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# UNMASKING UNMASKED DEPRESSION IN CHILDREN AND ADOLESCENTS: THE PROBLEM OF COMORBIDITY

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**ABSTRACT.** *The unmasking of childhood depression has stimulated considerable research, but the field has drifted unintentionally toward the reification of childhood depression as an entity. In turn, the reification, often embodied in the form of diagnostic categorization, may contribute to premature closure on our understanding of childhood and adolescent depression. One of the major underemphasized characteristics of depression is that it rarely occurs by itself in children. Comorbidity is the rule rather than the exception, and thus, much of what we think we know about the disorder may be shaped by its co-occurrence with other disorders and symptoms. Accordingly, we discuss the conceptual and measurement issues in depression in youngsters, identify the extent of comorbidity, and then discuss some of the implications of comorbidity. Several research issues are raised concerning exploration of the meaning of comorbidity and its possible origins.*

PREVAILING beliefs about childhood depression that were common until fairly recently have generally been dispelled: "if it exists at all it is rare"; "if it exists it is masked as a depression equivalent such as behavior and conduct problems, school difficulties, somatic complaints, or adolescent turmoil"; "it is transitory"; "it is a developmentally normal stage." Dispelling the first two myths, Carlson and Cantwell (1980) demonstrated that many children referred for treatment for other problems actually met adult diagnostic criteria for depression if only the clinician looked beyond the "masking" symptoms. Other investigations have demonstrated that the incidence of diagnosed depression increases

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from preschool through school ages, and in adolescence approaches the frequency of adult depression (Rutter, 1991). Although relatively few in number, recent studies have documented the clinical course of the disorder, and its debilitating toll in terms of academic and social functioning (reviewed in Gotlib & Hammen, 1992). Acknowledging the significance of the problem of depression in youngsters, attempts at downward extensions of adult models of depression also have increased in recent years (reviewed in Hammen, 1990).

We applaud—and hope to contribute to—the increasing interest in depressive experiences in children and adolescents and we welcome the focus on this long-neglected topic and the very real and very impairing depression that youngsters may experience. Nevertheless, we fear that there is yet another myth or misconception in the making. Failure to attend to the matter may impede or misdirect our efforts to understand what child-adolescent depression means, and its implications for etiological and treatment studies.

The problem that commands our attention is *comorbidity*, the fact that most depression co-occurs with other disorders and symptoms in children and adolescents. Stated baldly, we believe that much research on this topic has been guided by a narrow nosological concept that has tended to treat co-existing problems as secondary symptoms or epiphenomena. This has resulted in a situation where a great deal of what we think we know about depression in children and youth may not be about depression as such, but about the co-existing disorders or their mixtures.

Our goal in this article is to define and characterize this problem of co-existing symptoms and identify some of its implications. We then propose some research questions to more fully understand both the meaning of the fact of comorbidity and the meaning of the different patterns of comorbid symptomatology in youngsters.

## CONCEPTUAL AND MEASUREMENT ISSUES RELATED TO COMORBIDITY

Confusion has arisen in research on depressive phenomena, as well as in research on comorbidity of depression, in part as a result of confusion over definitions of the central concepts. It is essential to distinguish among three levels of depressive phenomena: depressed mood, depressive syndromes, and depressive disorders.

### *Depressive Phenomena*

The term depression has been used to describe a wide range of emotions, symptoms, syndromes, and disorders that vary in their severity, duration, and scope. We distinguish between depressed mood, depressive syndromes, and depressive disorders (e.g., Angold, 1988; Cantwell & Baker, 1991; Compas, Ey, & Grant, 1993; Kovacs, 1989; Peterson et al., 1993). Although these three concepts are closely related, they each reflect different underlying assumptions about the assessment and taxonomy, and appear to represent distinct but related levels of depressive phenomena. It is likely that they all have something to contribute to the exploration of the phenomena of co-occurrence of symptoms and disorders.

*Depressed mood and affect* are symptoms of depression and refer to the presence of sad mood, unhappiness, or blue feelings for an unspecified period of time. This focus has emerged from developmental research in which depressive emotions are studied along with other features of development. Research in this tradition often employs self-report scales that emphasize moods and emotions (e.g., Kandel Depression Scale, Kandel & Davies, 1982; Emotional Tone Scale of the Self-Image Questionnaire for Young Adolescents, Petersen, et al. 1984).

A second approach is concerned with constellations of behaviors and emotions that reflect depression; *depressive syndromes or constellations* are identified empirically through the

reports of children, adolescents and other important informants (e.g., parents, teachers). This strategy involves the use of multivariate empirical methods in the assessment and taxonomy of child and adolescent psychopathology, represented by the multiaxial taxonomy of Achenbach (1985; 1991). The concept of a depressive syndrome (constellation) refers to a set of emotions and behaviors that have been found statistically to occur together in an identifiable pattern at a rate that exceeds chance, without implying any particular model for the nature or causes of these associated symptoms.

