner, 1986). For example, the DSM-5 criteria for schizophrenia are not isomorphic with the latent construct of schizophrenia; they are merely fallible, albeit at least partly valid, indicators of this construct. Yet, some authors commit the error of reifying and deifying the categories within the current classification system, with some appearing to regard them almost as fixed Platonic essences rather than as useful approximations to the true state of nature (see Ghaemi, 2003; Michels, 1984, for criticisms). 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, or when authors refer to certain measures as 'gold standards' of the constructs of interest (see Skeem and Cooke, 2010, for a critique).

One limitation 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; see 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 (vs. heterogeneity) and factor structure (Waldman *et al.*, 1995). For example, if analyses suggest that a diagnosis intended to be homogeneous consists of multiple and largely independent subtypes, it would be of questionable validity.

Facilitating Understanding

Finally, a crucial goal of all classification systems is to help us better understand the state of nature. In the case of psychopathology, the primary goal of classification is to reduce the substantial heterogeneity of the mental illness domain by creating more homogeneous and ideally more psychologically meaningful categories (Lilienfeld *et al.*, 2013). By doing so, psychologists and psychiatrists can strive to better identify the causes of specific mental disorders and ultimately treat and prevent these conditions. Just as in the biological sciences, in which Linnaeus' hierarchical taxonomy categorizes fauna and flora, and in chemistry, where Mendeleev's periodic table orders the elements, a psychiatric classification system organizes the bewildering varieties of abnormality into more orderly subgroupings.

Nevertheless, because classification systems attempt to 'carve nature at its joints,' to use Plato's famous phrase (Gangestad and Snyder, 1985), they begin with the assumption that there are valid joints – points of rarity – in nature to carve. Put somewhat differently, psychiatric classification systems traditionally presume that at least some mental disorders are 'taxa,' that is, genuine categories in nature (Meehl and Golden, 1982). Nevertheless, burgeoning evidence from taxometric studies, which allow researchers to determine whether a single observed distribution is underpinned by one or more distinct distributions, suggests that many and perhaps most major mental disorders, including mood and anxiety disorders and most personality disorders, are undergirded by one or more dimensions (continua) rather than taxa (Haslam *et al.*, 2012). The most persuasive evidence for taxonicity probably exists for schizophrenia and autism spectrum disorders, although even in these domains the evidence is not entirely consistent (Haslam *et al.*, 2012).

The finding that many or most mental disorders may not be taxonic in nature may help to explain why all psychiatric

classification systems past and present, including DSM-5, have fallen well short of the goal of an ideal taxonomy: to provide a system of discrete categories that are largely mutually exclusive, with few intermediate cases (Frances, 1980). Indeed, one of the findings bedeviling the DSM-5 and other systems of psychiatric classification is the high level of so-called 'comorbidity' – traditionally defined as diagnostic overlap – among diagnoses (Cramer *et al.*, 2010; Lilienfeld *et al.*, 1994). Such comorbidity is especially rampant among personality disorders. For example, patients with one DSM personality disorder on average meet diagnostic criteria for two additional personality disorders, with 10% meeting criteria for four or more personality disorders (Stuart *et al.*, 1998). One patient in a published study met diagnostic criteria for all ten DSM personality disorders (Widiger *et al.*, 1998)! The extent of such comorbidity is often underestimated in routine clinical practice because of a phenomenon known as 'diagnostic overshadowing' (Garb, 1998), in which the presence of salient and dramatic conditions (e.g., borderline personality disorder) often leads clinicians to overlook less vivid conditions (e.g., generalized anxiety disorder). When standardized diagnostic criteria are used, the full magnitude of comorbidity becomes apparent.

In light of these and other findings, such as the fact that the boundaries between related disorders are often fuzzy, numerous authors have proposed that the field should move away from a traditional classification system, which presumes the existence of latent categories, to a dimensional system, which presumes that mental normality and abnormality lie along a continuum (Eysenck *et al.*, 1983; Krueger *et al.*, 2005). Nevertheless, as we will discuss later, this alternative approach has met resistance on both scientific and pragmatic grounds.

