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Comorbidity Between and Within Childhood Externalizing and Internalizing Disorders: Reflections and Directions

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The term and concept of "comorbidity" has been mired in controversy, although there is little question that the existence of covariation among psychiatric diagnoses poses significant challenges to current models of psychiatric classification and diagnosis. The papers in this *Special Section* underscore a number of important issues relevant to the comorbidity between and within childhood externalizing and internalizing disorders, and illustrate both methodological and substantive reasons for such comorbidity. Weiss, Susser, and Catron's distinction among common, broad-band specific, and narrow-band specific features provides a helpful framework for understanding the comorbidity of childhood externalizing and internalizing disorders (B, Weiss, K. Susser, & T. Catron, 1998). Hierarchical models of psychopathology help to dissolve the distinction between "splitters" and "lumpers" and point to variables that may elucidate the etiology of externalizing and internalizing disorders.

KEY WORDS: comorbidity; externalizing disorders; internalizing disorders; classification; diagnosis; psychopathology.

Ever since the term "comorbidity" was coined by the late Yale epidemiologist Alvin Feinstein over three decades ago (Feinstein, 1970), its application to psychopathology has sparked disagreement and at times sharp controversy. In particular, there has been a lack of consensus regarding the proper uses of the term comorbidity, its causes, and its implications for psychiatric classification and diagnosis. Some authors (e.g., Lilienfeld, Waldman, & Israel, 1994) have argued that the term "comorbidity" is meaningful only in the context of well-validated disease entities, that is, conditions in which pathology and etiology are reasonably well understood. More recently, Meehl (2001) maintained that this term is meaningful only in the context of taxonic conditions, that is, conditions that are underpinned by a discrete causal agent. According to Meehl, attaching the term "comorbidity" to dimensional conditions is inappropriate, because it is not meaningful to speak of the overlap between two diagnostic classes created by establishing scientifically arbitrary cutting scores on one or more dimensions (see also Waldman & Lilienfeld, 2001). More broadly, concerns have been raised that the term and concept of comorbidity have been misapplied. Indeed, when I spoke with Alvin Feinstein in the early 1990s in preparation for an article my colleagues and I were writing on comorbidity, he agreed that this term was being overused and that he had never intended it to be applied indiscriminately to all cases of diagnostic overlap.[3]

Whatever the conceptual problems with the term "comorbidity," we appear to be stuck with it for the foreseeable future. And whatever the conceptual problems associated with this term, there can be little dispute that the presence of comorbidity poses a serious challenge to existing classification systems and etiological models of psychopathology. Such comorbidity indicates that "classical" models of classification, which are characterized by discrete and mutually exclusive categories with few or no intermediate cases, apply to few, if any, domains

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[3]In the remainder of this paper, I use the term "comorbidity" to refer to covariation among diagnoses across individuals rather than co-occurrence among diagnoses within individuals. The distinction between these two frequently conflated meanings of comorbidity is important on methodological and substantive grounds (Lilienfeld et al., 1994; see also Keiley et al., 2003).

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of psychopathology (see also Maser & Cloninger, 1990). Whether this fact indicates that existing models of classification have not yet carved nature at its joints and are therefore flawed, as Jerome Kagan and others contend, or whether it indicates that nature is intrinsically messy and that existing models of classification accurately mirror this messiness, is unclear. The provocative papers in this *Special Section* of the *Journal of Abnormal Child Psychology* illustrate some of the conceptual and methodological challenges posed by comorbidity in the domain of childhood externalizing and internalizing disorders, although they bear significant implications for psychiatric classification and diagnosis in general.

POTENTIAL SOURCES OF COMORBIDITY

As readers familiar with this literature are aware, several authors (e.g., Caron & Rutter, 1991; Frances, Widiger, & Fyer, 1990; Klein & Riso, 1993; see also Keiley, Lofthouse, Bates, Dodge, & Pettit, 2003) have delineated numerous potential reasons for comorbidity in the domain of psychopathology, and I will not reiterate all of these reasons here. Nevertheless, it may be useful to outline several potential sources of comorbidity that are particularly relevant to the covariation between internalizing and externalizing childhood disorders. In doing so, I'll focus primarily on what Angold, Costello, and Erkanli (1999) termed *heterotypic comorbidity*, that is covariation between disorders in different diagnostic classes. As Angold and his colleagues noted, heterotypic comorbidity is typically more interesting from a substantive standpoint than is homotypic comorbidity, that is, covariation between disorders in the same diagnostic class (see also Youngstrom, Findling, & Calabrese, 2003), because the former type of comorbidity is more difficult to explain in terms of criterion overlap and other methodological artifacts.

