

## Toward *DSM-V* and the Classification of Psychopathology

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The *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV)* developed by the American Psychiatric Association (1994) is a compelling effort at a best approximation to date of a scientifically based nomenclature, but even its authors have acknowledged that its diagnoses and criterion sets are highly debatable. Well-meaning clinicians, theorists, and researchers could find some basis for fault in virtually every sentence, due in part to the absence of adequate research to guide its construction. Some points of disagreement, however, are more fundamental than others. The authors discuss issues that cut across individual diagnostic categories and that should receive particular attention in *DSM-V*: (a) the process by which the diagnostic manual is developed, (b) the differentiation from normal psychological functioning, (c) the differentiation among diagnostic categories, (d) cross-sectional vs. longitudinal diagnoses, and (e) the role of laboratory instruments.

The impetus for the development of official nomenclatures for the diagnosis of mental disorders was the crippling confusion generated by their absence. Prior to their development, "confusion reigned" (Kendell, 1975, p. 87). "Every self-respecting alienist, and certainly every professor, had his [*sic*] own classification" (Kendell, 1975, p. 87). Communication among clinicians and researchers is problematic, to say the least, in the absence of a common, uniformly accepted nomenclature. On the other hand, the science of psychopathology may not be sufficiently advanced at this time to develop an adequately conclusive or even authoritative nomenclature. Innovative research may at times be constrained by a requirement to use a standard, uniform nomenclature (Clark, Watson, & Reynolds, 1995; Pincus, Frances, Davis, First, & Widiger, 1992). The *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV)* developed by the American Psychiatric Association (1994) does appear to be a compelling effort at a best approximation to date of such a nomenclature, but even its authors have acknowledged that its diagnoses and criterion sets are highly debatable (Frances, Pincus, Widiger, Davis, & First, 1990; Spitzer, Williams, & Skodol, 1980).

There might not in fact be one sentence within *DSM-IV* for which well-meaning clinicians, theorists, and researchers could not find some basis for fault. Some points of disagreement, however, are more fundamental than others. In this article, we discuss process and format issues that cut across individual diagnostic categories, issues that we believe should receive particular consideration for *DSM-V*. We begin with the process by which the

diagnostic manual is developed, followed by a discussion of the differentiation from normal psychological functioning, the differentiation among diagnostic categories, cross-sectional versus longitudinal diagnoses, and the role of laboratory instruments in diagnosis. Space limitations prohibit a detailed discussion of specific proposals for individual domains of psychopathology, nor can we cover all of the important issues facing the authors of *DSM-V*, including whether to provide alternative criterion sets for different ethnic, gender, and cultural groups, but informative reviews of these issues are available elsewhere (see, e.g., Garb, 1997; Hartung & Widiger, 1998; Kirmayer, Young, & Hayton, 1995; Okazaki & Sue, 1995; Whaley, 1997).

### Process of Development

As a common language for communication across clinicians and researchers with divergent theoretical orientations toward the etiology, pathology, and treatment of mental disorders, a nomenclature should not favor one particular theoretical perspective over another (Frances et al., 1990; Spitzer & Williams, 1985, 1987; Spitzer et al., 1980; Widiger & Trull, 1993). A nomenclature governed by a particular theoretical model (see, e.g., Follette & Houts, 1996) would provide a premature authority for that model and would not be usable by persons who did not share the same theoretical perspective. A uniform language is also advantageous in providing to researchers a means by which to develop a common data set with which to compare the validity of alternative theoretical models (Wakefield, 1998).

However, considerable disagreement over the language to be contained within an official nomenclature is inevitable, given the substantial diversity in theoretical orientations among the researchers and clinicians who use the nomenclature, the significant impact that mental disorder diagnoses can have on society and clinical practice, and the lack of unambiguous research to govern final decisions. Most every critique of a decision made for *DSM-III* through *DSM-IV* has questioned the support for, or process by which, the decision was made (see, e.g., Caplan, 1991; Clark et al.,

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1995; Follette & Houts, 1996; Kirk & Kutchins, 1992; Sarbin, 1997; Zimmerman, 1998). The process by which the nomenclature is developed is as important, and, in anticipation of the inevitable dispute and controversy, should perhaps be given as much attention, as the decisions themselves.

### Historical Background

*DSM-I (American Psychiatric Association, 1952).* The first edition of the *DSM* was developed largely by a central committee of leading clinicians and researchers on the basis of their own clinical experiences and their implicit understanding of the existing literature (Blashfield, 1984; Nathan, 1994). The *DSM-I* committee (chaired by George Raines) submitted a draft to 10% of the membership of the American Psychiatric Association, but no specific results of this survey were ever reported.

*DSM-II (American Psychiatric Association, 1968).* *DSM-II* was constructed by a process similar to *DSM-I*. A central committee of leading clinicians and researchers (chaired by Ernest Gruenberg) reviewed proposed revisions and reached a consensus. A draft was sent to 120 psychiatrists with a special interest in diagnosis, but again, specific results were not published.

*DSM-III (American Psychiatric Association, 1980).* In 1974, the American Psychiatric Association appointed a committee (chaired by Robert Spitzer) to revise *DSM-II* in a manner that would incorporate research innovations (Spitzer & Williams, 1985). A draft of *DSM-III* was published in 1978, but no systematic survey of support for the proposals was conducted. "In attempting to resolve various diagnostic issues, the [*DSM-III*] Task Force relied, as much as possible, on research evidence relevant to various kinds of diagnostic validity" (American Psychiatric Association, 1980, p. 3), including, for example, "the largest reliability study ever done" (Spitzer et al., 1980, p. 154). However, the American Psychiatric Association acknowledged that there was virtually no systematic research for many of the criterion sets and inadequate research support for all but a few. "It should be understood . . . that for most of the categories the diagnostic criteria are based on clinical judgment" (American Psychiatric Association, 1980, p. 8). "Thus, the subjective judgment of the members of the task force . . . played a crucial role in the development of *DSM-III*, and differences of opinion could only rarely be resolved by appeal to objective data" (Spitzer, 1985, p. 523). Spitzer (1985) further acknowledged that "because appeals to objective data for resolving nosologic controversies were relatively rare, speaking and writing skills (rhetoric) played an important role in resolving controversies" (p. 523).

*DSM-III-R (American Psychiatric Association, 1987).* Soon after the publication of *DSM-III*, a number of problems with the criterion sets became evident. The American Psychiatric Association (1987) indicated that there were "many instances in which the criteria were not entirely clear, were inconsistent across categories, or were even contradictory" (p. xvii). Therefore, in 1983, the American Psychiatric Association appointed a work group (chaired by Robert Spitzer) to make corrections and clarifications. The field trials this time focused on validity as well as reliability. "The purpose of these field trials was to examine the feasibility of proposed criteria for the disorders and to determine the optimal number of items to require for maximizing sensitivity and specificity, using . . . clinicians' diagnoses as the criterion" (Spitzer &

Williams, 1988, p. 872). Field trials were conducted on draft criteria for autism, disruptive behavior disorders, agoraphobia, self-defeating personality disorder, and substance dependence (see, e.g., Rounsaville, Kosten, Williams, & Spitzer, 1987; Spitzer & Williams, 1988; Spitzer, Williams, Kass, & Davies, 1989).

Two drafts of proposed revisions were made available, although no systematic survey of the support for these drafts was conducted. The authors of *DSM-III-R* made a more concerted effort to review systematically the prior research on each diagnosis, facilitated in part by the substantial amount of research generated by *DSM-III* (Spitzer & Williams, 1987). Skodol and Spitzer (1987), for example, compiled within an annotated bibliography "all of the data available to the American Psychiatric Association's Work Group to Revise *DSM-III*" (p. xi), including 2,010 citations. However, one indication of the potential inadequacy of the process by which *DSM-III-R* was developed was the eventual need to include an appendix for "Proposed Diagnostic Categories Needing Further Study" (American Psychiatric Association, 1987, p. 367) in which to place three diagnoses approved for inclusion by the *DSM-III-R* Work Group that were subsequently vetoed by the American Psychiatric Association's Board of Trustees, who concluded that there had been insufficient empirical support to offset compelling objections. Late luteal phase dysphoric disorder, self-defeating personality disorder, and sadistic personality disorder were placed in this appendix. A fourth diagnosis approved by the *DSM-III-R* central committee, paraphiliac rapism, was deleted altogether from the manual.

*DSM-IV (American Psychiatric Association, 1994).* In May of 1988, the American Psychiatric Association's Board of Trustees appointed a committee (chaired by Allen Frances) to begin work on *DSM-IV*. The *DSM-IV* committee aspired to use a more conservative threshold for the approval of new diagnoses and to have the decisions be guided more explicitly by the scientific literature (Nathan & Langenbucher, 1999). Frances, Widiger, and Pincus (1989) suggested that "the major innovation of *DSM-IV* will not be in its having surprising new content but rather will reside in the systematic and explicit method by which *DSM-IV* will be constructed and documented" (p. 375). Proposals for additions, deletions, or revisions were guided by 175 literature reviews that were required to use a specific format that maximized the potential for critical review, containing, for example, a method section that documented explicitly the criteria for including and excluding studies and the process by which the literature had been reviewed. Testable questions that could be addressed with existing data sets were also explored in 36 studies, which emphasized the aggregating of multiple data sets from independent researchers (Clark, 1992). Finally, 12 field trials were conducted to provide reliability and validity data on proposed revisions (Nathan & Langenbucher, 1999). These field trials emphasized the comparison of alternative proposals (primarily the proposed research criteria for ICD-10; World Health Organization, 1992) with respect to multiple internal and external validators assessed across multiple research sites that provided relevant clinical populations. Critical reviews of written reports from each of these 223 projects were obtained by sending initial drafts to advisors or consultants for a respective work group, by presenting drafts at relevant conferences, and by submitting drafts to peer-reviewed journals.

