

# A Critical Examination of the Use of the Term and Concept of Comorbidity in Psychopathology Research

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**The term and concept of comorbidity has been receiving increasing currency in the psychopathology literature. Nevertheless, most uses of this term in contemporary psychopathology research do not adequately distinguish between the nature of conditions in organic medicine (which typically approximate "diseases") and those in descriptive psychopathology (which are typically "syndromes" or, more rarely, "disorders"), and blur the distinction between latent constructs and manifest indicators. Specific problems with use of the term comorbidity include its (a) application to childhood and personality disorders and (b) inconsistent usage. We conclude that, with the possible exception of its use to describe some organic mental disorders, application of the term comorbidity to psychopathological syndromes encourages the premature reification of diagnostic entities and arguably has led to more confusion than clarification.**

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We have recently observed a marked increase in the use of the term "comorbidity" in psychopathology research (e.g., Brady & Kendall, 1992; Maser & Cloninger, 1990a; Rachman, 1991). A search of the psychological and psychiatric literature reveals that the terms *comorbidity* or *comorbid* first appeared in an English or foreign-language journal abstract or title in 1984, that they did not reappear

in 1985, and that they appeared only twice in 1986. Nevertheless, the number of journal abstracts or titles containing these terms increased to 21 in 1987, 43 in 1988, 97 in 1989, 147 in 1990, 192 in 1991, 191 in 1992 and 243 in 1993.

In addition, a number of prominent researchers have recently emphasized the importance of examining and understanding the comorbidity among psychopathological conditions. For example, Lewinsohn, in his introduction to Maser and Cloninger's (1990a) book, asserted that "with meteoric speed, 'comorbidity' has emerged as the single most important concept for psychiatric research and practice. Its potential implications for theory and for treatment are just beginning to be realized" (p. ii). Maser and Cloninger (1990b) noted that "psychiatric comorbidity raises many fundamental questions about psychopathology and emerges as a test of our classification systems" (p. 4). Given the recent upsurge in use of the term and concept of *comorbidity* in psychopathology research, we believe that it is an appropriate time to take stock of the literature on comorbidity and to evaluate the application of this term to the overlap among psychopathological syndromes.

The term *comorbidity*, which derives from the medical epidemiology literature, refers to "any *distinct additional entity* [*italics added*] that has existed or that may occur during the clinical course of a patient who has the index disease under study" (Feinstein, 1970, p. 467; also see Shea, Widiger, & Klein, 1992, p. 859). Blashfield (1990) has similarly defined comorbidity as "the co-occurrence of *different diseases* [*italics added*] in the same individual" (p. 61). The increasing use of this term in psychopathology appears to have paralleled an enhanced appreciation

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of the covariation among diagnoses within a variety of domains, including childhood disorders (Abikoff & Klein, 1992; Biederman, Newcorn, & Sprich, 1991), personality disorders (Oldham et al., 1992; Widiger & Rogers, 1989), anxiety disorders (Brown & Barlow, 1992; deRuiter, Rijken, Garssen, Van Schaik et al., 1989), and mood disorders (Lewinsohn, Rohde, Seeley, & Hops, 1991), as well as between many of these domains (Hecht, von Zerssen, & Wittchen, 1990; Shea et al., 1992).

We regard this increased appreciation of diagnostic overlap as encouraging, because it appears to reflect a growing consensus that most existing psychiatric categories (i.e., as codified in DSM-III-R; American Psychiatric Association, 1987) do not conform to a simple "classical" model of categorization, in which indicators are both singly necessary and jointly sufficient for a diagnosis (Cantor, Smith, French, & Mezzich, 1980; Kendell, 1975). Two major implications of the classical model for psychiatric classification are that diagnostic categories (a) possess distinct boundaries and (b) co-occur infrequently within individuals, and covary negligibly with each other in the population. Nevertheless, unclear boundaries and extensive co-occurrence and covariation among diagnostic categories are the rule, rather than the exception, in psychopathology (Widiger & Frances, 1985).

Indeed, it has become increasingly apparent that most existing diagnostic categories conform to a "prototypal" model, in which indicators are neither singly necessary nor jointly sufficient for a diagnosis (Cantor et al., 1980; Widiger & Frances, 1985). In contrast to the classical model, the prototypal model implies that at least some diagnostic categories will (a) possess fuzzy boundaries and (b) co-occur frequently within individuals, and covary moderately or highly with each other in the population. Consequently, comorbidity among diagnoses is to be expected in many cases. Note, however, that a prototypal model does not imply that the *latent entities* underlying current diagnostic categories necessarily possess fuzzy boundaries. Thus, a prototypal model of a diagnostic category is entirely compatible with the existence of a latent taxon (i.e., a category existing in nature) (Meehl & Golden, 1982) manifested by multiple indicators (i.e., diagnostic criteria) that are fallibly related to the taxon.

The awareness that the prototypal model is optimal for most current psychiatric categories appears to have contributed to increased recognition of the extensive co-occurrence among these categories. Moreover, this

heightened awareness has spurred the adoption of polythetic criteria that more adequately reflect the probabilistic nature of the relations between diagnostic indicators and their respective categories (Widiger & Frances, 1985). Finally, this awareness appears to have led to intensified efforts to uncover etiological factors shared by covarying diagnostic categories (e.g., Cloninger, 1978).