Classification and Diagnosis: Fundamental Concepts

Classification and diagnosis are often confused, but they differ in crucial ways. A system of psychiatric classification provides an overarching taxonomy of mental illness, whereas 'diagnosis' is the act of placing an individual, based on a constellation of signs (observable indicators, such as pressured speech in a patient in the manic phase of bipolar disorder), symptoms (subjective indicators, such as racing thoughts in the same patient), or both, into a category within that taxonomy. Classification is a prerequisite for diagnosis.

In psychiatric classification, as in other domains of medicine, we can distinguish among syndrome, disorder, and disease (Kazdin, 1983) on the basis on our levels of understanding of their pathology – the physiological changes that ostensibly accompany the condition – and etiology, that is, causation (Gough, 1971; Lilienfeld *et al.*, 1994). As we move from syndrome to disorder to disease, our understanding of pathology and etiology gradually increases.

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. Panic disorder is an exemplar of a syndrome because its signs (e.g., sweating) and symptoms (e.g., intense fears of losing control or of becoming psychotic) tend to covary across individuals. Nevertheless, the pathology and etiology of panic disorder remain largely unknown, although plausible theories abound. Many classic psychiatric syndromes reflect what engineers term 'known failure modes' (Marcus, 2009), that is, characteristic patterns of breakdown of adaptive systems. For example, the panic attacks of panic disorder appear to be 'false alarms,' that is, massive bursts of sympathetic nervous system activity reflecting fight-flight reactions that occur in the absence of genuine threat (Barlow, 2001). As a consequence, these attacks comprise constellations of signs and symptoms representing a coordinated emergency response to a perceived, but imaginary, threat (indeed, the 'sympathetic' nervous system acquires its name from the fact that the nerves of this system respond 'in sympathy' – that is, in unison – to environmental challenges).

In rare cases, syndromes comprise constellations of signs and symptoms that display minimal covariation across individuals, but that point to an underlying etiology (Lilienfeld *et al.*, 1994). For example, Gerstmann's syndrome in neurology (Benton, 1992) is marked by four major features: 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 features are negligibly correlated across individuals in the general population, they co-occur dependably following damage near the angular gyrus in the left hemisphere's parietal lobe.

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 DSM-5, 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 (hair-pulling disorder), and illness anxiety disorder (a new DSM-5 condition that is similar to hypochondriasis in DSM-IV), we can be reasonably certain that an individual exhibiting marked obsessions, compulsions, or both, suffers from a well-defined disorder, namely OCD.

At the highest rung of the hierarchy of understanding lie diseases, which are disorders whose pathology and etiology are well understood (McHugh and Slavney, 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.

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 and Jablensky, 2003; Kendler, 2005). This fact reminds us that the pathology in most cases of psychopathology is largely unknown, and their etiology poorly understood.

Psychiatric Classification from DSM-I to DSM-5

Prior to the 1950s, the state of psychiatric classification in the United States was largely disorganized, because no standard system was in place for operationalizing mental disorders. Indeed, prior to World War I, there was scant interest in developing a systematic classification of mental disorders (Grob, 1991), and even following 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' would often bear scant correspondence to what another diagnostician meant by the same term.

This situation began to change in 1918, when the U.S. Bureau of the Census released the Statistical Manual for the Use of Institutions of the Insane, which classified mental disorders (mostly psychotic disorders) into 22 groups. This manual underwent 10 revisions through 1942. Nevertheless, this manual was greatly limited in its coverage of psychopathology. It was not until 1952, when the American Psychiatric Association (APA) released the first edition of the *Diagnostic and Statistical Manual of Mental Disorders*, first edition abbreviated as DSM-I (American Psychiatric Association, 1952), that a full-fledged diagnostic system came into place. Although DSM-I was a slim 132 pages in length, it was a landmark. For the first time, it offered reasonably explicit, albeit brief, descriptions of psychiatric diagnoses, thereby facilitating inter-rater reliability among clinicians and researchers. DSM-II appeared 16 years later (American Psychiatric Association, 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 (e.g., Beck, 1962) and DSM-II were marked by relatively low levels of inter-rater reliability, probably because their somewhat vague narrative descriptions of disorders allowed substantial room for subjective judgment. In addition, DSM-I and DSM-II were shaped 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 behavioral and cognitive–behavioral psychologists, found these classification systems challenging to implement. 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 unhealthy reactions to life events (Lief, 1948).