It is naive to suggest that research will clearly indicate, without any dispute or controversy, that a particular diagnosis or criterion ought to be included, deleted, or revised. . . . However, the inevitable ambiguity and disagreement are themselves strong arguments for providing explicit, written documentation of the rationale, justification, and empirical support for any proposals, which is widely and critically reviewed before decisions are made. (Widiger, Frances, Pincus, Davis, & First, 1991, pp. 286–287)

The final versions of each report were eventually published within a series of archival texts (e.g., Widiger et al., 1998).

### Discussion

It is evident from the above overview that the process by which the diagnostic manual is constructed is becoming increasingly well documented and empirically supported (Clark et al., 1995; Nathan & Langenbucher, 1999; Widiger et al., 1991). Nevertheless, it is also evident that valid concerns remain. We discuss below the criteria for revision, participation, critical review, and pilot testing.

*Criteria for revision.* A concern common to many critiques of the *DSM* is whether the decisions have reflected simply the biased perspectives of a small group of persons, often characterized derogatorily as a reliance on expert consensus (see, e.g., Clark et al., 1995; Follette & Houts, 1996; Sarbin, 1997; Zimmerman, 1988). Even the authors of the diagnostic manual have expressed this concern. The authors of *DSM-IV* were critical of the extent to which the decisions made for *DSM-III* “were based on expert opinions rather than systematic evidence” (Frances et al., 1990, p. 1439). Spitzer (1991), in turn, was equally skeptical of the success achieved by the authors of *DSM-IV*: “My own prediction is that when final decisions are made about *DSM-IV*, they will still be based primarily on expert consensus, rather than on data, as was the case with the *DSM-III* and *DSM-III-R*” (p. 294).

Criticism of a reliance on expert consensus is for the most part an expression of skepticism concerning the ability of any particular group of persons to be fair, objective, or accurate in their interpretation of the literature. Persons critical of a reliance on expert consensus allude to an alternative approach in which decisions are purportedly made instead on the basis of objective scientific data, ignoring the point that scientific data are inevitably inadequate, ambiguous, and inconclusive, and do not speak for themselves (Faust & Miner, 1986; Kendler, 1990; Widiger & Trull, 1993). No diagnostic manual can be constructed without a group of fallible persons interpreting the results of existing research. These persons ideally would be consensus scholars with no preconceptions and with an adequate understanding of the research and issues (Cooper, 1984), but “participants are [in fact] rarely neutral with respect to the issues they are addressing, and it can be difficult for them to provide a dispassionate, balanced, and objective review and interpretation of the research” (Widiger & Trull, 1993, p. 73).

One recommendation has been to constrain the decision-making authority. Blashfield, Sprock, and Fuller (1990), for example, proposed the development of specific and explicit criteria for the addition or deletion of diagnostic categories, analogous to the reliance on specific and explicit diagnostic criteria that constrain the decision-making power of practicing clinicians (Spitzer et al., 1980). This proposal would not eliminate expert consensus, as the final decisions regarding the algorithm(s) to use would themselves be controversial in part because they would have to be developed

through a committee process of expert consensus. Blashfield et al. had proposed in particular the requirement that there be at least 50 journal articles (with at least 25 reporting empirical data) published within the preceding 10 years for a diagnosis to be approved for inclusion. Reaching a consensus on a precise number of studies, including the quality of their methodology and the nature of their findings, would be difficult (perhaps impossible) to achieve (Widiger & Trull, 1993). Nevertheless, the effort toward the development of explicit algorithms might facilitate more consistent and objective decision making, as well as a fuller understanding and appreciation of the many variables and concerns that can have an impact on a particular decision.

*Participation.* Sadler (in press) has suggested that much of the controversy and disgruntlement with each edition of the *DSM* has been due to the absence of adequate opportunity for persons with divergent viewpoints to participate in the decision-making process (see, e.g., Caplan, 1991; Kirk & Kutchins, 1992; Schacht, 1985; Schacht & Nathan, 1977). Sadler proposed that the final decisions be made on the basis of a democratic vote of clinicians (e.g., members of the American Psychiatric Association) or, alternatively, the committee members could be elected through a democratic process to ensure that all clinicians’ perspectives are being represented. Increased participation in the process of decision making, however, may in fact have no appreciable effect on the extent of criticism of or disagreement with the final decisions. In any case, Spitzer et al. (1980) have argued compellingly for narrowing rather than increasing participant involvement. Opinion surveys regarding the final decisions were not solicited for *DSM-III* because “no one wanted to repeat the scene of the general membership voting on a presumably ‘scientific’ issue, as was done in 1973 on the issue of the elimination of homosexuality from the *DSM-II* classification” (Spitzer et al., 1980, p. 152).

True scientific progress, which we believe should be the basis for revisions to the diagnostic manual, would not proceed through a process of democratically voting on the validity of alternative theories. Voting on whether or not premenstrual dysphoria is a mental disorder that should receive official recognition in *DSM-V* would make the decision less scientific and more political. Disputes are inevitable in any scientific field and are in fact desirable and healthy components of scientific progress (Weimer, 1979). The goal is to resolve the controversy in a manner that has the most validity rather than in the manner that is the most representative of general opinion. Decisions should be informed by a fair hearing of the diversity of perspectives, and these viewpoints and perspectives should be systematically, comprehensively, and enthusiastically solicited. However, the most scientifically valid decision may at times be politically incorrect. The authors of the diagnostic manual should then have the authority to make innovative decisions that are scientifically justified even when they are contrary to general clinical consensus (Clark et al., 1995). Nevertheless, with this authority comes substantial power and responsibility. The selection of those who are to be given this authority might then be as important as the process itself.

*Critical review.* The *DSM-IV* Task Force addressed, in part, skepticism regarding the ability of persons to reach a fair, balanced, or optimal interpretation of inconclusive or inadequate research by obtaining critiques of the literature reviews and proposals, a fundamental component of any scientific process (Widiger & Trull, 1993). Nevertheless, perhaps this process was itself

inadequately systematic, comprehensive, or documented. The authors of *DSM-IV* indicated that "the methods and results of each stage of review were shared with advisors who evaluated initial drafts of the literature reviews for inaccuracies, gaps in coverage, and biased interpretations of the research" (Davis et al., 1998, p. 6), and there are indeed indications that considerable effort was made to obtain critical review and to maintain quality control. However, the credibility and success of the process of review would be facilitated by a more systematic solicitation and documentation of critiques. Informed persons outside the decision-making process, including persons likely to be critical of a proposal, should be requested to provide written critiques, with the understanding that they would be published with the original reviews in an archival document (see, e.g., Widiger et al., 1996).

*Pilot studies.* Existing research is usually inadequate in documenting the likely effects of proposed revisions, and it is perhaps beyond the expertise of the authors of a nomenclature to fully anticipate them (Blashfield, Blum, & Pfohl, 1992). The need to have *DSM-III-R* to correct the many unanticipated problems and errors contained within *DSM-III* is itself strong testament for the importance of adequate pilot testing. The failure to conduct pilot studies of a criterion set is uncomfortably comparable to releasing a psychological test for publication in the absence of validation data (Blashfield & Livesley, 1991). It is remarkable that no field testing was conducted for many of the diagnostic criterion sets that received final approval for inclusion within the diagnostic manual, given the substantial significance of this official nomenclature for many important social, forensic, and clinical decisions (Frances et al., 1990). The rationale for diagnostic criteria may be well intended and even compelling, but how the criteria will in fact be used or understood by clinicians and researchers and how they will relate empirically to other diagnostic criteria within typical clinical settings will often be surprisingly problematic (Blashfield et al., 1992; Clark & Watson, 1995; Morey, 1988).

The field testing conducted for *DSM-IV* was much more substantial than had been conducted for prior editions of the manual (Davis et al., 1998), but the pilot research still fell far short of being comprehensive, due in part to the absence of adequate funding. The *DSM-IV* field trials were funded largely by the National Institute of Mental Health (NIMH). The entire budget was generous, but when parialed out across the numerous sites of the 12 studies, the funding was grossly inadequate. The budget for a sufficient number of field trials to cover all of the proposed revisions would be substantial and well beyond any reasonable support by the NIMH, but the costs may in fact be well within the range of the profits that have been generated by the sales of the book (Nietzel, 1996; Zimmerman, 1988).

### Boundary With Normality

An issue fundamental to the validity of the nomenclature has been whether the diagnostic system can differentiate abnormality from normality. A difficult task facing the authors of *DSM-V* will be establishing meaningful boundaries or points of demarcation between abnormal and normal psychological functioning, if any such distinctions can in fact be made. The absence of adequate guidance within the current system for establishing the threshold for any particular mental disorder's diagnosis has been problematic throughout the nomenclature.