The third approach is based on assumptions of a disease or disorder model of psychopathology, and is currently reflected in the categorical diagnostic system of the *Diagnostic and Statistical Manual, (3rd ed.)-Revised (DSM-III-R)* of the American Psychiatric Association (1987) and the *International Classification of Diseases and Health Related Problems (ICD-10)* of the World Health Organization (1990). This *categorical diagnostic* approach assumes that depression not only includes the presence of an identifiable syndrome of associated symptoms, but it also assumes that these symptoms are associated with significant levels of current distress or disability and with increased risk for impairment in the indivdiual's current functioning. Differences between individuals are considered in terms of quantitative and qualitative differences in the pattern, severity, and duration of symptoms.

Note that numerous measures of childhood and adolescent depressive symptomatology show mixtures of features of these three definitional types. For instance, self-report questionnaires such as the Children's Depression Inventory contain subfactors concerning depressed mood (e.g., Weiss et al., 1991) but also include other items representative of syndromal features of depression besides mood. However, they do not in themselves constitute measures of the diagnostic category of depression. Also, the self-report inventories such as the CDI and the Child CES-D were developed and are scored and interpreted according to rationally-derived principles based on psychiatric syndrome features, unlike the multiaxial inventories that are scored according to empirically-derived factors.

### *Implications of Different Definitions and Measures*

There are several reasons to emphasize the distinctions among these conceptually different meanings of depression and their associated assessment procedures. First, depending on how depression is defined, it implies either a categorical or a dimensional concept. If categorical, by definition we must make a yes-or-no decision using a diagnostic algorithm and in effect ignore the presence of symptoms not relevant to category placement. To some extent, as we discuss later, comorbidity is an artifact of this method of conceptualizing depression. If dimensional, we generally assume that most individuals have at least some of the symptoms and differ mainly on degree, whether or not they also have symptoms of other conditions. Accordingly, co-occurrence of different symptoms is expected because people may be classified on multiple dimensions.

Second, assessment methods based on the different constructs of depression selectively sample emotions and behaviors and selectively label their products. Brief mood measures, for instance, only show the presence of depressed mood but tell us nothing about any other experiences that might also be present. Other instruments might sample more broadly but the labels chosen for what is measured might be overly narrow. Thus, instruments covering constellations of depressive symptoms are called depression scales, when they might more accurately be termed *negative affectivity*, or measure symptoms not unique to the depressive syndrome.

There are several different semi-structured or structured interview methods for determining clinical diagnosis in children; for a review of their psychometric characteristics and attributes see Kazdin and Petti (1982) and Gotlib and Hammen (1992). Most investi-

gators emphasize the need for separate interviews of the parent and the child. It has been well-established that both sources of information are needed, because the convergence between self- and parent-reported symptoms is modest at best (see Gotlib & Hammen, 1992). Children have better access to subjective states than do their parents, for the most part, and commonly report more depressive symptoms than do their parents.

A surprisingly limited amount of research has addressed the question of whether dimensional and categorical methods of assessment yield similar findings. A large portion of such research has examined the psychometric question of whether dimensional methods have convergent validity when compared with the criterion of clinical diagnosis. When approached in this fashion the answer is a limited yes: high scores on the major self-report instruments commonly predict clinical diagnoses of major depression. On the other hand, there is far from complete correspondence, and the questionnaire instruments should not be regarded as diagnostic instruments when specific cut-off scores are used. The reason for this is the imperfect correspondence between depression scores and clinical diagnoses. Research often finds high rates of false positives (e.g., Kazdin & Heidish, 1984; Roberts, Lewinsohn, & Seeley, 1991). Elevated scores often represent transitory depressive states or other psychopathology besides depression. Similar findings and conclusions have also been drawn from questionnaire measures of adult depression (e.g., Kendall, Hollon, Beck, Hammen, & Ingram, 1987).

Another approach to the convergence question has been to compare scores on questionnaire methods with diagnosis not for validation but for the specific goal of determining the extent of agreement. Such an approach was used, for example, by Edelbrock and Costello (1988), comparing CBCL scales and DISC diagnoses for a large sample of clinically referred children and adolescents. Of particular interest to the current discussion was the finding that the depression scores on the CBCL corresponded closely to diagnoses of major depression and dysthymia but that children with diagnosed depression tended to score high on both the Internalizing and Externalizing scales. Jensen et al. (1993) found relatively modest convergence between DISC diagnoses and children's self-reported scores on depression scales or CBCL scores. They note that it is presently unclear whether the low convergence represents shortcomings of the diagnostic system, the DISC, the scales, or conceptualizations of childhood psychopathology.