If Disorder A covaries significantly with Disorder B across individuals, what are the most likely explanations for such comorbidity? To oversimplify matters somewhat, there are two broad classes of explanations, methodological and substantive—and the papers in this *Special Section* touch on one or both of these sets of explanations.

On the methodological front, Disorders A and B may overlap because of shared diagnostic criteria. Second, Disorders A and B may seemingly overlap because of method covariance resulting from shared modes of assessment. Third, Disorders A and B may overlap because of referral biases. Such biases, which are often insufficiently appreciated in psychopathology research, include Berksonian bias (Berkson, 1946) and clinical selection bias (e.g., DuFort, Newman, & Bland, 1993). Berksonian bias is a purely mathematical consequence of the fact that an individual with two disorders can obtain treatment for either disorder (see McConaughy & Achenbach, 1994, for a further discussion), whereas clinical selection bias, which can be thought of as the "straw that breaks the camel's back" phenomenon, results from the fact that individuals with two disorders may be often be especially impaired and therefore more likely to seek treatment than are individuals with only one disorder. Although some authors (e.g., McConaughy & Achenbach, 1994) have not clearly distinguished Berksonian bias from clinical selection bias, these two biases stem from different sources and are worth differentiating on methodological and theoretical grounds.

Turning to substantive reasons, Disorder A may cause or predispose to Disorder B, or Disorder B may cause or predispose to Disorder A. These two possibilities correspond to what Kaplan and Feinstein (1974) termed *pathogenetic comorbidity*, whereby one condition directly causes or contributes to another. A more interesting third possibility is that Disorder A and B are influenced by shared etiological factors, such as higher-order dimensions of personality or temperament.

Certainly, there are other potential reasons for comorbidity that overlap with those already presented (see Klein & Riso, 1993), such as one disorder representing a subgroup of another disorder or the diagnostic classification system attaching separate labels to two different manifestations or *formes frustes* of the same underlying condition. This lattermost explanation is reminiscent of Youngstrom et al.'s point that bipolar disorder is characterized by both internalizing and externalizing symptoms and can therefore produce the appearance of comorbidity in categorical studies of such symptoms (Youngstrom et al., 2003).

DECONSTRUCTING COMORBIDITY BETWEEN AND WITHIN CHILDHOOD EXTERNALIZING AND INTERNALIZING DISORDERS

How do these different potential explanations for comorbidity fare when considering the overlap between internalizing and externalizing disorders? Let us begin by considering primarily methodological reasons for comorbidity.

Methodological Reasons

The possibility of method covariance causing or contributing to observed comorbidity rates warrants careful consideration, and several of the papers in this *Special Section*, particularly those of Youngstrom et al. (2003)

and Beyers and Loeber (2003), bear either directly or indirectly on this issue. It is possible, for example, that raters can overestimate the covariation between internalizing and externalizing disorders because of implicit personality theories regarding the association between these disorders, or what Guilford (1954) termed *logical errors* (which are sometimes loosely but erroneously referred to as halo effects). If so, it may be worth considering the possibility that internalizing disorders, externalizing disorders, or both, exert *suppressor effects* (see Nunnally, 1967) in studies of childhood psychopathology. That is, if method covariance is operating, controlling statistically for measures of one set of disorders could *increase* the relations between measures of the other set of disorders and relevant outcome measures, because the variance shared between these two sets of measures may be acting to decrease discriminant validity (see Keiley et al., 2003, for a related point). Of course, in considering the possible role of method covariance, it is essential to disentangle the role of unique information sources *per se* from that of unique behaviors that are observable in different settings.