### Absence of Meaningful Distinction

The definition of mental disorder provided in *DSM-IV* (American Psychiatric Association, 1994) is largely the result of an effort by the authors of *DSM-III* (American Psychiatric Association, 1980) to develop a set of specific and explicit criteria for deciding whether a behavior pattern (homosexuality in particular) should be classified as a mental disorder and therefore warrant inclusion (Spitzer & Williams, 1982). The intense controversy over homosexuality has largely abated, but the issues raised in this historical debate over whether to classify a particular behavior pattern as a mental disorder apply as well to the current decisions regarding the circumstances under which any particular behavior pattern already classified as a mental disorder should be diagnosed.

For example, for an adult to be diagnosed with the mental disorder of pedophilia, *DSM-III-R* (American Psychiatric Association, 1987) required only that the adult have recurrent intense urges and fantasies involving sexual activity with a prepubescent child over a period of at least 6 months and have acted on them or be markedly distressed by them. Because virtually every adult who had engaged in a sexual activity with a child would meet this threshold for the diagnosis of pedophilia, the *DSM-III-R* diagnosis in essence presumed that no adult was capable of willfully engaging in this deviant sexual act or fantasy for longer than 6 months without being compelled to do so by the presence of a mental disorder. The same criticism applied to the other paraphilias, including exhibitionism, fetishism, sexual masochism, voyeurism, and transvestic fetishism.

The authors of *DSM-IV*, therefore, added the requirement that "the behavior, sexual urges, or fantasies cause clinically significant distress or impairment in social, occupational, or other important areas of functioning" (American Psychiatric Association, 1994, p. 523). Spitzer and Wakefield (1999), however, have concurred with a concern raised by the National Law Center for Children and Families that *DSM-IV* may have now normalized pedophilic (and other paraphilic) behavior by allowing the diagnosis not to be applied if the persons who have engaged in these acts are not themselves distressed by their behavior or do not otherwise experience any impairment to social or role functioning. In response, Frances, First, and Pincus (1995) had argued that these deviant sexual "behaviors are inherently problematic because they involve a nonconsenting person (exhibitionism, voyeurism, frotteurism) or a child (pedophilia) and may lead to arrest and incarceration" (p. 319). Therefore, any person who engaged in an illegal sexual act for longer than 6 months would be exhibiting a clinically significant social impairment and would therefore meet the *DSM-IV* threshold for the diagnosis of a mental disorder. The arguments of Frances et al. might provide a compelling rejoinder to the concerns of Spitzer and Wakefield, but falling back on the illegality of the behavior undermines the original rationale for the inclusion of the impairment criterion, namely, to provide a meaningful basis for determining when deviant sexual acts or fantasies are or are not due to a mental disorder. As stated in the *DSM-IV* definition of a mental disorder, "neither deviant behavior (e.g., political, religious, or sexual) nor conflicts that are primarily between the individual and society are mental disorders unless the deviance or conflict is a symptom of a dysfunction in the individual" (American Psychiatric Association, 1994, p. xxii).

In sum, missing from both the *DSM-III-R* and the *DSM-IV* paraphilia criterion sets are the means by which to determine whether the sexually deviant behaviors or fantasies are the result of a dysfunction (or pathology) within the individual. This is not to say that the deviant sexual behaviors are not illegal or that the sexual exploitation of a child is not harmful, but that neither *DSM-III-R* nor *DSM-IV* provides adequate guidance for how to distinguish a willful and voluntary deviant sexual behavior that is engaged in for longer than 6 months from sexual activities that are compelled by the presence of a mental disorder.

Regier, Kaelber, et al. (1998) raised a related concern regarding the absence of a meaningful boundary between normal and abnormal functioning for the diagnoses of less controversial disorders. Regier et al. were concerned with the apparently high prevalence rates obtained for many of the anxiety, mood, and other mental disorders by the NIMH Epidemiologic Catchment Area program (ECA; Robins & Regier, 1991) and the National Comorbidity Survey (NCS; Kessler et al., 1994).

Based on the high prevalence rates identified in both the ECA and NCS, it is reasonable to hypothesize that some syndromes in the community represent transient homeostatic responses to internal or external stimuli that do not represent true psychopathologic disorders. (Regier, Kaelber, et al., 1998, p. 114)

This statement suggests that there is a definable boundary between normal versus pathologic expressions of anxiousness and depressiveness that the diagnostic criterion sets are not adequately demarcating.

It is possible that many people with currently defined mental syndromes (in particular among the affective and anxiety disorders) not brought to clinical attention may be having appropriate homeostatic responses that are neither pathologic nor in need of treatment—e.g., other equivalents of grief reactions that meet clinical criteria but are not considered pathologic. (Regier, Kaelber, et al., 1998, p. 114)

They suggested that researchers use a higher threshold when providing diagnoses within community samples by requiring “additional severity, impairment, comorbidity, and duration criteria beyond those in the ICD-10 and the *DSM-IV*” (Regier, Kaelber, et al., 1998, p. 114).

The proposal of Regier, Kaelber, et al. (1998) would be consistent with an understanding of the criterion sets as fallible indicators of the presence of psychopathology and with an alteration of the threshold for diagnosis when the base rate of a respective disorder is appreciably different than that obtained within the clinical populations for which the diagnostic criteria were primarily constructed (Meehl & Rosen, 1955). Considerable evidence, however, challenges the assumption of a distinct boundary between true psychopathology versus homeostatic responses (see, e.g., Judd, Paulus, Wells, & Rapaport, 1996; Klein, Lewinsohn, & Seeley, 1996). Thus, the challenge facing the developers of *DSM-V* may not be to differentiate more clearly between normal and pathologic expressions of behavior; rather, it may be to determine whether or not a qualitative distinction can in fact be made. A more realistic goal might be to develop arbitrary but reasonable and meaningful quantitative points of demarcation along more continuous distributions of functioning.

### *Presence of Pathology?*

Wakefield (1997) provided examples of criterion sets from *DSM-IV* that he argued failed to make a necessary distinction between maladaptive problems in living and true psychopathology because of the reliance within the criterion sets on indicators of distress or impairment rather than indicators of an underlying pathology. For example, the *DSM-IV* criterion set for major depressive disorder currently excludes uncomplicated bereavement presumably because depressive reactions to the loss of a loved one are normal (nonpathological) problems in living. Depression after the loss of a loved one is considered a mental disorder if the symptoms “are characterized by marked functional impairment, morbid preoccupation with worthlessness, suicidal ideation, psychotic symptoms, or psychomotor retardation” (American Psychiatric Association, 1994, p. 327) or if “the symptoms persist for longer than two months” (American Psychiatric Association, 1994, p. 327). Allowing 2 months to grieve before one is diagnosed with a mental disorder might be as arbitrary and meaningful as allowing a person to engage in a sexually deviant act for 6 months before the behavior is diagnosed as a paraphilia.

Explicit within *DSM-IV*'s definition of a mental disorder is that the condition “must currently be considered a manifestation of a behavioral, psychological, or biological dysfunction in the individual” (American Psychiatric Association, 1994, pp. xxi–xxii), but very few of the diagnostic criterion sets refer explicitly to the hypothesized behavioral, psychological, or biological dysfunction, emphasizing instead the distress and impairment that are presumably the manifestations of this pathology. Important questions for *DSM-V* are whether to incorporate explicitly a representation of an underlying pathology within each criterion set and whether the presence of this pathology is necessarily categorical or could be viewed quantitatively. If the latter, then the concern for defining a precise boundary between normality and psychopathology might become less important than determining the appropriate professional response to different variants and degrees of pathology (Spitzer & Williams, 1982; Widiger & Corbitt, 1994).

The boundaries of the diagnostic manual are increasing with each edition, and there has been vocal concern that much of this expansion represents an encroachment on normal problems of living (Caplan, 1995; Follette & Houts, 1996; Rogler, 1997). There were indeed a number of new diagnoses included within an appendix to *DSM-IV* that were disputed precisely because they might be below an appropriate threshold for diagnosis, such as mixed anxiety–depressive disorder, premenstrual dysphoric disorder, age-related cognitive decline, and minor depressive disorder. The inclusion of the underlying pathology within a diagnostic criterion set would go far in providing a scientifically and clinically meaningful distinction between (or a dimension along which to distinguish) normal and abnormal psychological functioning (Regier, Kaelber, et al., 1998; Spitzer & Wakefield, 1999; Wakefield, 1997). However, a substantial limitation of this proposal is that there is unlikely to be much agreement regarding the fundamental pathology that should be required for many of the disorders included within the diagnostic manual. The presence of such pathology is not currently required in large part because there is insufficient empirical support to give preference to one cognitive, interpersonal, neurochemical, psychodynamic, or other theoretical model over another. Wakefield (1997), for example, suggested that

for the diagnosis of major depressive disorder (vs. normal bereavement), it should be

necessary to formulate some account... of the evolutionary programming of the mechanisms with respect to what kinds of triggering circumstances are supposed to cause which kinds of responses (e.g., loss-response mechanisms are designed so that perceptions of major losses trigger roughly proportional sadness responses). (p. 647)

Evolutionary theory has enriched current understanding of the etiology and pathology of many mental disorders, but it may be unable to provide empirical guidance concerning the natural functions of specific behavioral and psychological response mechanisms, and, given that it is itself a particular theoretical model of psychopathology, it might not be capable of serving as a general definition of mental disorder that is compatible with or could integrate alternative perspectives (Bergner, 1997; Kirmayer & Young, 1999; Lilienfeld & Marino, 1995, 1999).