Largely in response to these criticisms, the APA, with psychiatrist Robert Spitzer at the helm, released DSM-III in 1980 (American Psychiatric Association, 1980). DSM-III was a wholesale revision of the diagnostic manual that represented a radical change in thinking and approach from all that came before. Moreover, it has provided the template for all subsequent DSMs (Klerman, 1984; Mayes and Horwitz, 2005). Coming in at a hefty 494 pages, a nearly fourfold increase from DSM-II, DSM-III not only dramatically increased the coverage of mental disorders – from 163 to 224 – but presented far more detailed guidelines than its predecessors for establishing diagnoses. The operational and philosophical approach of DSM-III was neo-Kraepelinian (Compton and 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.

In accord with its neo-Kraepelinian emphasis, DSM-III provided (1) standardized diagnostic criteria and (2) algorithms, or decision rules, for each diagnosis in an effort to enhance inter-rater reliability. 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, DSM-III was influenced by the pioneering efforts of the St. Louis psychiatric group at Washington University (including Robins, Guze, Winokur, and other major figures in 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 a manic episode, which is required for the diagnosis of bipolar disorder, DSM-III mandated that individuals experience (1) 'one or more distinct periods with a predominantly elevated, expansive, or irritable mood' (p. 208) and (2) at least three of seven signs and symptoms (or four of seven if the person's mood is predominantly irritable rather than elevated or expansive), such as excessive talkativeness, decreased need for sleep, and inflated self-esteem, most of the time for at least 1 week.

DSM-III also outlined 'hierarchical exclusion rules' for many diagnoses; such rules prevent clinicians and researchers from making these diagnoses if other diagnoses can better 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 or closely related conditions, or if it appeared to be due to either an organic mental disorder (e.g., hypothyroidism) or uncomplicated bereavement (a prolonged grief reaction).

The inter-rater reliability of DSM diagnoses has been further enhanced by the development of structured and semi-structured diagnostic interviews, such as the Structured Clinical Interview for DSM (SCID; First *et al.*, 2002), which are coordinated explicitly around DSM criteria. These interviews consist of standardized questions – to be read verbatim by interviewers – and both required and recommended follow-up probes with which to assess specific diagnostic criteria (First *et al.*, 2002). A version of the SCID for DSM-5 is now available.

In contrast to its predecessors, DSM-III was essentially agnostic with respect to theoretical orientation, such as psychoanalysis. As a consequence, 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).

DSM-III-Revised (DSM-III-R), which appeared in 1987 (American Psychiatric Association, 1987), and DSM-IV, which appeared in 1994 (American Psychiatric Association, 1994) (and in a more expanded text revision in 2000), retained the major features and innovations of DSM-III. Nevertheless, they continued to increase their coverage of psychopathology;

DSM-IV, 943 pages long, contained 374 diagnoses (American Psychiatric Association, 2000).

Both DSM-IM-III-R and DSM-IV gradually moved away from a 'monothetic' approach to diagnosis, emphasized in much of DSM-III, and toward a 'polythetic' approach. In a monothetic approach, signs and symptoms are singly necessary and jointly sufficient for a diagnosis. In contrast, in a polythetic approach, signs and symptoms are neither necessary nor sufficient for a diagnosis. The potential disadvantage of a polythetic approach is extensive symptomatic heterogeneity. In DSM-5, for example, 256 different sign/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-5 criteria for obsessive-compulsive disorder and to share no diagnostic criteria. More recently, Galatzer-Levy and Bryant (2013) found that there are a remarkable 636 120 possible sign/symptom combinations of DSM-5 symptom criteria for posttraumatic stress disorder (PTSD), an eightfold jump up from the mere (!) 79 794 combinations among DSM-IV criteria. Nevertheless, most scholars agree that the disadvantage of heterogeneity is outweighed by the higher inter-reliability of the polythetic approach (Widiger *et al.*, 1991). In a monothetic approach, disagreement regarding the presence or absence of a single criterion necessarily leads to disagreement regarding the presence or absence of the diagnosis. In contrast, in a polythetic approach, such disagreement often has no impact on levels of agreement regarding the presence or absence of the diagnosis, because raters can typically 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 so characteristic of a disorder that it 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 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).