We emphasize the importance of considering both dimensional and categorical aspects of disorders (e.g., Compas et al., 1993; see also Angold & Costello, 1993). Altogether, however, the various meanings of the depression construct and their assessment methods contribute to, or obscure, comorbidity and covariation issues—and indeed, require us to clarify our conceptions of whether depressive phenomena are a dimension varying in severity or separate conditions. We need to be reminded that the ways in which we construe depression are a product of both the phenomenology and characteristics of depression as well as the limitations imposed by our theories and methods.

## *Comorbidity/Covariation of Depressive Disorders in Children and Adolescents*

We begin by defining two constructs. Statistical *covariation* refers to multivariate empirical methods as described earlier. Principal components analyses of checklist responses by parents, teachers, and adolescents have been used to identify sets of behaviors and emotions that co-occur in the reports of these informants (Achenbach, 1991a). Correlations among these syndromes that are scored on continuous scales are then examined to determine the degree of covariation among different syndromes of child/adolescent psychopathology. Achenbach and colleagues have identified substantial covariation among core syndromes that exist across age, sex, and source of information (Achenbach, 1991b).

*Comorbidity* refers specifically to the co-occurrence of two or more disorders within the framework of a categorical diagnostic system. Comorbidity usually means that two independent conditions co-occur and diagnostic entities are implied. The validity of the definition, however, requires that the nosological constructs themselves be valid. The nosological system itself may contribute to the prevalence of comorbidity as we discuss later on.

With regard to categorical approaches, Maser and Cloninger (1990) refer to a distinction between diagnostic, pathogenic, and prognostic comorbidity. Diagnostic comorbidity may result from overlapping diagnostic criteria. In general, there are very few signs and symptoms that are specific to a particular disorder. No single feature is a necessary and sufficient indicator of a disorder. The more severe an indivdiual's psychiatric difficulties, the more likely numerous symptoms might be apparent. For instance, agitation, irritability, poor concentration, and negative thoughts about the self and the world are likely to occur in many disorders besides depression, possibly leading to artifactually high rates of comorbid diagnoses.

Pathogenic comorbidity arises when a particular disorder leads to certain other symptoms or disorders, which are therefore considered to be etiologically related. Maser and Cloninger (1990) noted, for example, that diabetes is related to both renal and cardiovascular problems. Depressive disorders may be associated with other symptoms and problems that co-occur with sufficient duration and intensity that they meet criteria for the presence of another separate disorder. In effect, the depression leads to other disorders; for example, efforts to cope with depressive symptoms may cause substance use disorders or bulimia.

Prognostic comorbidity occurs when one disorder represents an earlier manifestation of another (Caron & Rutter, 1991). It has been hypothesized, for example, that anxiety disorders in children may be an early version of depressive disorders (e.g., Kovacs, Gatsonis, Paulauskas, & Richards, 1989). There are critical unresolved issues about the progression of depressive (and other) disorders in children that might be embedded in the comorbidity findings.

Clearly, the ways in which comorbidity may occur as a result of conceptual, definitional, and assessment problems are numerous. These matters have not been addressed with the attention they require. Moreover, as we discuss later, there are numerous possible explanations for true comorbidity when it has been determined to exist apart from artifactual sources. Together, the complexities of the meaning of comorbidity represent strikingly under-studied issues for researchers of childhood and adolescent depression.

## *Diagnostic Comorbidity*

As various investigators have observed, *diagnostic comorbidity* appears to be the rule rather than the exception in childhood and adolescent depression. At the outset, it is important to note that this is true for all childhood disorders—not just depression—as revealed in community surveys of children's disorders. For instance, Anderson et al. (1987) found a 55% comorbidity rate (among those diagnosed, 55% had more than one diagnosis) in a sample of 11-year-olds in Australia. As Caron and Rutter (1991) have observed, rates of co-existing disorders in community samples significantly exceed those which would be expected if separate disorders occurred together by chance alone.

Both clinical and community samples of depressed youngsters have indicated high rates of comorbidity. In a recent review of six community studies, Angold and Costello (1993) found that the presence of depression increases the probability of another disorder around

20 times—but up to a 100-fold increase. Rohde et al. (1991) found an overall rate of 42% comorbidity among those with a diagnosis of depression in their large-scale community sample of adolescents, and Keller et al. (1988) reported a 53% rate of comorbidity in their adolescent clinical sample. The generality of the comorbidity finding is important to note; we would expect to see higher rates of comorbidity in clinical samples because treatment would be sought more often in such multiple problem cases. The demonstration of a similar effect in community samples suggests that the comorbidity phenomenon is not an artifact of sampling.