We remain concerned, however, about the use of the term *comorbidity* in contemporary psychopathology research for several reasons. Specifically, we believe that this term has been (a) prematurely applied to psychopathological conditions in general, (b) prematurely applied to childhood and personality disorders in particular, and (c) used inconsistently. Despite the frequency with which the term *comorbidity* has been utilized in psychopathology, the use of this term has seldom been subjected to critical scrutiny. For example, although the book by Maser and Cloninger (1990a) contains 40 chapters dealing with comorbidity and its implications for mood and anxiety disorders, relatively few of the authors question the application of this term to psychopathology research.

Nevertheless, a number of the arguments we raise are not new. For example, Frances, Widiger, and Fyer (1990) delineate 10 factors that influence the rates of psychiatric comorbidity and provide examples of how this overlap can result from diagnostic artifacts, such as item overlap across categories. Caron and Rutter (1991) similarly outline potential sources of comorbidity among childhood disorders, and discuss how comorbidity can be falsely produced by detection artifacts and inappropriate diagnostic practices, such as subdividing syndromes into overly narrow subcategories. Although our critique draws from these and other contributions, we attempt to build upon and extend the arguments of previous authors in three principal ways. First, we examine the assumptions underlying the term *comorbidity* and trace the origins of the term to its roots in medical epidemiology and organic medicine. Second, we outline the distinctions among different levels of explanation in psychopathology (e.g., syndrome, disorder, and disease) and the implications of these distinctions for comorbidity, and briefly illustrate how latent variable techniques and similar approaches can help to resolve the sources of diagnostic overlap. Third and finally, we address specific limitations and problems (e.g., inconsistent usage) associated with the use of the term *comorbidity* in psychopathology research. In contrast

to others (but see Carson, 1993) who have discussed ambiguities associated with the concept of comorbidity, we contend that the very use of this term in current psychopathology research is with few exceptions problematic and potentially misleading.

We should point out that our objections regarding the use of *comorbidity* are more than semantic. Instead, we maintain that casual or imprecise use of this term, as appears to have occurred in much of the recent psychopathology literature, has been both a cause and a result of unclear thinking about diagnostic entities and their interrelations (also see Carson, 1993). Before delineating these objections, however, a review of the history of the comorbidity concept and its application to psychopathology research is necessary.

#### HISTORICAL BACKGROUND

In what appears to be the initial use of the term *comorbidity*, Feinstein (1970) introduced this concept in the context of a discussion of medical diseases that can affect the natural history of other diseases. For example, he discussed the malignant impact that diseases such as coronary artery disease and cerebral arteriosclerosis can have upon the prognosis of cancer. Kaplan and Feinstein (1974) later delineated three different meanings of comorbidity: (a) *diagnostic*, in which one disease can simulate the signs and symptoms of a coexisting disease (e.g., diabetes mellitus, which often results in excessive urination, coexisting with a renal disease that can also result in excessive urination); (b) *prognostic*, in which "an ailment predisposes, either by itself or in combination with the main disease, to the future development of adverse target events" (p. 391) (e.g., coexisting diabetes mellitus and hypertension resulting in increased risk for certain conditions compared with diabetes mellitus alone); and (c) *pathogenetic*, in which two diseases are etiologically related to one another (e.g., diabetes mellitus resulting in retinopathy). In addition, Feinstein (1970) referred to *therapeutic* comorbidity, in which a disease can influence the assessment of treatment efficacy for a coexisting disease.

It was not until recently, however, that *comorbidity* came to be applied with some frequency in psychopathology research. The *Diagnostic Statistical Manual of Mental Disorders* (3rd ed.; hereafter DSM-III; APA, 1980) instituted a number of changes that led to a marked increase in comorbidity among psychopathological conditions (Frances et al., 1990). These changes included

substantially increased coverage, increased "splitting" (as opposed to "lumping") of diagnostic categories, encouragement of multiple diagnoses, use of overlapping diagnostic criteria, and provision of separate axes for major mental disorders and personality disorders (Frances et al., 1990; Frances et al., 1992; Kendall & Clarkin, 1992; Maser & Cloninger, 1990b), thereby allowing syndromes that are phenotypically quite similar to each other (e.g., obsessive-compulsive disorder and obsessive-compulsive personality disorder) to be diagnosed concurrently. DSM-III-R appears to have further increased comorbidity by its relaxation of a large number of hierarchical exclusionary rules (First, Spitzer, & Williams, 1990) and increased adoption of polythetic diagnostic criteria (Widiger, Frances, Spitzer, & Williams, 1988). The development of structured and semistructured diagnostic interviews, which force interviewers to attend to diagnoses of subsidiary importance, have also contributed to increased rates of comorbidity in research studies (Frances et al., 1990).