The assumption that the expansion of the nomenclature is subsuming normal problems in living may itself be questionable. Persons critical of the nomenclature have suggested that the expansion is more political than scientific (see, e.g., Caplan, 1995; Follette & Houts, 1996; Kutchins & Kirk, 1997; Rogler, 1997). However, it would have been more surprising to find that scientific research and increased knowledge had led to the recognition of fewer instances of psychopathology rather than more (Wakefield, 1998). The not-otherwise-specified category is the most frequently provided diagnosis in general clinical practice, perhaps because the nomenclature is currently inadequate in its coverage (Clark et al., 1995; Westen & Arkowitz-Western, 1998). The expectation that an assessment of pathology will be successful in identifying a qualitatively distinct point of demarcation may not withstand empirical scrutiny. Even the ostensibly normal cases of bereavement described by Regier, Kaelber, et al. (1998) and Wakefield (1997) might display indicators of an irrational cognitive schema or a neurochemical dysregulation (Prigerson et al., 1999). There is currently no consensus within the bereavement literature as to when grief should be regarded as abnormal (Horowitz et al., 1997; Rosenzweig, Prigerson, Miller, & Reynolds, 1997). Optimal psychological functioning, as in the case of optimal physical functioning, might represent an ideal that is achieved by only a small minority of the population. The rejection of a high prevalence rate of psychopathology may reflect the best of intentions, such as concerns regarding the stigmatization of mental disorder diagnoses (Kutchins & Kirk, 1997) or the potential impact on funding for treatment (Regier, Kaelber, et al., 1998), but these social and political concerns could also hinder a more dispassionate and accurate recognition of the true rate of a broad range of psychopathology within the population (Widiger & Sankis, 2000).

### *Presence of Impairment or Distress?*

Impairment and distress may in fact provide the optimal means by which to identify a meaningful boundary between, or an important parameter for quantifying, normal and abnormal psychological functioning, if these constructs are more precisely defined, calibrated, and assessed. The clinically significant impairment criterion

helps establish the threshold for the diagnosis of a disorder in those situations in which the symptomatic presentation by itself (particular-

ly in its milder forms) is not inherently pathological and may be encountered in individuals for whom a diagnosis of "mental disorder" would be inappropriate. (American Psychiatric Association, 1994, p. 7)

However, no guidelines are provided in *DSM-IV* for determining what is meant by a clinically significant impairment. It is only stated that "assessing whether this criterion is met, especially in terms of role function, is an inherently difficult clinical judgment" (American Psychiatric Association, 1994, p. 7) and that consideration should be given to information provided by family members and other third parties. Widiger and Corbitt (1994) offered a number of alternative interpretations for a clinically significant impairment, each of which had quite different implications for a diagnostic threshold (e.g., impairments for which treatment would be necessary or beneficial for improved, adequate, optimal, desirable, or necessary functioning).

A useful model for the classification of psychopathology might be provided by the diagnosis of mental retardation (Widiger, 1997). Mental retardation is currently defined in large part as the level of intelligence below an intelligence quotient (IQ) of 70 (American Psychiatric Association, 1994). This point of demarcation does not carve nature at a discrete joint, distinguishing the presence versus absence of an underlying pathology. It is an arbitrary point of demarcation along a continuous distribution of cognitive functioning. There are persons below an IQ of 70 for whom a qualitatively distinct disorder is evident, but the disorder in these cases is not mental retardation; it is a physical disorder (e.g., Down's syndrome) that can often be traced to a specific biological event (the diagnosis in such instances should perhaps be mental retardation due to a general medical condition). Intelligence is itself a multifactorial construct with a variety of complexly interacting etiologies that is best described as a continuous variable (Neisser et al., 1996), and there does not appear to be a discrete break in its distribution that would provide a qualitative distinction between normal and pathologic intelligence.

This is not to say that any point of demarcation along the continuum of intellectual functioning has been chosen randomly or would be necessarily meaningless, inappropriate, or unreasonable (Wakefield, 1999). On the contrary, a substantial amount of thought and research has supported a selection of an IQ of 70 as providing a meaningful and reasonable point at which to characterize lower levels of intelligence as resulting in a clinically significant level of impairment that warrants professional intervention. A consideration for the authors of *DSM-V* might be the development of comparable scales for the characterization and demarcation of clinically significant impairments in other domains of functioning (Regier, Kaelber, et al., 1998). This effort would be facilitated by the more dimensional models of classification being developed across the diagnostic nomenclature (Clark et al., 1995; Widiger, 1997). Regrettably, space limitations prohibit us from doing more than listing the main domains in which such models have shown promise for deepening the understanding of disorder. These include (but are not limited to) the personality disorders (see, e.g., Clark, Livesley, Schroeder, & Irish, 1996; Livesley, 1998; Trull et al., 1998), the mood and anxiety disorders (see, e.g., T. A. Brown, Chorpita, & Barlow, 1998; Clark & Watson, 1991; Mineka, Watson, & Clark, 1998; Zinbarg & Barlow, 1996), and the psychotic disorders (see, e.g., Grube, Bilder, & Goldman, 1998;

Kwapil, Miller, Zinser, Chapman, & Chapman, 1997; Ratakonda, Gorman, Yale, & Amador, 1998; Toomey, Faraone, Simpson, & Tsuang, 1998; Verdoux et al., 1998). It would be difficult to reach a consensus as to a precise point of demarcation, and even the effort might be perceived as fundamentally problematic to the expectation that there is a qualitative point of demarcation. Nevertheless, the task would at least facilitate a productive effort to define and assess more precisely what is meant by a clinically significant level of distress or impairment within various domains of functioning that would address the virtual absence of any such guidelines in the current nomenclature.

### Differentiation Among Disorders

Difficulty in delineating a point of demarcation between normal and abnormal psychological functioning is paralleled by an equally fundamental problem in differentiating the mental disorders from each other. "In *DSM-IV*, 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" (American Psychiatric Association, 1994, p. xxii). This carefully worded disclaimer, however, is somewhat hollow, as "*DSM-IV* is a categorical classification that divides mental disorders into types based on criterion sets with defining features" (American Psychiatric Association, 1994, p. xxii). Clinicians who follow the guidelines provided within *DSM-IV* diagnose and interpret its disorders for the most part as discrete clinical conditions; much of the effort exerted by the authors of each edition is for the development of criteria that would clarify diagnostic boundaries; and the purpose of most of the new additions to the manual (e.g., bipolar II, mixed anxiety-depressive disorder, depressive personality disorder, and postpsychotic depressive disorder) is to fill in holes and gaps along the boundaries of existing categories (although at the same time creating new diagnostic co-occurrences and problematic boundaries).

### Extensive Diagnostic Co-Occurrence

Many prior reviews have documented the high degree of diagnostic co-occurrence across a wide range of disorders (see, e.g., Clark et al., 1995; Klein & Riso, 1993; Lilienfeld, Waldman, & Israel, 1994; Sher & Trull, 1996; Widiger, 1997) and have explored in some detail the basis for the phenomenon in specific domains, such as anxiety and depression (see, e.g., Mineka et al., 1998). We reference these reviews, augmenting their findings with the contributions of recent papers and discussing implications for future editions of the *DSM*. Most commonly, researchers have used the term comorbidity to describe this phenomenon, but there is controversy over this terminology and, relatedly, the underlying meaning of diagnostic co-occurrence (Lilienfeld et al., 1994). We will use the more neutral term co-occurrence.

Diagnostic co-occurrence was minimized to some extent in *DSM-III* through the provision of exclusionary rules affecting some 60% of all disorders (Boyd et al., 1984). A diagnosis was not made if a disorder was occurring in conjunction with another disorder that was considered on some basis to warrant precedence (T. A. Brown & Barlow, 1992; Spitzer & Williams, 1987). These exclusion criteria proved highly controversial, however, because there was little empirical evidence and inadequate theoretical ra-

tionale to justify their use; they were vague and difficult to apply; and they hindered the study of disorders lower in the diagnostic hierarchy (Clark et al., 1995; Klein & Riso, 1993). When researchers explored the implications of ignoring these exclusion criteria, they discovered widespread co-occurrences among disorders that potentially had important diagnostic and treatment implications. Accordingly, the large majority of such exclusion criteria were eliminated in *DSM-III-R* (American Psychiatric Association, 1987).

The ensuing research documented overwhelmingly that in community samples, it was more common for individuals to have co-occurring than single disorders, whereas in clinical samples, diagnostic purity was a rare and atypical phenomenon (Clark et al., 1995; Klein & Riso, 1993; Lilienfeld et al., 1994; Sher & Trull, 1996). Moreover, subsequent studies have extended understanding of the phenomenon in various ways. For example, subsyndromal diagnoses, such as recurrent brief depression (Angst, 1996), show co-occurrence patterns similar to those seen with full diagnoses. This suggests that estimates of diagnostic co-occurrence based only on full-syndrome diagnoses may underestimate the extent of the phenomenon and that studies of diagnostic co-occurrence that include subsyndromes may help to inform investigations of the normal-abnormal continuum.