Finally, DSM-IV added a section for 'culture-bound syndromes' in its Appendix in recognition of the fact that some conditions vary, or at least vary markedly in their expression, across cultures (Draguns and Tanaka-Matsumi, 2003). Most culture-bound syndromes are widely known in non-Western cultures, although their etiology and linkages to conditions diagnosed in Western cultures are controversial. 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, or nonverbal behavior. It may be a *formes frustes* – a variant – of social anxiety disorder (social phobia) that is prevalent in cultures, especially in Asia, that stress group harmony above individual autonomy (Kleinknecht *et al.*, 1994). The section on culture-bound syndromes has been retained in revised form in the DSM-5 Appendix under the heading 'Cultural Concepts of Distress.'

Following the publication of DSM-IV in 1994 (American Psychiatric Association, 1994) and its text revision in 2000, a great deal of data accumulated regarding the prevalence and correlates of DSM diagnoses. In an effort to accommodate these new data, DSM-5, spearheaded by David Kupfer and Darrel Regier, was published in May of 2013 (American Psychiatric Association, 2013) amidst a host of criticisms. By and large, DSM-5 retained the principal format and categories of DSM-IV. One goal of DSM-5 was to stem the tide of the perceived proliferation of new diagnoses by relying on rigorous validity data for potential new conditions; nevertheless, many scholars contend that the manual was at best only partially successful in these goals.

Although DSM-5 is the predominant system for psychiatric classification around the world, it is not without competitors. In particular, Chapter V of the *International Classification of Diseases*, 10th revision (ICD-10) of the World Health Organization (1993) is an alternative to DSM-5 that has been adopted in many countries outside of the United States (ICD-11 is under development as of this writing). Although ICD-10 is similar in many ways to DSM-5, such as its use of explicit diagnostic criteria and algorithms, some of the categories differ in nontrivial ways. In one study of over 1300 patients, the concordance between ICD-10 and DSM-IV for disorders ranged from a low of 33% for substance abuse to a high of 87% for dysthymic disorder (which is termed persistent depressive disorder in DSM-5; Andrews *et al.*, 1999). A comprehensive analysis of the overlap between ICD-10 and DSM-5 awaits further research.

Criticisms of DSM-5 and Future Directions

Even prior to its publication, DSM-5 was a lightning rod for scientific controversy. One major concern was that the field trials for DSM-5 were inadequate, focusing largely on clinical feasibility, with scant examination of the (1) validity of new diagnostic categories or (2) potential effects of alterations in extant categories on the prevalence of DSM disorders (Frances and Widiger, 2012).

In addition, numerous critics raised concerns that much of the DSM-5 'overmedicalized' normality, that is, transformed problems of everyday living, such as relatively mild concerns regarding physical health or teenage anger outbursts, into disorders (Frances, 2012). It has done so, these authors contend, by (1) increasing the number of diagnoses and (2) lowering the threshold for a number of extant diagnoses. In this way, DSM-5 may pathologize a host of largely normative behaviors, emotions, and thoughts. For example, the DSM-5 diagnosis of disruptive mood dysregulation disorder, which is intended to capture some cases of what some authors believe to be pediatric bipolar disorder, was criticized by Frances (2012) for 'turn(ing) temper tantrums into a mental disorder.'

Another potential example is the DSM-5 category of minor neurocognitive disorder, which some authors contend may unduly pathologize mild forgetfulness and other largely normative cognitive problems often associated with aging (Batstra and Frances, 2012). These and another 'psychiatric hangnails,' as these diagnoses are sometimes called pejoratively, may extend the range of psychopathology into the normal range, thereby blurring the distinction between psychological health and abnormality.

As noted earlier, the growing evidence for the dimensionality of many psychiatric conditions, particularly personality disorders, has led many authors to recommend replacing or supplementing the DSM with a set of dimensions derived from the basic science of personality (Krueger *et al.*, 2011; Widiger and Clark, 2000). One candidate for a dimensional framework is the five-factor model (Goldberg, 1993), which comprises five dimensions that have emerged repeatedly in factor analyses of omnibus (broadband) measures of personality: extraversion, neuroticism, agreeableness, conscientiousness, and openness to experience. More recently, the framers of DSM-5 considered a related dimensional model for personality disorders influenced by the work of Harkness and his colleagues (see Harkness and McNulty, 1994). In this model, five dimensions of antagonism, detachment, negative affectivity (similar to but broader than neuroticism), disinhibition, and psychoticism would have been used to describe all personality variation in the abnormal range. Nevertheless, this proposal was vetoed at the eleventh hour by the APA Board of Trustees, in part because its clinical feasibility was deemed to be insufficiently demonstrated. These dimensions instead appear in Section 3 of DSM-5, devoted to 'Emerging Measures and Models,' in an effort to encourage further research with an eye toward future revisions of the manual (American Psychiatric Association, 2013).