***Depression and anxiety disorders.*** Reports of clinical samples of children and adolescents find that anxiety disorders (and anxiety symptoms) are extremely frequent in children diagnosed with major depression or dysthymia. In her review of existing studies, Kovacs (1990) concluded that among clinically depressed children and adolescents, up to 70% had significant anxiety symptoms, and 30%–75% of those had diagnosable anxiety disorders. Brady and Kendall (1992) reviewed seven studies and found up to 62% comorbidity of depression and anxiety disorders. Kovacs and colleagues (1989) found that 41% of the children in this sample had anxiety disorders during their index episode of depression: onset was usually between 9 and 11 years of age, with anxiety disorders most commonly developing before major depression but after onset of dysthymic disorder. Those with comorbid anxiety disorders appeared to have earlier age of onset of depression. Kovacs (1990) also noted that the few studies of children referred to treatment for anxiety disorders also indicated high rates of co-existing or lifetime major depressive disorder as well.

Anxiety disorders that co-exist with depression commonly involve the full spectrum, including separation anxiety, overanxious disorder, severe phobias, or obsessive compulsive disorder. Kovacs (1990) speculated that sometimes depression and anxiety are actually a single disorder with anxiety often temporally preceding depression but that sometimes they are distinct entitles and the combination marks a particularly greater vulnerability and negative prognosis.

Diagnostic comorbidity of depressive and anxiety disorders has also been observed in community surveys. Fleming and Offord (1990) reviewed such studies and found a range from 0%–75% comorbidity of the two disorders (see also Angold & Costello, 1993; Nottelmann & Jensen, in press). The wide variation in these figures is likely due to differences in methods, informants, ages, and time frame. For instance, Kashani et al. (1987) surveyed adolescents and found a 75% rate of concurrent anxiety and depressive disorders, whereas Rohde et al. (1991) found a 21% rate of lifetime comorbidity of depression and anxiety disorders in an adolescent sample.

***Depression and conduct/behavioral disorders.*** Behavioral disorders involving antisocial and oppositional, conduct, and substance use disorders, have all been found to be associated with depressive disorders. As noted earlier, externalizing disorders sometimes appear to "mask" the depression in the sense that the behavioral disturbances are usually the ones that come to parental attention and may result in treatment referral.

Considering community samples, rates of conduct disorder/oppositional defiant disorder occurring with depression are quite high (Nottelmann & Jensen, 1993). In their review of studies of child and adolescent depression, Fleming and Offord (1990) reported 17%–79% comorbidity with conduct disorder, 0%–50% with oppositional-defiant disorder, 0%–57% with attention deficit disorder, and 23%–25% with alcohol or drug abuse. The community studies by Kashani et al. (1987) and Rohde et al. (1991) offer an interesting comparison. Kashani et al. (1987) reported that 33% of their depressed sample also

received a diagnosis of conduct disorder, and 50% received a diagnosis of oppositional-defiant disorder. Rohde et al. (1991) combined conduct disorder and oppositional-defiant disorder diagnoses into a single category labeled disruptive behavior and found a current comorbidity rate of 8.0% in the depressed sample (compared to 1.6% in the nondepressed group) and a lifetime comorbidity rate of 12.1% in the depressed group (compared to 6.1% in the nondepressed group). As with anxiety disorders, the comorbidity of depression and disruptive behavior disorders exceeded the level that would be expected from the base rates of the disorders in their samples.

Several studies have examined depression and conduct disorders in clinical samples of children and adolescents and have found substantial rates of comorbidity. Puig-Antich et al. (1982) reported that 33% of a sample of depressed children also had a conduct disorder. In a somewhat overlapping sample of both children and adolescents Ryan et al. (1987) reported that at least mild conduct disorder symptoms were present in 38% of the children and in 25% of the adolescents with more severe levels in 16% of the children and 11% of the adolescents. In their longitudinal study of 104 depressed children, Kovacs et al. (1988) reported a rate of 16% concurrent conduct disorder and a lifetime probability of 36% comorbidity with conduct disorder in this sample. Mitchell et al. (1990) reported rates of comorbidity of conduct disorder and depression of 16% in a sample of 45 depressed children (26% in boys; 0% in girls) and 14% in a sample of 50 depressed adolescents (10% of the males and 17% of the females).

Relatedly, depressed adolescents have been found to have higher rates of illicit drug use and alcohol use or abuse than do depressed children. In the Kashani et al. (1987) community survey of adolescent depression, all of the youth who met criteria for depression also had other diagnoses, including 25% alcohol abuse and 25% drug abuse (see also Keller et al., 1988).

In addition to studies of co-existing diagnoses in depressed children and adolescents, studies of high risk children of depressed parents also find that comorbidity of depression and additional diagnoses is common. For instance, Hammen (1991) found that 58% of children of parents with affective disorders who were diagnosable had more than one diagnosis (see also Weissman, 1988).

Studies of diagnostic comorbidity have also noted other characteristics of comorbid patterns. For instance, Rohde et al. (1991) found that typically the depression followed rather than preceded onset of the other disorder (although Nottelmann & Jensen, in press, drew the opposite conclusion from their review of studies). Also, comorbidity was associated with greater frequency of suicidal behaviors and treatment-seeking but did not affect the duration or severity of depression. Others have suggested that the comorbid patterns predict a more chronic or recurrent course of depression (Keller et al., 1988; Kovacs et al., 1989).