Early studies were cross-sectional and examined either concurrent or retrospectively determined lifetime diagnoses, but there are problems with both of these methods. Because of shared symptoms, estimations of concurrent diagnoses may exaggerate the true rate of co-occurrence (Caron & Rutter, 1991; Widiger & Shea, 1991). In the case of lifetime diagnoses, estimates may suffer from such methodological problems as biases in retrospective recall. Nevertheless, although the levels of co-occurrence differed (with lifetime higher than concurrent rates), the co-occurrence patterns that have emerged from these two types of studies have been quite similar, lending support to the validity of each (Mineka et al., 1998). Moreover, longitudinal studies have yielded similar findings. For example, Ball, Otto, Pollack, and Rosenbaum (1994) studied panic disorder patients, one third of whom initially reported a prior episode of depression and 35% of whom initially reported a concurrent generalized anxiety disorder (GAD). Over a 2-year period, a quarter of the sample experienced a major depressive episode, with those who had reported either past depression and/or concurrent GAD at far greater risk than those who did not.

### Bases for Co-Occurrence

In an extensive longitudinal, epidemiological study, Krueger, Caspi, Moffitt, and Silva (1998) used structured interviews to assess a range of psychopathology in a large unselected birth cohort in New Zealand at ages 18 and 21. Using structural equation modeling to examine cross-sectional and longitudinal co-occurrence patterns, they determined that a two-factor model provided the best fit to the data. Of the diagnoses that had sufficiently high base rates to be included in their model, various depressive (major depressive disorder, dysthymia) and anxiety (agoraphobia, GAD, social phobia, simple phobia, and obsessive-compulsive) disorders were indicators of a latent internalizing factor, whereas conduct disorder, marijuana dependence, and alcohol dependence were indicators of a latent externalizing factor. From age 18 to 21, the internalizing factor was moderately stable and the externalizing

factor highly stable. Moreover, attempts to model a more differentiated structure that distinguished a larger number of specific disorders were unsuccessful. Thus, these data supported a dimensional approach to diagnosis over a more traditional, categorical one.

Taken together, these data suggest that the co-occurrence of mental disorders cannot be explained simply on the basis of either symptom overlap or methodological bias. Rather, they add support to the conclusion that common disorders are "reliable, covariant indicators of stable, underlying 'core pathological processes'" (Krueger et al., 1998, p. 216). In anticipation of *DSM-V*, investigations into the nature of these underlying core processes will become increasingly important. Existing data provide evidence that both genetic and environmental influences may contribute to diagnostic co-occurrence, as well as to the differentiation of disorders (Livesley, Jang, & Vernon, 1998; Ratakonda et al., 1998; Sherman, Iacono, & McGue, 1997). Thus, common diagnostic syndromes may result from the systematic interplay of definable genetic traits and current stressors.

To examine the effect of past and current stressors on anxiety and depression, G. W. Brown, Harris, and Eales (1996) studied 404 working-class women in London who were married or cohabiting and had a child at home. Adversity in both childhood (e.g., sexual/physical abuse and neglect) and current adulthood (e.g., significant deaths, divorce or separation, or domestic violence) were assessed through extensive interviews. Log-linear analyses revealed that about 30% of the overlap between anxiety and depression could be explained by the fact that childhood adversity independently increased the prevalence of both types of disorders, thus leading to greater co-occurrence by chance alone. That is, because chance co-occurrence is determined solely by the frequency of each disorder, their increased prevalence leads to greater chance co-occurrence. An additional 9% was due to the common effect of childhood adversity on the two types of disorder. That is, childhood adversity was a direct risk factor for both anxiety and depression and so increased their co-occurrence. Interestingly, adult adversity was a risk factor only for depression and thus did not contribute to the anxiety-depression overlap. These data indicate how environmental events may contribute to diagnostic co-occurrence both directly (as a common risk factor) and indirectly (through their influence on prevalence). In reaching conclusions for the *DSM-V* about diagnostic (re)organization based on co-occurrence data, it will be important to consider these two distinct types of environmental influence.

As for genetic data, beginning in the 1980s, numerous studies—both epidemiological and twin-based—examined the genetic links between anxiety and depressive symptoms and disorders (Mineka et al., 1998). These analyses indicated that the observed phenotypic covariation was due largely to a common genetic factor that also influenced neuroticism, a broad personality trait reflecting individual differences in subjective distress. Together with the finding mentioned earlier that subsyndromal and full diagnoses yield similar diagnostic co-occurrence patterns, these data suggest a link between studies on diagnostic co-occurrence and those investigating the normal-abnormal boundary. That is, it is plausible that these core pathological processes encompass both ends of continua ranging from normal sensibilities to highly maladaptive responses. As such, a wide variety of methods including the full population range may be used to study these processes, which may

further the understanding of psychopathological phenomena more rapidly than if investigations were limited to clinical samples.

Genetic studies have revealed that the greatest overlap among depressive and anxiety disorders is between major depression and GAD, whereas other anxiety disorders, such as panic or obsessive-compulsive disorder, show a more modest overlap with depression, as well as some genetic independence from each other despite their phenotypic co-occurrence (Kendler et al., 1995). Recently, T. A. Brown, Barlow, and colleagues (T. A. Brown, Antony, & Barlow, 1995; T. A. Brown et al., 1998) have demonstrated this same pattern at the phenotypic level. These data suggest a potential reorganization for *DSM-V*. Specifically, Mineka et al. (1998) recommended "rearranging the mood and anxiety disorders so as to place greater emphasis on the close affinity between distress-based disorders such as major depression and GAD" (p. 391).

Nowhere is this close affinity more evident than in children and, to a lesser extent, adolescents. Cole, Truglio, and Peeke (1997) conducted a large-scale, multitrait, multimethod study of anxious and depressive symptoms in children and early adolescents. Children in third and sixth grade, their parents, teachers, and peers completed multiple symptom measures of anxiety and depression. Confirmatory factor analyses revealed a unitary anxiety-depressive factor in the third-grade data, with modest differentiability of anxiety and depressive factors in the sixth-grade data. A strength of the study was the existence of multiple raters. That is, if the findings were based only on children's self-report, a methodological explanation would be plausible (e.g., sixth graders have greater cognitive capacity and so have greater ability to discriminate between symptoms). However, the data were consistent across raters, suggesting that anxiety and depression emerge developmentally from a single underlying distress construct that, based on the genetic data presented earlier, would also subsume neuroticism.

Cole et al. (1998) followed these children longitudinally, obtaining retests from the children and their parents every 6 months for 3 years. Using structural equation modeling, they showed that both anxiety and depression were highly stable across the 3-year period (cf. Krueger et al., 1998) and also that early anxiety predicted the emergence of depression, but not vice versa, a finding that is consistent with previous reviews of adult and nonhuman-primate data (Alloy, Kelly, Mineka, & Clements, 1990; Mineka et al., 1998). This suggests that anxiety is the more basic phenomenon (cf. the near identity of neuroticism and anxiety; Clark, Watson, & Mineka, 1994) from which depression—and perhaps anxiety disorders other than GAD, such as panic and obsessive-compulsive disorder—emerge as more complex disorders. Thus, longitudinal, developmental studies can provide further understanding of the origins of anxiety and depressive disorders that, in turn, can inform diagnostic (re)organization of *DSM-V*.

### *Models of Co-Occurrence*

To integrate the extensive anxiety-depression co-occurrence data with various other types of information that document heterogeneity within this domain, Mineka et al. (1998) recently proposed a hierarchical model for anxiety and depression that builds on Clark and Watson's (1991) tripartite model and Barlow's (1991; Zinbarg & Barlow, 1996) hierarchical model of anxiety. In the integrated model, each syndrome contains both a common and

a unique component. The shared component, which primarily accounts for the co-occurrence data, represents broad individual differences in general distress (i.e., neuroticism). Each disorder also includes a unique or specific distinguishing component. For instance, anxious arousal is the specific component of panic disorder (T. A. Brown et al., 1998). An important task for future researchers is to specify more precisely the nature of these unique components (see Watson, 1999, for consideration of the specific component in other anxiety disorders), as well as the role of the general and specific components in each disorder. For example, the general distress component is central in both depression and GAD but appears to be of lesser importance in obsessive-compulsive disorder, social phobia, and specific phobia (see, e.g., T. A. Brown et al., 1998; Kendler et al., 1995).

We have focused on the anxiety and depressive disorders because extensive investigation into this domain has provided a wealth of data to examine. However, the phenomenon of co-occurrence is by no means limited to these disorders (Klein & Riso, 1993; Lilienfeld et al., 1994; Sher & Trull, 1996). Mineka et al. (1998) reported that the anxiety and depressive disorders themselves co-occur with a range of additional disorders including substance-use, hypochondriacal, somatization, eating, conduct, attention-deficit, and personality disorders. Extensive data suggest that this is because the general distress component is not at all specific to anxiety-depression but is quite broadly related to psychopathology (Hinden, Compas, Howell, & Achenbach, 1997; Watson & Clark, 1984, 1994). Significant elevations in this factor have been reported in a wide array of syndromes, including substance-use, somatoform, eating, personality, and conduct disorders, and schizophrenia (see, e.g., Krueger, Caspi, Moffitt, Silva, & McGee, 1996; Trull & Sher, 1994; Watson & Clark, 1994). Indeed, Widiger and Costa (1994) concluded that "neuroticism is an almost ubiquitously elevated trait within clinical populations" (p. 81). Thus, this integrative model clearly need not be confined to the mood and anxiety disorders and, as the profession looks toward *DSM-V*, researchers may need to expand the scope of the diagnostic manual to incorporate a broad range of associated phenomena.