Finally, another potential future direction for psychiatric classification is a shift away from the longstanding 'sign and symptom' approach of previous manuals, including DSM-5 and ICD-10. Many scholars have contended, with some justification, that the current nearly exclusive focus on signs and symptoms to classify psychopathology has resulted in categories that do not adequately detect the true state of nature. Endophenotypic markers, which are presumably closer to the gene end of the gene-behavior pathway than are traditional signs and symptoms, might assist researchers to better identify psychobiological dimensions that are dysfunctional in mental disorders (Casey *et al.*, 2013).

Until recently, most of the proposals to implement endophenotypic markers were limited to supplementing the diagnosis of existing psychiatric categories, such as major depression or bipolar disorder. With one exception, none of these proposals has been accepted given that most of these markers display only modest validity for their respective diagnoses. The lone exception is for the diagnosis of narcolepsy, a sleep disorder. Beginning with DSM-5, the diagnostic criteria now include low levels of hypocretin in cerebrospinal fluid (Casey *et al.*, 2013).

A more radical and ambitious proposal emanates from the recent initiative supported by the National Institute of Mental Health (NIMH) to develop Research Domain Criteria (RDoC) as a full-fledged alternative to the DSM-5 and ICD-10. As of this writing, RDoC is more of an envisioned research approach than a proposed classification system. Its goal is to measure well-established psychobiological circuits that undergird both healthy functioning and psychopathology (Morris and Cuthbert, 2012), along with promising endophenotypic markers of these circuits. Potential examples of such brain circuits include reward systems, fear systems, impulse control systems, and working memory. In turn, each of these circuits would be measured using indicators at differing levels of analysis, including observable behavior, self-report measures, laboratory measures, and neuroimaging (Insel *et al.*, 2010; Sanislow *et al.*, 2010). Ultimately, RDoC could supplement or even supplant the extant DSM system, but its long-term success remains to be seen. Nevertheless, given the limitations of the DSM and similar systems of classification, it seems prudent to encourage the emergence of competing frameworks.

See also: Clinical Assessment. Culture and Mental Health. Personality Disorders

References

American Psychiatric Association, 1952. Diagnostic and Statistical Manual of Mental Disorders, first ed. Washington, DC: APA.
American Psychiatric Association, 1968. Diagnostic and Statistical Manual of Mental Disorders, second ed. Washington, DC: APA.
American Psychiatric Association, 1980. Diagnostic and Statistical Manual of Mental Disorders, third ed. Washington, DC: APA.
American Psychiatric Association, 1987. Diagnostic and Statistical Manual of Mental Disorders, third ed. (revised). Washington, DC: APA.
American Psychiatric Association, 1994. Diagnostic and Statistical Manual of Mental Disorders, fourth ed. Washington, DC: APA.
American Psychiatric Association, 2000. Diagnostic and Statistical Manual of Mental Disorders, fourth ed. (text rev.). Washington, DC: APA.
American Psychiatric Association, 2013. Diagnostic and Statistical Manual of Mental Disorders, fifth ed. Washington, DC: APA.
Andreasen, N.C., 1995. The validation of psychiatric diagnosis: New models and approaches. American Journal of Psychiatry 152, 161–162.
Andrews, G., Slade, T., Peters, L., 1999. Classification in psychiatry: ICD-10 versus DSM-IV. The British Journal of Psychology 174, 3–5.
Barlow, D.H., 2001. Anxiety and its Disorders: The Nature and Treatment of Anxiety and Panic, second ed. New York, NY: Guilford Press.
Batstra, L., Frances, A., 2012. Diagnostic inflation: Causes and a suggested cure. Journal of Nervous and Mental Disease 200, 474–479.
Beck, A.T., 1962. Reliability of psychiatric diagnoses: 1. A critique of systematic studies. American Journal of Psychiatry 119, 210–216.
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: Lilienfeld, S.O., O'Donohue, W.T. (Eds.), The Great Ideas of Clinical Science: 17 Principles That Every Mental Professional Should Understand. New York, NY: Routledge, pp. 93–118.
Blashfield, R.K., Draguns, J.G., 1976. Evaluative criteria for psychiatric classification. Journal of Abnormal Psychology 85, 140–150.
Casey, B.J., Craddock, N., Cuthbert, B.N., *et al.*, 2013. DSM-5 and RDoC: Progress in psychiatry research? Nature Reviews Neuroscience 14, 810–814.
Caspi, A., Houts, R.M., Belsky, D.W., *et al.*, 2013. The p Factor: One general psychopathology factor in the structure of psychiatric disorders? Clinical Psychological Science. doi:10.1177/2167702613497473.
Compton, W.M., Guze, S.B., 1995. The neoKraepelinian revolution in psychiatric diagnosis. European Archives of Psychiatry and Clinical Neuroscience 245, 196–201.
Cramer, A.O., Waldorp, L.J., van der Maas, H.L., Borsboom, D., 2010. Comorbidity: A network perspective. Behavioral and Brain Sciences 33, 137–150.
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.