### *Covariation of Depressed Moods and Symptom Constellations*

Several studies have shown that although children and adolescents clearly experienced depressed mood, it may not be a distinct emotional state. Studies have generally failed to distinguish depressed mood from other negative emotions including anxiety, anger, and hostility (Finch, Lipovsky, & Casat, 1989; Saylor, Finch, Spirito, & Bennett, 1984; Wolfe et al., 1987). Finch et al. (1989) suggested that anxiety and depression are not separable in children and adolescents and that the distinction between these two forms of negative affect should be put to rest.

These findings can be understood by considering them within the broader framework of theories of emotion (e.g., Watson & Tellegen, 1985). Extensive evidence from studies

of the structure of emotions in children, adolescents, college students, and adults indicates that self-rated mood is dominated by two broad factors: *negative affect*, which is comprised of negative emotions and distress, and *positive affect* which is made up of positive emotions (e.g., Watson, 1988; Watson & Tellegen, 1985). Depressed mood is one component of the broader construct of *negative affectivity*, whereas positive emotions are important in distinguishing among subtypes of negative emotion (Watson & Clark, 1984). Numerous studies have shown that a constellation of negative emotions including sadness, fear, guilt, anger, contempt, and disgust are moderately to strongly intercorrelated in self-reports (Watson & Kendall, 1989). It appears that depressed affect is most often experienced in combination with these other negative emotions. Depressed mood and negative affect (including both depressed and anxious mood) have been conceptualized as two points on a continuum of depressive problems in children and adolescents (Compas et al., 1993). Moreover, the role of both positive and negative affect needs to be considered in a broader perspective on child/adolescent mental health (Compas, 1993).

Similarly, factor analytic studies of multivariate measures of child and adolescent emotional and behavioral problems have consistently identified a broad-band factor that has been labeled *Internalizing* or *Overcontrolled* problems (Achenbach & Edelbrock, 1978). This syndrome includes problems related to depression, anxiety, social withdrawal, and somatic difficulties.

Depressed mood or negative affectivity has also been shown to covary with symptoms other than negative affect. For example, Cole and Carpentieri (1990) found that in a nonclinical community sample of children, depressed mood (as reflected in symptoms on the CDI) and symptoms of conduct disorder overlap considerably. The correlation between depressive and conduct disorder symptom scores as reported by children, parents, and peers was 0.73, after controlling for sources of shared method variance. Similarly, Garber and colleagues found that reports of symptoms of depression and aggression from children, parents, teachers and peers were significantly correlated ($r = 0.42$) after controlling for method variance (Quiggle, Garber, Panak, & Dodge, 1992).

In research on depressive syndromes, the most direct evidence of the covariation of depressive symptoms with other types of problems is evidenced by the failure of a "pure" depressive syndrome to emerge from the principal components analyses of the reports of parents, teachers, and adolescents (Achenbach, 1991a). Depressive symptoms loaded on a syndrome that included both anxious and depressed symptoms. This strong interrelationship among depressed and anxious symptoms parallels the findings regarding the association between depressed mood and the broader construct of negative affectivity. Thus, from the outset, research on depressive syndromes reflects covariance between depression and anxiety.

Examination of the intercorrelation of the Anxious/Depressed core syndrome with the other core syndromes also indicates substantial levels of covariance. These correlations have been reported separately for the Child Behavior Checklist (CBCL), Teacher Report Form (TRF), and Youth Self-Report (YSR) for clinically referred and nonreferred adolescent boys and girls (Achenbach, 1991). Although these correlations vary substantially, ranging from $r = 0.25$ to $r = 0.80$, the overall mean correlation of the Anxious/Depressed syndrome with the other core syndromes is $r = 0.48$, indicating substantial covariation. Furthermore, the Anxious/Depressed syndrome correlated highly with both internalizing syndromes (withdrawn, somatic complaints) and externalizing syndromes (aggressive, attention problems).

Overall, therefore, there is considerable evidence that the underlying phenomenology of depressed mood and syndrome symptoms in children and adolescents includes a mix-

ture of both internalizing and externalizing emotions and behaviors. We now proceed to a selective discussion of some of the implications of comorbidity and covariation.

## IMPLICATIONS OF COMORBIDITY IN CHILDHOOD/ADOLESCENT DEPRESSION

The presence of the high levels of depression comorbidity and co-existence with other symptoms and behaviors has several implications.