Indeed, there is strong evidence for extensive comorbidity among DSM-III and DSM-III-R diagnoses (Maser & Cloninger, 1990a; Widiger & Frances, 1985). Moreover, this comorbidity is not limited to one or a few diagnostic categories. Boyd et al. (1984) found that the presence of any DSM-III syndrome increased the odds of almost any other DSM-III syndrome within that individual, suggesting at least some degree of covariation among virtually all psychopathological syndromes. Moreover, the number of individuals with two DSM-III conditions was 116.5 times greater than would be expected by chance (i.e., if there were no covariation among any DSM-III conditions). Although Boyd et al. ignored the DSM-III hierarchical exclusionary rules, they also reported substantial positive covariation between syndromes that are not hierarchically associated in DSM-III, such as antisocial personality and obsessive-compulsive disorders.

Two domains in which the degree of comorbidity appears to be particularly marked are childhood disorders (Caron & Rutter, 1991) and personality disorders (Widiger & Frances, 1985). With regard to childhood disorders, Anderson, Williams, McGee, and Silva (1987) found that of 14 children with depression, 11 had at least one coexisting disorder, and 8 had a coexisting anxiety disorder, conduct disorder, and attention-deficit hyperactivity disorder. Last, Hersen, Kazdin, Finkelstein, and

Strauss (1987) reported that, of 48 children sampled consecutively for either separation anxiety disorder ( $n = 22$ ) or overanxious disorder ( $n = 26$ ), 21 children (44%) met DSM-III criteria for both conditions. In addition, a number of children in the sample met criteria for other diagnoses, such as affective disorders and oppositional disorder. Livingston, Dykman, and Ackerman (1990) found that, of 182 children who satisfied DSM-III criteria for attention-deficit disorder, 54% met criteria for at least one other diagnosis, including affective disorder, conduct disorder, and oppositional disorder.

With regard to personality disorders, Fyer, Frances, Sullivan, Hurt, and Clarkin (1988) reported that 91% of patients with borderline personality disorder (BPD) satisfied criteria for at least one additional DSM-III Axis I diagnosis, 42% satisfied criteria for two or more diagnoses, and 13% satisfied criteria for three or more diagnoses. The results of this study are especially striking given that Axis II conditions were not assessed. Assessing Axis II conditions only, Nurnberg, et al., (1991) reported that 82% of patients with BPD met criteria for at least one additional personality disorder. Lilienfeld, VanValkenburg, Larntz, and Akiskal (1986) reported that 63% of patients with antisocial personality disorder fulfilled criteria for histrionic personality disorder, and that the same percentage of patients with histrionic personality disorder fulfilled criteria for antisocial personality disorder. In general, approximately 50% of patients with a personality disorder appear to satisfy criteria for at least one other personality disorder (Grove & Tellegen, 1991).

Thus, it seems clear that the degree of comorbidity among DSM-III and DSM-III-R categories is both substantial and pervasive. Nevertheless, despite the increasingly frequent adoption of the term comorbidity in psychopathology research, we are not convinced that any of the four meanings introduced by Kaplan and Feinstein adequately apply to the modal case in psychopathology. Specifically, as discussed in the following section, we contend that the typical use of this term in psychopathology research represents a misapplication of the "disease" concept in organic medicine to psychopathological conditions.

#### **LEVELS OF UNDERSTANDING: SYNDROMES, DISORDERS, AND DISEASES**

We find Kazdin's (1983) discussion of the distinction among syndromes, disorders, and diseases to be useful in

this context, and have adopted his phrase "levels of understanding" (p. 83) for the purposes of our argument (also see Gough, 1971, for a similar discussion of three "levels of diagnosis"). Syndromes, Kazdin notes, can be defined as constellations of signs and symptoms that covary across individuals. For the sake of simplicity, we ignore the rare cases of syndromes consisting of signs and symptoms that exhibit minimal or no covariation across individuals, but that suggest an underlying pathological state, for example, Gerstmann's syndrome (Benton, 1959).

In turn, disorders can be defined as syndromes that cannot be accounted for by other, more "basic" conditions. By more "basic" conditions, we mean those that are earlier in the causal chain, that is, that are capable of producing the clinical picture of the syndrome in question. Consider as an example a pattern of signs and symptoms consisting of persistent fear of a stimulus, immediate anxiety response to that stimulus, marked avoidance behavior, significant interference in functioning, and recognition that the fear is excessive or unreasonable. This pattern constitutes a syndrome, because these signs and symptoms tend to covary across individuals. Nevertheless, according to DSM-III-R (APA, 1987), this syndrome is considered a disorder, viz., simple phobia, only if these signs and symptoms cannot be accounted for by the presence of obsessive-compulsive disorder or post-traumatic stress disorder. In this case, DSM-III-R makes the implicit assumption that these two latter conditions are causally primary when the focus of the anxiety in these conditions is identical or closely related to that of the simple phobia. Most or all of the hierarchical exclusion rules in DSM-III and DSM-III-R appear to involve similar assumptions regarding causal primacy (Boyd et al., 1984; Frances et al., 1990).