Even so, as proposed, the Mineka et al. (1998) model addresses—or primarily has the capacity to address—the internalizing or distress disorders. However, on the basis of Krueger et al.'s (1998) results reported earlier, researchers will need a parallel model to account for the externalizing disorders, and an extensive literature already documents the overlap among various externalizing disorders, such as the substance-use, conduct, and antisocial personality disorders (Clark et al., 1995; Lilienfeld et al., 1994; Watson & Clark, 1993). It is likely that these disorders also are linked by a major common factor, analogous to the role of neuroticism in the internalizing disorders. A most likely candidate is another major personality dimension, disinhibition (Watson & Clark, 1993). This dimension was first identified (and misnamed as psychoticism) by Eysenck. Later, he characterized it as impulsivity, callousness, or toughmindedness (Eysenck & Eysenck, 1975), with the opposite end described as conscientiousness (McCrae & Costa, 1990; Wiggins, 1996) or constraint (Tellegen, 1985). Although the role of disinhibition in externalizing disorders is well known (see, e.g., Sher & Trull, 1994; Watson & Clark, 1993), little work has been done to investigate potential unique factors that would serve to differentiate specific disorders within this domain.

Yet another large literature reports the high degree of overlap among personality disorders and between the personality disorders and Axis I disorders (Clark et al., 1995; Oldham et al., 1992; Widiger, 1997; Widiger & Costa, 1994). It may be possible to integrate the personality disorders with either the Axis I internalizing or externalizing disorders, as suggested by the Krueger et al. (1998) data. Alternatively, given the limited diagnoses used in those data, more complex models might be needed (see, e.g., Slutske et al., 1998).

### *Dimensions, Categories, or Both?*

It is important to recognize that, most likely, specificity itself will prove to be relative rather than absolute. That is, it is highly unlikely that any factor (as opposed to a single, unique symptom) will be found to be entirely unique to a single disorder across the entire *DSM*. Moreover, one must be prepared for the patterns that emerge from examinations of symptom specificity not to conform neatly to existing diagnostic categories. For instance, anhedonia is an important symptom of depression but is not confined solely to that domain; it also characterizes (though perhaps to a lesser degree) schizophrenia, social phobia, and other disorders (see, e.g., T. A. Brown et al., 1998; Clark et al., 1994; Mineka et al., 1998; Watson & Clark, 1995). This suggests that the field must move toward more complex, multilevel hierarchical models in which groups of symptoms are classified at varying levels of specificity. Some types of symptoms will be broadly applicable to a wide range of disorders (e.g., those related to neuroticism may serve to help define the normal-abnormal continuum), others will apply to a moderate range of disorders, and still others will be focal for a small subset of disorders.

Furthermore, it may prove best to view individual disorders as representing particular combinations of different sets of symptoms, with each symptom type showing varying degrees of nonspecificity and with no symptom type being entirely unique to any single disorder. Ultimately, as one builds toward *DSM-V*, what may emerge is a structured set not of categorical diagnoses but of component dimensions, a set of symptom-cluster building blocks from which the panoply of diagnoses could be constructed. It is possible that some of these symptom-cluster dimensions will occur with sufficient regularity and that research may reveal an etiological basis for their co-occurrence, thus defining in such instances perhaps a meaningful categorical diagnosis. In other cases, regularly co-occurring sets of symptoms will be given labels and treated as categorical diagnoses for practical, descriptive purposes, even when lacking a specific etiology. The key point of this analysis is that the fundamental structure of future *DSMs* may not be composed of individual diagnoses as it is now. Rather, it may consist of an ordered matrix of symptom-cluster dimensions, a diagnostic table of the elements that are used in combination to describe the rich variety of human psychopathology.

### *Longitudinal Course of Disorders*

Specific and explicit criterion sets were a major innovation of *DSM-III*, credited in part to a return to a Kraepelinian approach to diagnosis (Blashfield, 1984; Klerman, 1986; Spitzer et al., 1980). Kraepelin (1919), however, gave as much emphasis to longitudinal observations and to the consideration of the course of a disorder as

he did to specific, behavioral indicators. Diagnostic course was fundamental to the success of Kraepelin's differentiation of schizophrenia from other mental disorders, evident today in the requirement that its symptoms must be evident for at least 6 months before the disorder can be diagnosed. As expressed by the neo-Kraepelinian psychiatrists Goodwin and Guze (1984) in their historical text on diagnosis, "a rose is a rose is a rose . . . because it remains a rose" (p. ix), emphasizing the importance of course for the establishment of an authoritative diagnosis. They in fact intoned, "diagnosis is prognosis" (p. ix). *DSM-III*, *DSM-III-R*, and *DSM-IV*, however, have continued instead to emphasize cross-sectional diagnoses. Duration requirements are included among all of the criterion sets, but with only a few notable exceptions (e.g., a history of a manic episode requires a diagnosis of a bipolar mood disorder in someone who is currently within a major depressive episode), these requirements are minimal and have played only a secondary role to current symptomatology.

One of the more apparent artificial temporal distinctions has been that between disorders of childhood and adulthood. "The provision of a separate section for disorders that are usually first diagnosed in infancy, childhood, or adolescence is for convenience only and is not meant to suggest that there is any clear distinction between 'childhood' and 'adult' disorders" (American Psychiatric Association, 1994, p. 37). The separate classification reflects instead the particular time within the life span that a researcher is studying or at which a clinician is intervening. The authors of *DSM-IV* did expend some effort to bridge artificial boundaries between childhood and adulthood. Information was provided in the text to indicate how each disorder varies in its presentation across the life span (Davis et al., 1998), and some *DSM-III-R* disorders of childhood and adulthood were collapsed into single diagnoses to provide a more developmental, life span perspective, including gender identity disorder, social phobia, and GAD. Diagnoses were also subtyped to characterize their longitudinal course, notably the specifiers for mood disorders (American Psychiatric Association, 1994). However, it is apparent that the amount of life span information that is provided in *DSM-IV* is only the tip of the iceberg of what should in fact be known. There are likely to be important differences between types of disorders that typically first appear at different points in the life span (cf. autism versus schizophrenia vs. bipolar disorder) that may be illuminated by a better understanding of developmental processes. Prospective longitudinal studies from childhood into adulthood (and into aging) are sorely needed to document empirically how particular disorders sustain, alter, or remit in their presentation across the life span (Lynam, 1996).

One of the more remarkable gaps in knowledge is the childhood antecedents for the personality disorders of adulthood. *DSM-III* included four childhood antecedents: identity disorder as an antecedent of borderline personality disorder, avoidant disorder as an antecedent of avoidant personality disorder, oppositional defiant disorder for passive-aggressive personality disorder, and conduct disorder for antisocial personality disorder (American Psychiatric Association, 1980); only one has remained. It is unclear why there would be so much empirical support for the childhood antecedent of antisocial personality disorder but almost none for any of the others (Widiger & Sankis, 2000).

The importance of considering the longitudinal course of a disorder, however, is becoming increasingly appreciated (Sher & Trull, 1996). For example, with increased specification of symp-

toms in recent editions of the *DSM*, limitations of cross-sectional diagnosis have come into focus. Research has clarified that symptom remission is typically a relative and partial phenomenon, with only a subset of patients achieving a truly complete and lasting recovery. Although this has long been the view for some disorders, such as schizophrenia (Goodwin & Guze, 1984) or personality disorders (American Psychiatric Association, 1980, 1987, 1994), it now has been documented for a wide range of disorders, including unipolar and bipolar depression (see, e.g., Angst, 1992; Coryell et al., 1994; Solomon, Keitner, Miller, Shea, & Keller, 1995), obsessive-compulsive disorder (Eisen & Steketee, 1997), GAD (Yonkers, Warshaw, Massion, & Keller, 1996), panic disorder (Katschnig & Amering, 1998), and substance-use disorders (Stanton & Joyce, 1993; Stoeffelmayr, Mavis, & Kasim, 1994). The failure to recognize this phenomenon has a number of potential consequences. A classification of individuals as noncases who are not asymptomatic but are simply below the arbitrary thresholds for diagnosis in *DSM-IV* yields lower estimates of diagnostic stability than using a more nuanced scale that includes subsyndromal ratings. Moreover, the psychosocial impairment in many subclinical cases will often have clinically and statistically significant effects on course, outcome, and treatment responsiveness, so it can be quite misleading to classify them as being without the presence of meaningful psychopathology (see, e.g., Judd et al., 1996; Skoog & Skoog, 1999).

Current symptomatology needs to be considered in the context of prior disorders. In addition, studies of lifetime diagnostic co-occurrence have revealed sets of disorders that, over time, appear repeatedly in the same individuals. For example, panic disorder and major depression may occur simultaneously but commonly occur sequentially, with panic disorder more likely to precede major depression than vice versa (see, e.g., Breier, Charney, & Heninger, 1984; Clark, 1989; Mineka et al., 1998). The meaning of such sequential comorbidity is an important issue. Over the course of a lifetime, many individuals suffer from phenotypically distinct medical disorders (e.g., strep throat and then later a stomach flu) without a question of sequential co-occurrence. By contrast, comorbid lifetime psychological disorders can be meaningfully related in a number of ways.