Such phenomena raise conceptual and definitional questions. It appears that the current conception of depression as either a categorical disorder or as a discrete mood state is too simple and too static to capture its complexity and meaning. Thinking about depression from a categorical perspective as a disease syndrome, for instance, requires us to "cut at the joints" to fit our observations to a recognizable pattern but perhaps leaving behind a considerable pile of scraps. This is not an argument for a dimensional or constellational approach to replace the categorical. Each system has its advantages and its disadvantages as Klein and Riso (1992) have discussed in a more general discussion of psychiatric comorbidity. Rather, our goal is to acknowledge that our typical methods of construing and measuring depressive phenomena in children are associated with findings both of high covariation of symptoms and modest agreement between methods. Perhaps the time has come to re-think and re-form our definitions of childhood and adolescent depression, and to include both categorical and dimensional features (e.g., Compas et al., 1993). Multiple measures are also needed to characterize course and risk factors for "pure" and comorbid groups of depressed youngsters.

A second implication of high comorbidity is related: much of what we thought we knew about depressive phenomena in youngsters may be based on mixed symptoms rather than on depression as such. Consider for a moment several of the apparent "truths" from the empirical studies of childhood and adolescent depression. One is that diagnostic depression in young children is rare, increases in school-age youngsters, and approaches adult rates in adolescence (e.g., Petersen et al., 1993; Rutter, 1991). Another is that there are no sex differences or that boys predominate in younger ages, but girls greatly exceed boys in depressive experiences in adolescence (e.g., Petersen et al., 1993). Another is that, in general, past depression is a strong predictor of future depression (e.g., Gotlib & Hammen, 1992). Another is that depression is commonly associated with dysfunctional self-concept and negative cognitions, peer difficulties, and poor relations with parents (e.g., Gotlib & Hammen, 1992).

Each of these empirically established patterns may be the result of comorbidity and the etiological factors that shape covarying symptom expressions. Increasing rates with age might have to do with developmental processes including changes in gender-specific patterns of the expression of symptoms and of coping mechanisms. The impact of past depression on future course might be shaped by comorbid symptoms and the negative impact and impairment that result from the associated symptoms in addition to the effects of depression. That is, depression predicts depression because both affective symptoms and comorbid disorders disrupt the child's life and block sources of self-esteem while creating further stressors.

The correlates of depression concerning social, academic, and family function may also reflect to an unknown extent the influence of co-existing disorders. The degree to which our information about childhood and adolescent depression is dependent on typical existing depressed samples (i.e., those with mixtures of depression and other disorders) means

that the findings are attributed to the depression, while the influence of co-existing disorders is unknown either because it was not controlled or not acknowledged. It is quite rare, for example, for research on children's depressive cognitions to characterize co-existing disorders or to compare purely depressed groups with appropriate control groups. Similarly, studies of children's peer and family functioning—with a few exceptions—have generally ascribed effects to the depressive symptoms while failing to characterize co-existing psychological problems. In view of the high rates of comorbidity, the practical issue of finding truly pure depression is a difficult one. However, we urge both research design and statistical methods to try to isolate empirical effects that are due to depression and those that are associated with other measurable symptoms.

It should also be stressed that many of the findings concerning childhood depression are additionally limited by being based on cross-sectional data, or on only limited samples such as treatment-seeking, or community, or mild symptoms in normal groups. It is not our intention to attribute all the gaps in our knowledge to unexamined comorbidity. We emphasize, however, that studies of the meaning of comorbidity will require longitudinal studies employing multiple populations.

A third practical implication concerns the likelihood that children and youth with comorbid disorders likely differ from and have different outcomes from those with purely depressive disorders. Some studies have evaluated this hypothesis and found it to be true in general of the course of disorder (Keller et al., 1988; Kovacs et al., 1984a, b; Rohde et al., 1991). However, relatively little is known of the social, academic, and family functioning of groups that have depression alone compared with comorbid disorders. Does a child with both depression and conduct disorder, for instance, differ both in terms of depressive experiences and psychological functioning from a child with depression alone? Limited data suggest worse academic and social outcomes for children with multiple disorders (e.g., Anderson et al., 1989), but further research is needed to compare specific groups of pure and mixed depressions.

Certainly, treatment and prevention of depressive disorders are practical consequences to be shaped by further studies of the meaning of comorbidity for functioning and course of disorder. Presence of comorbid conditions, for example, requires consideration of the timing, sequence, and selection of appropriate treatments—topics rarely addressed in the child treatment literature.

Finally, the fact of extensive comorbidity of depression and other disorders poses perhaps the greatest challenge of all: understanding etiology. Efforts to fully explore the meaning of co-existing disorders in children and adolescents present the opportunity to discover new clues about their origins. This is the topic to which we now turn.

## EXPLANATIONS AND ETIOLOGICAL IMPLICATIONS OF COMORBIDITY

What accounts for the extensive co-existence of depressive phenomena with other symptoms? The following is not intended to be an exhaustive list of possible origins of comorbidity. In view of the relative neglect of this topic, we view these as a jumping off point intended to provoke research that leads to further prospects and longer lists.