Finally, diseases can be defined as disorders in which the underlying pathogenic processes have been identified and in which the etiology is known or reasonably well understood. Although pathology is sometimes accorded greater emphasis than etiology in definitions of disease, at least some progress has been made toward identifying the causal processes underlying traditional disease entities. Thus, sickle-cell anemia is a prototypical example of a disease because both its pathology (crescent-shaped erythrocytes containing hemoglobin S) and etiology (the presence of two autosomal recessive alleles) have been identified (Sutton, 1980). In the case of somewhat less

clear-cut diseases, such as Alzheimer's disease (primary degenerative dementia of the Alzheimer type), the primary pathologic changes (i.e., senile plaques, neurofibrillary tangles, and granulovacuolar neuronal degeneration) have been identified, whereas the understanding of the etiology is evolving but incomplete (Selkoe, 1992).<sup>1</sup>

We acknowledge that some authors in organic medicine would draw somewhat different distinctions among syndrome, disorder, and disease from those outlined by Kazdin. For example, some definitions of disorder place primary emphasis upon an alteration in the function or structure of an organ or organ system (*International Dictionary of Medicine and Biology*, 1986). Moreover, some writers have suggested that pathology, rather than pathology plus etiology, is the sine qua non for the definition of disease (Carson, 1991; Spitzer & Wilson, 1975). Nevertheless, these alterations in definition would not change the primary thrust of our subsequent arguments.

As Kazdin pointed out, our current state of knowledge with respect to the pathology and etiology of psychopathological conditions necessitates that virtually all of these conditions (with the likely exception of some organic mental disorders; see "Conclusions") be viewed as syndromes, or at best and only rarely, disorders. This level-of-understanding issue is essentially acknowledged in the introduction to DSM-III-R, where it is noted that "for most of the DSM-III-R disorders . . . the etiology is unknown. . . . DSM-III-R can be said to be 'descriptive' in that the definitions of the disorders are generally limited to descriptions of the clinical features of the disorders" (APA, 1987, p. xxiii).<sup>2</sup> In contrast, most medical conditions either meet or closely approach the criteria delineated by Kazdin (1983) for diseases (also see Meehl, 1977).

Meehl and Golden (1982) similarly have pointed out that most medical diseases are defined by a conjunction of their pathology and etiology on the one hand, and their signs and symptoms on the other. In contrast, psychopathological conditions are almost invariably defined by means of their signs and symptoms alone. Consequently, *comorbidity* is typically used in psychopathology when overlap at the descriptive or phenotypic (i.e., sign or symptom) level is the only information available, whereas this term is typically used in organic medicine to refer to overlap at both the descriptive and pathological/etiological levels (also see Gough, 1971, and Meehl,

1973, pp. 284–289, for a discussion of the differences between functional and organic entities).

We contend that the term *comorbidity* is typically utilized in the medical literature to refer to covariation among *diseases*, that is, conditions whose pathologies and etiologies are relatively well understood (also see Belfer, 1993). As noted earlier, for example, Feinstein (1970) and Kaplan and Feinstein (1974) restricted the use of this term primarily or exclusively to disease entities, such as cerebral arteriosclerosis. This practice is consistent with its usage elsewhere in organic medicine (Feinstein, 1985; *Stedman's Medical Dictionary*, 1990), as well as with definitions of comorbidity in psychiatric writings (Blashfield, 1990). Caron and Rutter (1991) have similarly defined comorbidity as "the simultaneous occurrence of two or more unrelated conditions" (p. 1063). By "unrelated," we presume that Caron and Rutter mean "different at a latent level" (otherwise, they would exclude all cases of what Kaplan and Feinstein called "pathogenetic comorbidity"). But can one determine whether two conditions are "unrelated" without a reasonably good understanding of their pathology and etiology?

When *comorbidity* is used to refer to syndromes or disorders—that is, conditions whose pathologies and etiologies are either poorly understood or not understood (as is the case with virtually all psychopathological conditions)—the extent of comorbidity becomes a largely arbitrary consequence of the signs and symptoms selected as diagnostic criteria, and thus varies as a function of changing diagnostic practices. For example, does one really want to assert that DSM-III-R has reduced the comorbidity between histrionic and borderline personality disorders by the deletion of similar diagnostic criteria in both indicator lists (Widiger et al., 1988)? What has been reduced, it seems to us, is not comorbidity, but rather the covariation between two criteria sets (i.e., manifest indicators; see following section) that bear only fallible relations to the underlying constructs of interest.

Perhaps more importantly, the typical use of this term in descriptive psychopathology conflates two different levels of explanation: (a) the diagnosis (i.e., the descriptive level) and (b) the underlying disease entity putatively—and, in most or all cases, fallibly—assessed by the diagnosis (i.e., the pathological/etiological level). In the medical literature, *comorbidity* typically embraces both of these levels, because the pathological and etiological processes giving rise to the disease are largely implicit in the

diagnosis. Thus, it is in our view reasonable to discuss the (pathogenetic) comorbidity between diabetes mellitus and retinopathy, because the underlying pathologies and causal pathways of these two conditions are reasonably well articulated. In contrast, in psychopathology, *comorbidity* is typically employed to describe overlap at the diagnostic level alone, because the pathological and etiological processes are largely or entirely unknown. We find this latter usage inappropriate because this overlap is not at the disease level, which is the level at which the application of the term *comorbidity* appears to be intended (Blashfield, 1990; Feinstein, 1985).