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 and his new clothes: DSM-III in perspective. American Journal of Psychiatry 143, 962–967.
Feighner, J., Robins, E., Guze, S., *et al.*, 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, NY: Biometrics Research, New York State Psychiatric Institute.
Frances, A., 1980. The DSM-III personality disorders section: A commentary. American Journal of Psychiatry 137, 1050–1054.
Frances, A.J., 2012. The DSM-5 is not a bible: Ignore its ten worst changes. Psychology Today. Available at: http://www.psychologytoday.com/blog/dsm5-in-distress/201212/dsm-5-is-guidenot-bible-ignore-its-ten-worst-changes (accessed 09.02.14).
Frances, A.J., Widiger, T., 2012. Psychiatric diagnosis: Lessons from the DSM-IV past and cautions for the DSM-5 future. Annual Review of Clinical Psychology 8, 109–130.
Galatzer-Levy, I.R., Bryant, R.A., 2013. 636,120 Ways to have posttraumatic stress disorder. Perspectives on Psychological Science 8, 651–662.
Gangestad, S., Snyder, M., 1985. "To carve nature at its joints": On the existence of discrete classes in personality. Psychological Review 92, 317–349.
Garb, H.N., 1998. Studying the Clinician: Judgment Research and Psychological Assessment. Washington, DC: American Psychological Association.
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.
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.
Greenberg, G., 2013. The Book of Woe: The DSM and the Unmaking of Psychiatry. New York, NY: Penguin.
Grob, G.N., 1991. Origins of DSM-I: A study in appearance and reality. American Journal of Psychiatry 148, 421–431.
Harkness, A.R., McNulty, J.L., 1994. The Personality Psychopathology Five (PSY-5): Issue from the pages of a diagnostic manual instead of a dictionary. In: Strack, S., Lorr, M. (Eds.), Differentiating Normal and Abnormal Personality. New York, NY: Springer, pp. 291–315.
Haslam, N., Holland, E., Kuppens, P., 2012. Categories versus dimensions in personality and psychopathology: A quantitative review of taxometric research. Psychological Medicine 42, 903–920.
Insel, T., Cuthbert, B., Garvey, M., *et al.*, 2010. Research Domain Criteria (RDoC): Developing a valid diagnostic framework for research on mental disorders. American Journal of Psychiatry 167, 748–751.
Kazdin, A.E., 1983. Psychiatric diagnosis, dimensions of dysfunction, and child behavior therapy. Behavior Therapy 14, 73–99.
Kendell, R., Jablensky, A., 2003. Distinguishing between the validity and utility of psychiatric diagnoses. American Journal of Psychiatry 160, 4–12.
Kendler, K.S., 2005. Toward a philosophical structure for psychiatry. American Journal of Psychiatry 162, 433–440.
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. Behavioral Therapist 17, 175–178.
Klerman, G., 1984. The advantages of DSM-III. American Journal of Psychiatry 141, 539–542.
Krueger, R.F., Eaton, N.R., Clark, L.A., *et al.*, 2011. Deriving an empirical structure of personality pathology for DSM-5. Journal of Personality Disorders 25, 170–191.
Krueger, R.F., Watson, D., Barlow, D.H., 2005. Introduction to the special section: Toward a dimensionally based taxonomy of psychopathology. Journal of Abnormal Psychology 114 (4), 491–493.
Lief, A.A. (Ed.), 1948. The Commonsense Psychiatry of Dr. Adolf Meyer: Fifty-Two Selected Papers. New York, NY: McGraw-Hill.
Lilienfeld, S.O., Smith, S.F., Watts, A.L., 2013. Issues in diagnosis: Conceptual issues and controversies. In: Craighead, W.E., Miklowitz, D.J., Craighead, L.W. (Eds.), Psychopathology: History, Diagnosis, and Empirical Foundations, second ed. Hoboken, NJ: John Wiley & Sons, pp. 1–35.