Another potential methodological reason for comorbidity, as we have seen, is the presence of shared diagnostic criteria. Nevertheless, this problem seems unlikely to account for most cases of heterotypic comorbidity, such as that between internalizing and externalizing disorders, because the amount of criterion overlap between most disorders in different diagnostic classes is minimal. Moreover, as Nicholls, Licht, and Pearl (1982) pointed out in an undeservedly neglected paper on conceptual quandaries in personality assessment, removing overlapping criteria or items in conducting analyses frequently creates more problems than it solves. Because overlapping diagnostic criteria are often central to the constructs of interest, removing them often results in incomplete or atypical representations of such constructs.

Although Berksonian bias and other selection biases can also contribute to comorbidity in certain cases, such biases cannot provide an adequate or comprehensive explanation for the covariation between externalizing and internalizing disorders. As Angold et al. (1999) noted in their combined narrative and meta-analytic review of comorbidity, high levels of covariation are observed between internalizing and externalizing disorders even in community samples. For example, they found that in community samples attention-deficit/hyperactivity disorder (ADHD) and anxiety cooccurred at an odds ratio of 3.0, while conduct disorder and depression cooccurred at an odds ratio of 6.6. In their paper in this *Special Section*, Beyers and Loeber (2003) summarized some of the evidence demonstrating substantial levels of covariation between conduct problems and depression even in nonclinical (specifically, community) samples. So although referral biases may increase the levels of comorbidity between internalizing and externalizing disorders in some studies, the fact that one finds such comorbidity in nonclinical samples strongly suggests that it is more than a methodological mirage (see also Caron & Rutter, 1991).

Substantive Reasons

What about more substantive explanations for the comorbidity between internalizing and externalizing disorders? First, it is possible that internalizing disorders play a causal role in the genesis of externalizing disorders. For example, depression might in some cases impair individuals' concern about the adverse consequences of their actions, thereby increasing their risk for certain forms of antisocial behavior (Capaldi, 1991). The data Beyers and Loeber (2003) presented indicating that depression serves as an independent risk factor for later conduct problems is consistent with this possibility. If Beyers and Loeber are correct, depression could exert a main effect on rates of conduct problems or it might instead interact statistically with certain dispositions that increase risk for antisocial behavior, such as impulsivity, thereby exerting a causal role only in the presence of such dispositions.

The notion of internalizing disorders contributing to externalizing disorders harkens back to the old and perhaps prematurely abandoned concept of "neurotic psychopathy" (Karpman, 1941; see also Lykken, 1995), that is, chronic antisocial behavior resulting from anxiety, guilt, and overcontrol of impulses. A dimension corresponding to neurotic psychopathy was identified in several factor analyses of adolescent data in the 1960s by Herbert Quay and his colleagues (e.g., Peterson, Quay, & Tiffany, 1961; Quay, 1964), although the existence of this dimension does not resolve the issue of causal directionality between neurotic traits and antisocial behavior.

One problem with the neurotic delinquency concept is that in at least some studies (e.g., Walker et al., 1991) anxiety has been found to play a *protective* role among children with conduct problems. Although the evidence pertaining to this issue is not entirely consistent, there is at least some indication that among conduct-disordered children, the presence of cooccurring anxiety is an indicator of a less severe condition. This finding suggests that contrary to what some authors have suggested or implied (e.g., Dallam et al., 2001), comorbidity is not necessarily a marker of greater severity (see also Keiley et al., 2003) and that two disorders, like two heads, may sometimes be better than one.

A second possibility is that externalizing disorders may in some cases contribute to internalizing disorders. For example, following from the reasoning of Fowles (1987) and others (e.g., Tellegen, 1982) in the personality literature, Frick, Lilienfeld, Ellis, Loney, and Silverthorn (1999) hypothesized that antisocial behavior tends to give rise to recurrent state anxiety. Specifically, individuals who exhibit antisocial behavior are often confronted with adverse life consequences, such as legal difficulties, family conflict, and academic problems. If chronic, such antisocial behavior may produce repeated episodes of state anxiety, which then might be difficult or impossible to distinguish from the trait anxiety observed in most anxiety disorders (see also Keiley et al., 2003, for a discussion of how externalizing problems may lead to depression, anxiety, and other internalizing symptoms, perhaps through the mediating influence of peer rejection). This possibility, which remains conjectural, could be tested in part by longitudinal studies of the temporal ordering of antisocial behavior and anxiety, although even these studies would likely not be definitive in terms of causal primacy.