#### THE DISTINCTION BETWEEN LATENT CONSTRUCTS AND MANIFEST INDICATORS

More generally, the use of the term *comorbidity* in contemporary psychopathology research seems to reflect a less than adequate appreciation of the distinction between latent constructs and manifest indicators. For example, Maser and Cloninger (1990b), after reviewing evidence for the extensive comorbidity among psychopathological conditions, concluded: "It is clear that the classic Kraepelinian model in which all psychopathology is comprised of discrete and mutually exclusive diseases must be modified or rejected" (p. 12). In contrast, we would emphasize that the categories embodied in the current psychiatric classification system may not correspond to the latent entities underlying these categories and, as noted earlier, cannot legitimately be viewed as diseases given our current state of knowledge. Furthermore, the manifest indicators of two or more taxa may exhibit considerable overlap and covariation (Meehl & Golden, 1982). Thus, abandonment or alteration of the Kraepelinian model on the basis of extensive comorbidity is premature, because such overlap may indicate only that the current diagnostic nomenclature has not "carved nature at its joints" (Gangestad & Snyder, 1985).

An illustration of the potential hazards of neglecting the distinction between latent constructs and manifest indicators can be seen in a study by Young, Tanner, and Meltzer (1982). These authors used latent class analysis to examine the agreement among four diagnostic systems for schizophrenia: the Research Diagnostic Criteria, Flexible 6 system, Schneider's first-rank symptoms, and the Taylor and Abrams' criteria. Young et al. found that these four systems exhibited modest levels of covariation. Nevertheless, they also reported that these four systems

assessed the same latent entity, but at differing levels of accuracy.

The implications of Young et al.'s findings for comorbidity are intriguing. Two or more diagnoses may be comorbid solely because they are assessing the same latent entity or dimension but at different thresholds of severity (Belfer, 1993) or different levels of accuracy (although Young et al. found evidence only for the latter). Thus, social phobia and avoidant personality disorder, which covary substantially (Turner & Beidel, 1989), may simply represent two different thresholds of severity on a single latent dimension of social anxiety. The same may hold for the condition of "double depression" (Keller & Shapiro, 1982), in which a major depressive episode is superimposed upon dysthymia. In many cases, these two syndromes may simply represent different points on a shared underlying continuum. When the clinical state of the dysthymic patient worsens, an essentially arbitrary diagnostic threshold may be exceeded, resulting in comorbidity between dysthymia and major depression.

Comorbidity can also arise from a quite different source, namely the covariation between the error components associated with two or more diagnoses. These error components, in turn, may stem from a variety of method factors that can affect different diagnoses. In the psychometric literature, error variance is traditionally considered to be any part of a measure's overall variance that is not due to the latent construct(s) of interest. Given that signs and symptoms can be viewed as fallible indicators of latent diagnostic entities, they are susceptible to the effects of measurement error, as are items on personality questionnaires or intelligence tests. Although measurement error often is assumed to have random effects on individuals' scores, at times such error may reflect method factors that exert a shared influence on the signs and symptoms of various diagnoses. For example, particular raters (e.g., parents or teachers) may hold certain biases or differ in their thresholds for endorsing behaviors indicative of a variety of syndromes. Alternatively, certain raters may possess implicit notions regarding the covariation among certain psychopathological conditions (e.g., attention-deficit hyperactivity disorder and conduct disorder). Each of these rater effects may contribute to the comorbidity among some syndromes. Fortunately, latent variable methods are useful for disentangling "trait" factors (e.g., latent diagnostic entities) from "method" factors (e.g., rater effects), as well as for distinguishing

among trait factors themselves (for an example, see Cole, 1987).

We should point out that many of our arguments have been anticipated by other authors. Caron and Rutter (1991), for example, refer to "artifactual comorbidity" (p. 1070) and "apparent comorbidity" (p. 1073) when discussing cases in which the co-occurrence of two conditions is a consequence of factors such as referral bias, overlapping diagnostic criteria, or inappropriate splitting of a disease entity into two or more conditions (also see Maser & Cloninger, 1990b). Frances et al. (1990) similarly refer to "artifactual comorbidity" (p. 58) and note that

*To say that conditions are comorbid in a given patient means no more than that the defining descriptive features tend to associate with one another. We must not reify the DSM syndromes into distinct disease entities and assume that comorbidity means the presence of two different even if presumptively related diseases. Our high rates of comorbidity are just as likely to emerge from simply the descriptive overlap included in DSM-III and DSM-III-R. (p. 57)*

They proceed to assert that "comorbidity determined by descriptive studies can never be understood until information on course, pathogenesis, family loading, and treatment response provides an independent means of determining the causal relationships underlying surface associations" (p. 56). In principle we concur, but wish to go further to argue that overlap among syndromes should not be referred to as comorbidity when such overlap is at a purely descriptive level.