The sequential vulnerability hypothesis is that the initial disorder may act either directly or indirectly as a vulnerability factor for the subsequent development of the second disorder (see, e.g., Breslau, Davis, Andreski, Federman, & Anthony, 1998; Regier, Rae, Narrow, Kaelber, & Schatzberg, 1998). This hypothesis is certainly true to some degree, but may have little power to explain why certain diagnoses occur sequentially more frequently than others. Discussions of this hypothesis tend to emphasize general factors such as demoralization, lowered social support, or other kinds of psychosocial impairment that would increase vulnerability to a wide range of other psychological problems.

The shared vulnerability hypothesis is that sequentially co-occurring disorders may share a common etiological factor. If shared genes act as a vulnerability factor for more than one disorder, it seems equally plausible that their effects could be manifested in either simultaneous or sequential appearance of seemingly distinct disorders (e.g., childhood attention-deficit disorder and either childhood conduct disorder or adult antisocial behavior; Babinski, Hartsough, & Lambert, 1999; Cadoret & Stewart, 1991). Of course, the shared factor need not be genetic per

se; for example, Breier et al. (1984) speculated that "depression and panic disorder may represent manifestations of a common pathogenic process among a subgroup of psychiatric patients" (p. 1129). Such a pathogenic process could have environmental as well as genetic origins. For example, childhood sexual abuse appears to act as a vulnerability factor for a wide range of psychopathological conditions (G. W. Brown et al., 1996; Wilsnack, Vogeltanz, Klassen, & Harris, 1997; but see Rind, Tromovitch, & Bauserman, 1998, for a counterview).

Depending on the age of onset, the prior disorder may serve as an indicator of a more severe variant for the subsequent disorder. For example, Kovacs (1996) compared the literature on major depression between children and adults and reported that persons who had their first depressive episode in childhood were two to three times more likely to develop bipolar disorder than those whose onset was in adulthood. Of course, early age of onset may also be associated with more severe manifestations of psychopathology (Moffitt, 1993). Hasin, Grant, and Endicott (1988) reported that early age of onset of alcohol abuse was correlated with increased social and occupational impairments. These both may be indicators of a more severe form of the disorder; alternatively, environmental factors may have contributed to the early age of onset, and the increased impairments may simply reflect the cumulative effects of longer years of alcohol abuse.

The interweaving of some disorders is so frequent and pervasive that it would not be unreasonable to consider them alternative manifestations of a single disorder rather than co-occurring disorders (see Watson & Clark, 1995, for a discussion of these and other models of co-occurrence). For example, approximately 75% of patients with dysthymic disorder have a lifetime history of major depression (Keller et al., 1995). How reasonable is it to consider these individuals as having two distinct disorders rather than a single disorder with more chronic and more episodic manifestations? Such a view would be analogous to a chronic physical disorder (e.g., arthritis, asthma, or diabetes) in which acute exacerbations occur from time to time. That some individuals do not have acute exacerbations (i.e., do not have major depressive episodes within dysthymia) or that others suffer primarily from relatively discrete episodes is not an argument against this view. Rather, it speaks simply to the longitudinal heterogeneity of clinical presentation, which is likely as common among psychological disorders as physical illness. In other words, heterogeneity is most visible cross-sectionally but can also be observed longitudinally (see, e.g., Coryell et al., 1994).

In sum, despite Goodwin and Guze's (1984) pronouncement that "diagnosis is prognosis" (p. ix), too little attention has been given to the implications of diagnostic course either in constructing diagnostic criteria or in considering how the interplay of biological and environmental factors influences course, both singly and across related disorders. *DSM-IV* currently provides a multi-axial system for recognizing cross-sectional heterogeneity (Frances et al., 1995). A comparable means of characterizing a developmental, life span history of a patient's symptomatology should perhaps also be provided in *DSM-V*—by recording, for example, age of onset, lifetime history of disorders, and their longitudinal course. If one comes to understand how an anxiety disorder develops into a depressive disorder with which it shares a common genetic vulnerability, it could be impossible to persist with the notion that they are separate and distinct disorders. Once

again we are led to the conclusion that the future of the diagnostic manual rests on investigating the factors—genetic structures and environmental processes—that underlie the domain of psychopathology and determining how these factors combine to produce the range of clinically observed disorders.

### Laboratory Findings

Each of the mental disorders included within *DSM-IV* is accompanied by a text discussion of its typical course, prevalence, associated features, and other information that might be relevant to its diagnosis. The authors of *DSM-IV* added to the section devoted to associated features new subsections concerned with laboratory and physical examination findings. The inclusion of this material within the text is in anticipation of its eventual inclusion within the diagnostic criterion sets, a future decision that will be innovative but among the more controversial issues for *DSM-V*.

"Diagnoses in the rest of medicine are often heavily influenced by laboratory tests" (Frances et al., 1995, p. 22). Laboratory tests within medical practice go beyond the assessment of the signs, symptoms, or patient complaints that only suggest the presence of a particular medical disorder to provide a more direct and objective assessment of the underlying physical pathology. Anxiety and depression are purportedly the result of neurophysiological mechanisms, and the diagnostic criteria for some mental disorders include explicit references to autonomic functioning (e.g., palpitations, pounding heart, accelerated heart rate, sweating, chest pain, nausea, dizziness, chills or hot flushes, and paresthesias in the diagnosis of a panic attack; American Psychiatric Association, 1994), yet no physiological tests are required for their diagnosis. In addition, patient self-description within psychiatry can often be unreliable and misleading, as respondents will at times be unable or unwilling to describe their symptomatology accurately (Westen, 1997). A hope is that laboratory tests can do the same for psychiatry as they have done for other domains of medicine (Nemeroff, Kiltz, & Berns, 1999). "The increasing use of laboratory tests in psychiatric research raises the question of whether and when these tests should be included within the diagnostic criteria sets" (Frances et al., 1995, p. 22).

It is now stated in *DSM-IV* that "neurotransmitters implicated in the pathophysiology of a Major Depressive Episode include norepinephrine, serotonin, acetylcholine, dopamine, and gamma-aminobutyric acid" (American Psychiatric Association, 1994, p. 324), as indicated by measures of their levels in blood, cerebrospinal fluid, or urine and platelet receptor functioning. "Other laboratory tests that have demonstrated abnormalities include the dexamethasone suppression test, other neuroendocrine challenges, functional and structural brain imaging, evoked potentials, and [polysomnographic findings]" (American Psychiatric Association, 1994, p. 324). Nevertheless, despite the extensive discussion of these various neurophysiological tests within the text of *DSM-IV*, it was also acknowledged that "no laboratory findings that are diagnostic of a Major Depressive Disorder have been identified" (American Psychiatric Association, 1994, p. 323).

Krishnan and his colleagues have proposed for inclusion in *DSM-V* a diagnosis of vascular depression, the criteria for which would include magnetic resonance imaging results (Krishnan, Hays, & Blazer, 1997). "Just as vascular dementia is a manifestation of cognitive deficits associated with cerebrovascular pathol-

ogy, vascular depression should be viewed as a manifestation of depressive symptomatology associated with such pathology" (Steffens & Krishnan, 1998, p. 708). Krishnan et al. (1997) documented well the distinguishing features of this form of depression, including the presence of profound apathy, psychomotor retardation, and cognitive impairments in executive functioning; onset typically after the age of 50; associated stroke, series of transient ischemic attacks, or focal neurologic signs; lack of a family history of mood disorder; and documented evidence of cerebrovascular disease and neuropsychological impairments. Clinical implications include a slower recovery, more residual depressive symptoms, and greater refractoriness to traditional somatic treatments.

Neuroimaging criteria for vascular depression should specify criteria for vascular change. For example, criteria may include evidence of infarct, with lesion greater than 5 mm in diameter and irregular in shape, or if more standard systems are used, a score greater than 2 using the criteria of Fazekas et al. (1988). (Steffens & Krishnan, 1998, p. 709)

Substantial attention is being given to structural and functional brain imaging studies for many of the existing mental disorders, with the hope in part that these instruments could be used to diagnose or at least confirm the presence of a neurophysiological pathology (Kennedy, Javanmard, & Vaccarino, 1997; Soares & Mann, 1997). However, clearly limiting these and other neurophysiological measures' potential for incorporation within diagnostic criterion sets is the virtual absence of research indicating their ability to provide independent, blind diagnoses. These laboratory assessment instruments are useful in exploring and perhaps documenting neurophysiological correlates (Soares & Mann, 1997) but not necessarily in validating a neurophysiological model for their etiology (Kennedy et al., 1997) or perhaps even their pathology (Miller, 1996). Mayberg et al. (1999) investigated with positron emission techniques two complementary alterations in mood: transient sadness provoked in healthy volunteers and treatment-induced resolution of dysphoria in clinically depressed patients. The results indicated "reciprocal changes involving nearly identical limbic-paralimbic and neocortical regions" (Mayberg et al., 1999, pp. 678–679):

With sadness, increases in limbic-paralimbic blood flow (subgenual cingulate, anterior insula) and decreases in neocortical regions (right dorsolateral prefrontal, inferior parietal) were identified. With recovery from depression, the reverse pattern, involving the same regions, was seen—limbic metabolic decreases and neocortical increases. (Mayberg et al., 1999, p. 675)

The neurophysiology of a mood disorder might be only quantitatively different from the neurophysiology of normal sadness (Clark & Watson, 1991; Knutson et al., 1998). There are substantial differences in the neuroanatomy of the evaluative, experiential, and expressive components of sadness, happiness, and other emotions (Lane, Reiman, Ahern, Schwartz, & Davidson, 1997) but perhaps no discernible differences at the boundaries between clinically normal and abnormal (pathologic) sadness or happiness. Despite the enthusiasm for their potential diagnostic value, there are currently no studies that have assessed the sensitivity and specificity of neuroimaging techniques for the diagnosis or differential diagnosis of specific mental disorders (Kennedy et al., 1997;

Soares & Mann, 1997; Steffens & Krishnan, 1998). Until such research is conducted, it would be difficult to include these instruments within existing diagnostic criterion sets.