We next turn to the third major substantive explanation for comorbidity, namely that internalizing and externalizing disorders stem in part from the same underlying causal factors, such as higher-order dimensions of personality or temperament. As Weiss, Susser, and Catron (1998) argued, it may be useful to conceptualize childhood disorders in terms of three levels of generality and specificity: (1) *common features*, which distinguish internalizing and externalizing disorders from normality, (2) *broad-band specific features*, which distinguish internalizing disorders from externalizing disorders, and (3) *narrow-band specific features*, which discriminate disorders within the internalizing and externalizing categories.

What Weiss and his colleagues termed common features would account at least partly for the covariation between internalizing and externalizing disorders. O'Connor, McGuire, Reiss, Hetherington, and Plomin (1998) showed that depressive symptoms and antisocial behaviors share a common genetic liability that accounts for approximately 45% of their observed covariation. They also found that both shared and nonshared environmental influences further contribute to this covariation.

Several broad-band dimensions are promising candidates for common factors. For example, both internalizing and externalizing disorders may share a disposition toward what Tellegen (1982) and his students (e.g., Watson & Clark, 1984) termed Negative Emotionality, a pervasive disposition to experience unpleasant affective states of many kinds, such as guilt, anxiety, mistrust, and irritability. This higher-order dimension of emotional maladjustment may characterize many or most childhood disorders and constitute a causal risk factor for many of these disorders. Keiley et al.'s finding (Keiley et al., 2003) that children with a different temperament (that is, with high levels of Negative Emotionality) were rated by mothers as having higher rates of comorbidity is consistent with this possibility. Weiss and his colleagues proposed both low self-esteem and excessive self-focus as likely candidates for common features, although one could argue that both of these characteristics are best conceptualized as lower-order dimensions of a more pervasive Negative Emotionality factor (see Watson & Clark, 1984).

In addition, a number of variables may distinguish internalizing from externalizing disorders and therefore represent what Weiss and his colleagues term broad-band specific features. On the basis of confirmatory factor analyses of data from the National Comorbidity Survey, Krueger (1999) contended that the comorbidity among many Axis I disorders in the adult literature can be explained by the presence of two moderately correlated internalizing and externalizing dimensions. The same may hold for many Axis I conditions in the childhood literature, although the question of which broad-band specific features best distinguish externalizing from internalizing disorders remains unresolved. Keiley et al. (2003) found that that child unadaptability was significantly related to internalizing symptoms, although it also predicted low levels of externalizing symptoms. Keiley et al. also reported data from both their research team and that of previous investigators suggesting that peer rejection is preferentially tied to externalizing disorders, whereas peer neglect is preferentially tied to internalizing disorders. The relations among these diverse indicators of internalizing disorders require clarification. For example, the possibility that unadaptability and peer rejection both stem from a shared source trait (Cattell, 1950), such as high levels of introversion or high levels of Constraint (Tellegen, 1982), warrants further investigation.

Other authors, including Krueger (1999) and his colleagues, have suggested that a broad dimension corresponding to behavioral disinhibition (see also Gorenstein & Newman, 1980) is relatively specific to externalizing disorders. Keiley et al.'s finding (Keiley et al., 2003) that resistance to control was more closely tied to externalizing than to internalizing behaviors is broadly consistent with Krueger's contention, as children who are behaviorally disinhibited (and therefore prone to weak impulse control) are presumably especially difficult to manage. The hypothesis that a higher-order behavioral inhibition factor is a source trait underpinning most or all externalizing disorders is worth examining using not only self-report measures, but also putative laboratory and biological correlates of behavioral disinhibition, such as performance

on go-no go tasks (e.g., Newman & Kosson, 1986) and measures of serotonin metabolites (e.g., Coccaro, 1989).