#### **SPECIFIC PROBLEMS WITH THE USE OF COMORBIDITY**

##### **Childhood and Personality Disorders**

Although we question the use of *comorbidity* in most areas of psychopathology research, we believe that the problematic use of this term is most clearly illustrated by diagnostic overlap in two domains: childhood disorders and personality disorders.

With regard to childhood disorders, it has long been noted that development in childhood is characterized by a process of increasing cognitive and emotional differentiation and complexity, that is, the orthogenetic principle (Werner, 1948; also see Cichetti & Schneider-Rosen, 1984). Consequently, comorbidity in childhood may be

a function of developmental level; specifically, children with comorbid syndromes may be at a stage in which the different developmental processes underlying these syndromes have yet to achieve full differentiation. A failure to appreciate the implications of the orthogenetic principle may partially explain the particularly high rates of comorbidity among many childhood disorders. For example, the extensive comorbidity among oppositional defiant disorder, conduct disorder, and attention-deficit/hyperactivity disorder in childhood (Hinshaw, 1987; Lilienfeld & Waldman, 1990) may reflect the presence of a relatively undifferentiated group of externalizing problems that have not yet diversified into distinct developmental trajectories (Loeber, 1990). We should also note that, although the orthogenetic principle has generally been discussed with regard to children and adolescents, some developmental psychopathologists might make a similar point for certain adult conditions.

With regard to personality disorders, it is conceivable that most or all of the personality disorder categories in DSM-III and DSM-III-R have resulted from a series of quasi-arbitrary demarcations in multidimensional space (Eysenck, Wakefield, & Friedman, 1983; Grove & Tellegen, 1991). If the latent structure of these personality disorders were in fact dimensional, and if the dimensions on which these demarcations are made were to some extent intercorrelated (which seems extremely likely, at least at the phenotypic level; see Cloninger, 1987, and Tellegen & Waller, in press), then the use of categorical diagnoses will ipso facto result in comorbidity, even though the true state of affairs does not involve covariation among different diseases (or perhaps even disorders) at all (Caron & Rutter, 1991). Thus, the use of *comorbidity* to describe the covariation among personality disorders, and perhaps many other psychopathological syndromes (Eysenck et al., 1983), appears to violate the spirit of the introduction to DSM-III-R: "There is no assumption that each mental disorder is a discrete entity with sharp boundaries (discontinuity) between it and other mental disorders, or between it and no mental disorder" (APA, 1987, p. xxii). Moreover, even if some personality disorders were produced by latent taxa, the true number of these taxa, as well as their boundaries, are unknown. Consequently, in the domain of personality disorders, we regard the use of *comorbidity* as misleading, because it implies (a) adoption of a categorical model that may be unwarranted for many or all of these conditions, and (b)

that the number and boundaries of these categories, if they exist, are known. Such use of this term thus implicitly prejudices two of the central issues that have bedeviled the study of personality disorders for decades.

Some of the difficulties arising from application of the comorbidity concept to personality disorders are illustrated by a recent exchange (Oldham et al., 1992; O'Boyle & Holzer, 1992). Oldham et al. suggest that patients with two or fewer personality disorder diagnoses be classified as having a "focal" personality disorder, whereas patients with three or more personality disorder diagnoses be classified as having an "extensive" personality disorder. In response, O'Boyle and Holzer, arguing that the term *extensive personality disorder* is pejorative and potentially ambiguous, suggested that the term *diverse* be substituted for *extensive*. In our view, however, such a discussion is premature, because it implicitly presupposes that the DSM-III-R personality disorder categories correspond to the underlying state of nature. It is entirely conceivable, for example, that patients with *extensive* (diverse) personality disorder possess a single condition that is manifested in multiple domains that cut across several DSM-III-R personality disorder categories.

#### **Inconsistent Usage: Co-occurrence Versus Covariation**

An additional problem with the term *comorbidity* has been an inconsistent usage that potentially confuses two quite different concepts. In some cases, *comorbidity* has been utilized to denote *co-occurrence* among diagnoses (e.g., August & Garfinkel, 1990; du Fort, Newman, & Bland, 1993; Fulop et al., 1987). In other cases, it has been utilized to denote *covariation* among diagnoses (Cole & Carpenter, 1990; Lewinsohn et al., 1991; Rudd et al., 1993). By co-occurrence, which is sometimes referred to as "dual diagnosis" (Belfer, 1993), we mean the simultaneous presence in an individual of two diagnoses, which are not necessarily correlated to an appreciable extent within the population. Thus, an individual may fulfill criteria for both somatization disorder and drug dependence, although these two diagnoses covary negligibly across individuals (Boyd et al., 1984). Co-occurrence among diagnoses may be informative for certain purposes. For example, patients with somatization disorder may have a poorer prognosis or be more refractory to treatment if they possess an additional diagnosis of drug dependence (this would be analogous to what Kaplan and Feinstein, 1974, refer to as prognostic comorbidity).

Thus, co-occurrence—even in the absence of covariation—may be relevant to how one condition moderates the prognosis or treatment of another. Nevertheless, this meaning of the term should not be confused with covariation, by which we mean the tendency of certain diagnoses to co-occur more often than expected by chance. Only the third form of comorbidity discussed by Kaplan and Feinstein (1974)—pathogenetic—appears to refer explicitly to covariation.