### *Artifacts of Measurement and Conceptualization*

As our earlier discussion implied, to some extent the patterns of co-occurrence may largely reflect whether our measurement system is based on dimensions or categories and the extent to which our assessment tools sample narrowly or broadly. As Klein and Riso (in press) have noted, characteristics of a nosological system can increase the rates of comorbidity in the following ways. The larger the number of categories the more opportu-

nities for comorbidity (in contrast to lumping together various disorders into more general conditions). Lower thresholds for diagnosing a disorder increase its prevalence, and as prevalence increases there is increased likelihood of co-occurrence with other disorders just by chance. Along this line, the Child Psychiatry Work Group of the American Psychiatric Association Task Force on *DSM-IV* notes that one of the reasons for the high prevalence of comorbid depression in children may be that current criteria for depressive disorders are too easily met (Shaffer et al., 1989). Also, the extent to which hierarchical exclusionary criteria are used affects comorbidity; *DSM-III-R* reduced the number of such hierarchies and thereby increased the likelihood of comorbidity as exemplified by anxiety disorders diagnoses when depression is present.

Overlapping diagnostic criteria also increase the likelihood of comorbidity, and as we noted earlier, it is extremely rare for a symptom to be unique to a single syndrome. Depressive experiences are a particularly problematic example; low mood, negative outlook, poor appetite and sleep, restlessness and poor concentration, for example, may occur under a variety of psychological and life conditions—possibly inflating the likelihood of comorbid diagnoses. Similarly, severity of a disorder is likely to increase the number of symptoms experienced, also increasing the likelihood that more than one disorder will exceed diagnostic thresholds.

Finally, the covariation of symptoms may be attributable to biases in the perceptions of single informants (e.g., parents, teachers). A first step in further research on comorbidity is to account for these potential artifacts and confounds. The degree of comorbidity of depression with other syndromes and disorders must be determined after accounting for overlapping symptoms and controlling for variance (bias) due to different informants.

### *Possible Invalidity of the Diagnostic Boundaries*

To a certain extent, artifactual inflation of comorbidity may be related to characteristics of the nosological system. However, these factors may also be related to yet a third matter: the extent to which the diagnostic formulation is valid. A full discussion of this issue is well beyond the scope of this paper. We note, however, that at least for children, there may be ways in which the boundaries between disorders have been arbitrarily drawn or depend on clinical theory or observation that are not appropriate for children (cf. Achenbach, 1985). Two examples come to mind—the frequent co-existence of depression and anxiety, and depression and anger in children. Earlier, we briefly noted diagnostic, symptom, and syndrome comorbidity of anxiety and depression, as well as conduct disorders and aggressiveness occurring with depression. These comorbidity data are open to multiple interpretations and several of them are discussed next.

With respect to depression and anxiety, two possibilities suggest that the boundaries are far less firm than the nosological system implies. One is the possibility of nonspecific negative affectivity in which depression, anxiety and other negative attitudes and internalized emotional experiences are best construed more generally. Another possibility suggests that anxiety may be a precursor of depression and at some point and under certain circumstances depression may emerge as a separate constellation (this does not mean that we should necessarily think of them as becoming separate disorders; they may continue as a mixture with different levels of mixed symptoms). Thus, it may be the case that at least in some stages of development, the child's experiences are not validly captured by our diagnostic decision that he or she is really depressed with comorbid anxiety disorders. We emphasize, however, that we do not expect that all of the overlap between depression and anxiety is due to a more general state of negative affectivity, and indeed, expect cases of truly comorbid conditions.

Along the same lines, perhaps diagnostic criteria for depression in children that do not include anger and its sequelae will inaccurately characterize the child's experiences (perhaps considering it to be depression with comorbid oppositional disorder). A recent study of the phenomenology of depressive experiences by Renouf and Harter (1990) examined the relation of anger to depressive experiences in young adolescents. In a normal community sample, fully 80% indicated that the most typical emotions they felt when depressed were sadness and anger (the majority indicated anger directed toward others or to the self and others). The authors contend that depressive experiences in young adolescents are best construed as a blend of emotions and speculate that, unlike adults, youngsters have not yet learned to take responsibility for negative social interactions and accordingly blame others. Whether the phenomena of depression undergo a developmental change and whether the findings extend to clinical samples, are topics for further research. Our point, however, is simply that there may need to be further exploration of depressive phenomena in children and adolescents and that what is a valid boundary between depression and other disorders in adults may not be valid for youngsters.