The inclusion of laboratory data in the diagnosis of a disorder was particularly controversial in the development of the *DSM-IV* for the sleep disorders. Most sleep disorder specialists use the International Classification of Sleep Disorders (ICSD) developed by the American Sleep Disorders Association (1990). The 12 *DSM-IV* sleep disorder diagnoses are coordinated with the ICSD but differ significantly in the absence of polysomnographic diagnostic criteria. "The most important and controversial question regarding the [sleep disorder] criteria sets . . . was the degree to which they should include the findings from sleep polysomnography" (Frances et al., 1995, p. 332). Detailed references are made to polysomnographic findings within the text discussion of various sleep disorders in *DSM-IV*, and it was acknowledged by its authors that "for sleep disorders other than insomnia, such as narcolepsy and sleep apnea, the utility of sleep laboratory testing is widely accepted" (Buysse, Reynolds, & Kupfer, 1998, pp. 1104–1105). Nevertheless, polysomnography findings are not required because "requiring laboratory information to establish a diagnosis would have set an important precedent for *DSM*" (Buysse et al., 1998, p. 1105). The precedent is apparently the suggestion that general clinicians would be unable to provide sleep disorder diagnoses without obtaining the consultation of a sleep disorder specialist who had access to the necessary laboratory equipment. The authors of *DSM-IV* indicated that requiring polysomnography would raise "several . . . questions: do laboratory studies provide unique and essential diagnostic information; do they change the role of the clinician in establishing a diagnosis; [and] could a diagnosis be made by a clinician without ready access to laboratory testing facilities" (Buysse et al., 1998, p. 1105). Frances et al. (1995) likewise indicated that the decision to not incorporate polysomnographic findings into the diagnostic criteria "reflect[s] the fact that most sleep disorder diagnoses can be made on clinical grounds without the sleep laboratory and the fact that sleep laboratory studies are expensive and not always readily available" (p. 332). Cost-benefit considerations are routinely considered in medical diagnoses, weighing such factors as the extent to which a confirmation through a laboratory test is in fact necessary, the health costs of diagnostic errors, and the financial costs of the assessment. However, at what point inability, unavailability, or expense actually justifies the failure to use more valid means of rendering psychiatric diagnoses is unclear.

The Standards of Practice Committee of the American Sleep Disorders Association appointed a task force to review the clinical indications and empirical support for polysomnography in the diagnosis of commonly encountered sleep disorders (Chesson et al., 1997) and concluded that polysomnography was essential for many of them. For example, in reference to the *DSM-IV* diagnosis of breathing-related sleep disorder (otherwise identified as sleep-related breathing disorder within the ICSD), Chesson et al. (1997) indicated that "attended polysomnography is the time-honored technique for confirming a diagnosis of sleep-related breathing disorders" (p. 424) and the American Sleep Disorders Association Indications for Polysomnography Task Force (1997) concluded that "polysomnography is routinely indicated" (p. 408) for its diagnosis.

There is in fact already a precedent in *DSM-IV* for the requirement of laboratory test findings obtained by a specialist. Laboratory tests are fundamental components of the diagnostic criteria for learning disorders and mental retardation. For example,

the essential feature of Mental Retardation is significantly subaverage general intellectual functioning . . . [and] general intellectual functioning is defined by the intelligence quotient (IQ or IQ-equivalent) obtained by assessment with one or more of the standardized, individually administered intelligence tests (e.g., Wechsler Intelligence Scales for Children—Revised, Stanford-Binet, Kaufman Assessment Battery for Children). (American Psychiatric Association, 1994, p. 39)

Psychological tests administered by a trained specialist using standardized equipment are essentially equivalent to laboratory testing. The *DSM-IV* text discussion of the diagnosis of mental retardation does note potential limitations of an IQ test (e.g., measurement error of approximately 5 points), as there are concerns and controversies concerning the validity of IQ tests (Neisser et al., 1996). Nevertheless, routine diagnoses of mental retardation by practicing clinicians in the absence of individually administered IQ tests would be substantially more problematic and controversial. Their inclusion within the criterion set is essential to maintaining current levels of reliability and validity for diagnoses of mental retardation.

The precedents established by mental retardation and learning disorders should perhaps be extended to the diagnosis of other mental disorders (Clark & Watson, 1991). Regier, Kaelber, et al. (1998) argued that the problematic variability in prevalence estimates across epidemiological studies for the anxiety and mood disorders is due in part to the absence of standardized assessment instruments. "Although diagnostic criteria are the framework for any clinical or epidemiological assessment, no assessment of clinical status is independent of the reliability and validity of the methods used to determine the presence of a diagnosis" (Regier et al., 1998, p. 114). The *DSM-III* innovation of providing relatively specific and explicit diagnostic criteria that are more likely to be assessed in a reliable manner is not realized if clinicians do not in fact adhere to the criterion sets, assessing each criterion in a comprehensive, systematic, and consistent fashion. Reliability in the diagnosis of mental disorders in clinical research is due as much to the use of structured instruments for assessment as it is to the presence of more specific and explicit criterion sets (Rogers, 1995). "Clearly, introduction of operationalized, specified, empirically derived, and standardized criteria for mental disorders in conjunction with construction of standardized structured diagnostic interviews has served to revolutionize the diagnostic process and improve reliability and validity" (Segal, 1997, p. 26). Researchers would be hard pressed to get their findings published if they failed to document that their clinical diagnoses were based on a systematic, replicable, and objective method, yet no such requirements are provided for clinical diagnoses, with the exception of mental retardation and learning disorders. Clinicians often prefer to rely on their own experience, expertise, and subjective impressions that are obtained during the course of an unstructured interview (Westen, 1997), but it is precisely this reliance on subjective and idiosyncratic clinical interviewing that undermines the reliability and ultimately the validity of clinical diagnoses (Rogers, 1995; Segal, 1997; Zimmerman & Mattia, 1999), in part by al-

lowing, if not fostering, false assumptions, attributional errors, and misleading expectations (Dawes, 1994; Garb, 1997).

A noteworthy exclusion from every discussion of laboratory and physical exam findings in the text of *DSM-IV* is references to various psychological tests and instruments that would greatly improve clinical diagnosis. It is ironic that psychological tests are included within the criterion sets for mental retardation and learning disorders, yet virtually no reference is made to them for any other mental disorder within the new sections of the diagnostic manual devoted to laboratory test findings. The discussion of laboratory instruments for the diagnosis of anxiety, mood, psychotic, and other mental disorders is confined in *DSM-IV* to measures of neurophysiology. The text of *DSM-IV* refers to specific neurotransmitters that might be involved in the pathophysiology of each mental disorder (see, e.g., American Psychiatric Association, 1994, p. 324), but no reference is made to cognitive, behavioral, or interpersonal models of pathology. Instruments that assess cognitive, behavioral, affective, or other components of psychological functioning that comprise explicitly the diagnostic criterion sets for these disorders and for which substantial research already provides specificity and sensitivity rates not obtained by the neurophysiological instruments should at least be acknowledged along with the neurophysiological measures.

## Conclusions

The issues and proposals addressed in this review cover a substantial range of possibilities, from the process by which the diagnostic manual is constructed, through the differentiation from normality, the differentiation among disorders, and the time duration covered by each diagnosis, to the means by which the disorders are diagnosed. *DSM-IV* is a compelling effort at a best approximation to date of a scientifically based, official nomenclature, but the breadth of this review's coverage is itself a testament to the extent to which the current manual is problematic and warrants revision. A consistent theme across the proposals provided herein is a move toward a more dimensional model of classification, wherein the continuum of functioning across existing diagnostic categories, across time, and into normal domains of psychological functioning is acknowledged and assessed by standardized psychological instruments. Reliable points of demarcation could be identified along these dimensions that would be optimal for different social and clinical decisions (e.g., hospitalization, insurance coverage, individual psychotherapy, or medication).

A conversion to a dimensional model for the classification of mood, anxiety, personality, and other mental disorders, using structured instruments with which to obtain quantitative scores along the respective domains of functioning, does represent a substantial shift from the format of the extant *DSM*, and other researchers may provide compelling arguments in opposition to these proposals. Hopefully, the process by which *DSM-V* is constructed will continue to be guided by empirical research in a manner that will foster innovative, productive revisions. In the meantime, the strongest recommendation to be made on the basis of this review is for researchers to continue to explore alternatives to *DSM-IV* that will ultimately inform the authors of *DSM-V* regarding which revisions to make. Progress will occur best through further critical review and through the exploration of

alternative perspectives rather than through a premature embracement of the current manual.

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