Although these two meanings of *comorbidity* appear to have often been used interchangeably, they carry substantially different implications in certain cases. For example, a dramatic rise in the base rate of a condition (e.g., from 50% to 95%) will, *ceteris paribus*, decrease covariation with other conditions due to restriction of range, but increase co-occurrence with other conditions (similar effects could result from a dramatic decrease in a condition's diagnostic threshold). In addition, increased diagnostic co-occurrence, but not increased diagnostic covariation, can be produced by selection factors such as Berksonian bias (Berkson, 1946) and clinical selection bias (du Fort et al., 1993), which involve a tendency for individuals who seek treatment to possess multiple diagnoses. Berksonian bias is purely mathematical and results from the fact that an individual with two conditions can obtain treatment for either condition (du Fort et al., 1993). Clinical selection bias, in contrast, results from the increased probability of treatment seeking for individuals with one condition because of the presence of another condition (du Fort et al., 1993). For example, individuals with antisocial personality disorder may be unlikely to seek treatment unless they have a concurrent depression.

Thus, these two meanings of *comorbidity* (co-occurrence versus covariation) possess very different, and in some cases opposite, implications. An example of the confusion arising from an inconsistent use of *comorbidity* can be found in an article by Belfer (1993), who asserts that "patients with comorbid conditions may be more likely to seek or enter treatment. Such a selection bias would be likely to yield spurious associations between substance abuse and comorbid psychiatric disorders" (p. 72). But such a selection bias would lead only to an increase in the reported co-occurrence between substance abuse and other conditions, and not necessarily to an increase in their reported association (i.e., covariation). Because of the potential confusion regarding the two uses of *comorbidity*, we recommend that authors specify



whether they are referring to co-occurrence or covariation when discussing diagnostic overlap.

## CONCLUSIONS

In summary, we have several objections to the use of the term and concept of *comorbidity* in contemporary psychopathology research. First, this term implies a model of disease and a corresponding level of understanding that is absent for the vast majority of psychopathological entities. Because the pathologies and etiologies of virtually all psychopathological conditions are presently poorly understood, the use of this term implicitly prejudges the question of whether two covarying conditions represent discrete latent entities. We find it premature and potentially misleading to label the covariation among diagnostic categories as *comorbidity* when the goal of much of psychological and psychiatric research has been to determine whether this covariation can be more parsimoniously explained by a single disease entity to begin with. Second, this term conflates the latent-construct and manifest-indicator levels of psychopathological entities and their interrelations. Third, this term does not account for developmental trends in the differentiation of latent entities, and implicitly assumes a categorical model of diagnosis that may be inappropriate for personality disorders and perhaps many other conditions. Fourth and finally, an inconsistent use of this term to refer to both diagnostic co-occurrence and diagnostic covariation is a potential source of confusion regarding diagnostic overlap.

As noted earlier, the seeds of many of our arguments can clearly be found in the writings of other authors (e.g., Belfer, 1993; Caron & Rutter, 1991; Carson, 1993; Frances et al., 1990; Kendall & Clarkin, 1992). Young (1983), for example, contended that researchers ideally should demonstrate that the internal structure of a diagnostic category is valid before conducting studies of that category's external validity, such as studies of its covariation with other diagnostic categories. Somewhat similar arguments have been made by Skinner (1981). The implications of Young's argument for this article seem clear: it is premature to discuss comorbidity among diagnoses until their internal validities have been reasonably well established (e.g., until studies have been conducted to ascertain whether their latent structure is categorical or dimensional).

We suspect that critics will find our arguments to be

vulnerable on two major grounds. First, skeptics might contend that we are quibbling over semantics. We think not. In our experience as educators, for example, we have observed that the use of the term comorbidity appears to have led some students to infer that diagnostic covariation reflects the coexistence of discrete disease entities. This premature reification of "open" into "closed" concepts (Meehl, 1977, 1986) can, in our view, lead to a dangerous confusion of levels of explanation (the perils of inappropriately applying a strictly empiricist methodology to the enterprise of psychiatric classification have been eloquently discussed by Faust & Miner, 1986). As Frances et al. (1990) noted with respect to the comorbidity between anxiety and mood disorders, "[some observers] may naively believe . . . that anxiety and depressive disorders are necessarily separate, distinct, and comorbid rather than holding open the possibility that these are the related surface manifestations of underlying unitary syndromes" (p. 57). If the Whorfian hypothesis of linguistic relativity has merit, which now appears to be the case (Hunt & Agnoli, 1991), then the use of imprecise language may lead to correspondingly imprecise thinking. In addition, we believe that the use of this term is in part a consequence of unclear thinking concerning differences in our understanding of conditions in organic medicine on the one hand and psychopathology on the other.