Lilienfeld, S.O., Waldman, I.D., Israel, A.C., 1994. A critical examination of the use of the term and concept of comorbidity in psychopathology research. Clinical Psychology: Science and Practice 1, 71–83.
Loevinger, J., 1957. Objective tests as instruments of psychological theory. Psychological Reports 3, 635–694.
Marcus, G., 2009. Kluge: The Haphazard Evolution of the Human Mind. Boston: Houghton Mifflin Harcourt.
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.
McHugh, P.R., Slavney, P.R., 1998. The Perspectives of Psychiatry, second ed. Baltimore, MD: Johns Hopkins University 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., 1989. Schizotaxia revisited. Archives of General Psychiatry 46, 935–944.
Meehl, P.E., Golden, R., 1982. Taxometric methods. In: Kendall, P.C., Butcher, J.N. (Eds.), Handbook of Research Methods in Clinical Psychology. New York, NY: Wiley, pp. 127–181.
Menninger, K., 1963. The Vital Balance. New York, NY: Viking Press.
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.
Michels, R., 1984. A debate on DSM-III: First rebuttal. American Journal of Psychiatry 141, 548–553.
Morey, L.C., 1991. Classification of mental disorder as a collection of hypothetical constructs. Journal of Abnormal Psychology 100, 289–293.
Morris, S.E., Cuthbert, B.N., 2012. Research Domain Criteria: Cognitive systems, neural circuits, and dimensions of behavior. Dialogues in Clinical Neuroscience 14, 29–37.
Nkam, I., Bocca, M.L., Denise, P., *et al.*, 2010. Impaired smooth pursuit in schizophrenia results from prediction impairment only. Biological Psychiatry 67, 992–997.
Robins, E., Guze, S.B., 1970. Establishment of diagnostic validity in psychiatric illness: Its application to schizophrenia. American Journal of Psychiatry 126, 983–987.
Ross, C., Pam, A., 1996. Pseudoscience in Biological Psychiatry: Blaming the Body. New York, NY: Wiley.
Sanislow, C.A., Pine, D.S., Quinn, K.J., *et al.*, 2010. Developing constructs for psychopathology research: Research Domain Criteria. Journal of Abnormal Psychology 119, 631–639.
Skeem, J.L., Cooke, D.J., 2010. One measure does not a construct make: Directions toward reinvigorating psychopathy research — Reply to Hare and Neumann (2010). Psychological Assessment 22, 455–459.
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: Millon, T., Klerman, G.L. (Eds.), Contemporary Directions in Psychopathology: Toward the DSM-IV. New York, NY: Guilford Press, pp. 307–330.
Stuart, S., Pfohl, B., Battaglia, M., *et al.*, 1998. The cooccurrence of DSM-III-R personality disorders. Journal of Personality Disorders 12, 302–315.
Sutton, E.H., 1980. An Introduction to Human Genetics. Philadelphia, PA. College: Saunders.
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.
Waldman, I.D., 2005. Statistical approaches to complex phenotypes: Evaluating neuropsychological endophenotypes of attention-deficit/hyperactivitydisorder. 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: Ollendick, T.H., Prinz, R.J. (Eds.), Advances in Clinical Child Psychology, vol. 17. New York, NY: Plenum Press, pp. 323–363.
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. *et al.*, (Eds.), 1998. DSM-IV Sourcebook, vol. 4. Washington, DC: American Psychiatric Press.
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.
World Health Organization, 1993. The ICD-10 classification of mental and behavioural disorders: Diagnostic criteria for research. Geneva, Switzerland: World Health Organization.