This recommendation is consistent with Youngstrom et al.'s concluding suggestion (Youngstrom et al., 2003) to place greater emphasis on what Gottesman and Shields (1982) termed *endophenotypic markers*, which are presumably more closely tied than are traditional phenotypes to underlying etiological processes. It should be noted, however, that the assumption that laboratory tasks are more closely tied to underlying etiological processes than are the signs and symptoms of disorders (e.g., see Kihlstrom, 2002) is just that, an assumption. For example, some laboratory indices of attention may lie further downstream causally from underlying etiological processes than do the signs and symptoms of the disorders (e.g., ADHD) themselves, because such indices may be fairly distal consequences of poor attention and low motivation.

Finally, we arrive at the level of narrow-band specific features, which differentiate specific disorders within the broader internalizing and externalizing classes. The data here are relatively sparse, although Zinbarg and Barlow (1996), for example, have conducted methodologically sophisticated analyses in the adult literature examining the lower-order variables that best distinguish among different anxiety disorders. According to Zinbarg and Barlow, the major DSM anxiety disorders conform to a hierarchical model with a broad general factor, perhaps corresponding to Negative Emotionality, at the apex. This higher-order factor, their findings suggest, coexists with various lower-order factors that distinguish among the specific anxiety disorders in the DSM. For instance, Zinbarg and Barlow found that the lower-order dimension of anxiety sensitivity (which is essentially equivalent to "fear of fear") is relatively specific to panic disorder, whereas the lower-order dimension of interpersonal fear is relatively specific to social phobia.

The tripartite model of anxiety and depression (Clark & Watson, 1991), which posits that depression and anxiety share a high Negative Emotionality component but can be differentiated on the basis of low Positive Emotionality (which is specific to depression) and high Physiological Arousal (which is specific to anxiety), is another important step toward delineating narrow-band specific features among childhood internalizing disorders. This model has been extended to children (e.g., Joiner et al., 1999; Joiner, Catanzaro, & Laurent, 1996) and holds promise for distinguishing among childhood internalizing disorders.

Regrettably, less progress appears to have been made along these lines with childhood externalizing disorders, perhaps in part because some of the existing diagnostic categories in this domain, particularly conduct disorder, appear to be highly heterogeneous etiologically (e.g., White, Bates, & Buyske, 2001; see also Youngstrom et al., 2003). Nevertheless, findings demonstrating that adolescents with conduct disorder and those with ADHD can be differentiated on the basis of measures of autonomic functioning (e.g., electrodermal responding, cardiac preejection period; see Beauchaine, Katkin, Strassberg, & Snarr, 2001) may represent a promising first step in this direction.

CONCLUDING COMMENTS

Hierarchical models of comorbidity help to partly dissolve the often contentious debates between "splitters" and "lumpers" (a hoary distinction that finds its origins in biological classification, e.g., Mayr, 1982) that have long bedeviled the field of psychiatric classification (see Youngstrom et al., 2003). From the perspective of a hierarchical model, the question of whether covarying Disorders A and B are the "same" or "different" conditions is often oversimplified (see also Lilienfeld, 1995), because Disorders A and B may be similar at one level of explanation (e.g., the level of a higher-order dimension of personality, such as Negative Emotionality) but different at another level of explanation (e.g., the level of a lower-order dimension of personality, such as anxiety sensitivity). If a hierarchical model successfully accommodates Disorders A and B, the question of whether to split or to lump these disorders becomes partly a matter of stylistic taste and preference, although important questions concerning clinical utility (e.g., Does one obtain pragmatically useful levels of incremental validity in predicting natural history or treatment outcome by using separate measures of Disorders A and B rather than a combined measure of both disorders?) almost inevitably arise.

The application of hierarchical models to childhood internalizing and externalizing disorders should help to clarify not only the sources of comorbidity between these broad diagnostic classes but also the unique factors that discriminate within them. Moreover, these models may point to potential etiological variables (e.g., individual differences in the septo-hippocampal behavioral inhibition system; Gray, 1982) that could account for the covariation between and within these classes of disorders (see Youngstrom et al., 2003). By elucidating both the common and unique dimensions underlying childhood disorders, such research should bear important implications for future efforts directed at their classification and diagnosis.

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