Perhaps more importantly, our arguments go considerably beyond semantics in their implications for the methodological approaches—such as latent class analysis (Young, 1983; Young, Tanner, & Meltzer, 1982), Meehl and colleagues' taxometric methods (Lenzenweger & Korfine, 1992; Meehl & Golden, 1982), and admixture analysis (Cloninger, Sigvardsson, von Knorring, & Bohman, 1984; Lenzenweger & Moldin, 1990)—needed for a better understanding of the sources of comorbidity. Although we do not regard any of these approaches as panaceas, we believe that greater familiarity with these and related techniques will encourage investigators to more closely examine the latent structure of psychopathological entities.

Second, critics might contend that we have exaggerated and oversimplified the concept of *disease* in organic medicine. Specifically, one could argue that a number of medical diseases (e.g., essential hypertension) have poorly understood pathologies, etiologies, or both, and that the distinction we have drawn between conditions in organic medicine and psychopathology lies more in degree than

in kind. In response, we acknowledge that the pathologies and etiologies of a number of medical diseases are poorly understood, but maintain that the use of *disease* to describe conditions for which neither the pathology nor etiology has been identified is imprecise and colloquial. Instead, we contend that the term *disease* refers to an ideal type that is approximated by most medical conditions but by very few psychopathological conditions. Thus, we grant the possibility that the distinction we have made between psychopathological and medical syndromes is primarily one of degree, but contend that this degree is so large in magnitude that it renders most arguments for the use of the term *comorbidity* in psychopathology essentially moot.

Are there *any* cases in which the use of *comorbidity* might currently be defensible in psychopathology? Although we are reluctant to proffer a definitive answer to this question, we believe that a plausible argument could perhaps be made for the application of this term to the overlap between certain organic mental disorders and other psychopathological syndromes. Both Alzheimer's disease and multi-infarct dementia, for example, are characterized by readily identifiable pathophysiologies and at least partially understood etiologies (Kaplan & Sadock, 1985). Even here, however, potential problems of interpretation arise. For example, there is evidence that multi-infarct dementia covaries with depression (Reding, Haycox, & Blass, 1985). Nevertheless, in some cases it may be unclear whether this depression represents an organic mood syndrome resulting from minor strokes (APA, 1987) or a secondary reaction to an awareness of cognitive deficits. Consequently, referring to the covariation between multi-infarct dementia and depression as *comorbidity* may imply a deeper level of understanding than is warranted. Moreover, the etiologies of certain organic mental disorders, such as organic personality disorder and organic mood disorder, are often largely or entirely unknown (APA, 1987, pp. 162–163).

We therefore conclude that the application of the term and concept of *comorbidity* to psychopathological syndromes is almost invariably misleading and arguably has led to more confusion than clarification. We thus recommend that, with the possible exception of certain organic mental disorders, the term *comorbidity* be avoided in psychopathology research, and that the terms *diagnostic co-occurrence* and *diagnostic covariation* instead be used to refer to the two major types of *comorbidity* discussed here. Unlike *comorbidity*, these terms do not connote an associa-

tion among disease entities, and refer to overlap at the descriptive, rather than pathological/etiological, level. Let us make clear, however, that we strongly encourage research aimed at (a) understanding and validating the latent structure of covarying psychopathological conditions and (b) clarifying the underlying factors that produce this covariation. We are not convinced, however, that most commonly employed approaches to these two issues are adequate, and intend to outline a conceptual and methodological framework for addressing these issues in a forthcoming article.

Finally, let us also make clear that we are not advocating a return to a classical model of categorization for psychopathological syndromes. A prototypal model clearly provides the most realistic representation of most *current* psychopathological conditions and their interrelations (Cantor et al., 1980). Nevertheless, the adoption of a prototypal model should not be interpreted to mean that a classical model could not provide a better representation of psychopathological categories and their interrelations in the future. A prototypal model, with its concomitant high levels of *comorbidity* (Widiger & Frances, 1985), may reflect nothing more than our limited understanding concerning the underlying structure and etiology of diagnostic entities. As Meehl (1977, 1986) has pointed out, virtually all diagnostic categories are best conceived of as “open” concepts (also see Pap, 1953) characterized by indefinitely extensible indicator sets, fallible relations between these indicators and the underlying construct, and an unclear “inner nature.” Nevertheless, open concepts can later become closed if research succeeds in clarifying their inner nature. In the case of a diagnostic entity, this inner nature can be viewed as its underlying pathology and etiology. Thus, just as it would be premature to return to a classical model for the psychiatric classification system of today, it would be equally premature (e.g., Carson, 1991, 1993; Maser & Cloninger, 1990b) to invoke *comorbidity* as a reason for dismissing this model for the psychiatric classification system of tomorrow.

## NOTES

1. The definition of disease may also require a value judgment concerning factors such as whether a patient's dysfunction is harmful or requires treatment (e.g., Gorenstein, 1984; Wakefield, 1992); but see Kendell (1975) for a dissenting view. Our position is that, even if the arguments of Gorenstein, Wakefield, and others possess

merit, the conditions specified by Kazdin—relatively well understood pathology and etiology—are nevertheless necessary, but not sufficient, for a syndrome to be considered a disease.

2. Note, however, that DSM-III-R uses the term *disorder* in a considerably broader fashion than does Kazdin.

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