### *Developmental and Temporal Sources of Comorbidity*

Both the fact of symptom covariation and the patterns of comorbidity direct us to explore and understand children's symptom experiences over time. By time, we refer both to temporal changes and to developmental changes. We generally know relatively little about the temporal patterning of depression and other disorders within children. Rohde et al. (1991) found that their depressed adolescent community sample had a 42% comorbidity rate with depression typically following rather than preceding the onset of the other disorder. Similarly, Kovacs and colleagues (1989) reported that their clinic sample of depressed children commonly experienced anxiety disorders before the onset of major depression. Not only do different temporal sequences have implications for understanding depression etiology as we discuss next, but may also indicate naturally-occurring patterns. Such patterns could reflect developmental progressions: e.g., from nonspecific affectivity to specific internalizing disorder or from anxiety to depression (Compas et al., 1992). Are there ways in which mixed disorders become more specific over time? Or, what conditions are associated with unchanging individual difference patterns, whether mixed or pure symptoms? What symptom patterns may reflect maturational changes and which might reflect socialization experiences that change with time? Are there differences between comorbidity of diffuse, chronic states such as dysthymia and conduct disorder, compared with more defined and circumscribed conditions such as specific phobias and major depressive episode?

Both substance use disorders and eating disorders are far more likely to arise in adolescence than childhood, presumably because of changing experiences of meaning attached to certain behaviors and the availability of different coping strategies. Are there ways in which earlier depression "turns into" substance use or eating disorders?

It is possible that some of the keys to unraveling the meaning of comorbid disorders and co-occurring distress concern developmental differences in symptom experience and expression. Some studies have explored this issue but further research is indicated. To date, there are two basic themes emerging from research on depressive experiences at different ages. The first theme is the different predominant patterns of symptom expressions at different ages. For instance, young children rarely report subjective dysphoria and unhappiness. Instead, they may be more likely to report physically unjustified or exaggerated somatic complaints (Kashani & Carlson, 1987) and/or irritability, apathy, disinterest, and uncooperativeness (Kashani, Holcomb, & Orvaschel, 1986). By early

adolescence, however, significant proportions of youngsters report depressed mood (Kandel & Davies, 1982; Petersen et al., 1991; Roberts et al., 1991; Rutter, 1986). This is especially true for girls (Allgood-Merten, Lewinsohn, & Hops, 1990; Kandel & Davies, 1982; Petersen et al., 1991), and adolescent girls are especially likely to report depressive experiences that include negative body image (Allgood-Merten et al., 1990; Petersen et al., 1991).

The other major theme concerning developmental trends in depressive phenomena is the relatively similar picture of diagnosable depression shown by children and adults (e.g., Carlson & Kashani, 1988). In general, investigators have suggested only minor developmental modifications such as irritability/anger as an equivalent to sadness and depressed mood, and low self-esteem as an equivalent to guilt (Ryan et al., 1987).

Overall, the relatively few systematic studies comparing presentation of depressive symptoms in different age groups have found more similarities than differences. Nevertheless, it is important to be sensitive to developmental differences in symptom expression that might signal the need to redefine childhood depression—e.g., as a mixture of anger and sadness rather than as two separate disorders of depression and conduct/oppositional disorder, or as a mixture of anxiety and somatic symptoms with depression rather than separate disorders. It is also important to bear in mind that many of the studies examining age-related expressions of symptoms have not controlled for the possible co-existence of other psychological conditions. Moreover, the extent to which co-existing diagnostic states themselves alter the developmental aspects of depression is itself an important and unresolved issue. And, to further complicate matters, there are likely developmental differences in symptom expression of nondepressive disorders that also affect patterns of comorbidity.

Additional research on the natural course of symptoms and disorders over time is needed. What appears to be comorbidity might more accurately reflect a developmental sequence characteristic of most children with the disorders or might reflect unique adaptations and experiences at the individual level. Clearly, longitudinal research designs rather than cross-sectional surveys are needed to address this matter.

### *Comorbidity as Causes and Consequences*

The presence of depression along with other disorders may imply that the depression is a consequence of another disorder or that depression is a cause of the other disorder. Rather than a static view of diagnoses in which one has or doesn't have a disorder, a more contextual, dynamic view is that one set of disruptive experiences may create another set, either as negative consequences or as maladaptive coping behaviors intended to deal with the first disorder.

An especially apt example concerns several recent related studies of children with co-existing conduct disorder and depressive symptoms. Patterson and Stoolmiller (1991) used both causal modeling statistical procedures and longitudinal methods to determine whether depression resulted from disrupted peer and academic roles. The results supported their *dual failure model*—depression resulting from maladaptive peer and school behaviors. Similarly, Capaldi (1991) studied boys in the Oregon Youth Study during grade 6, and found that boys with both depression and conduct problems were most poorly adjusted. She interpreted the results as consistent with the dual failure model in which those with noxious conduct behaviors have the worst peer and family relations, resulting in vulnerability to depressed mood. Thus, these studies suggest that depression is a consequence of the conduct disorder. Another similar example might include ADHD resulting in school and peer difficulties leading to depression.

It is also possible to imagine instances where a child's anxiety disorder might create depressive reactions. A youngster suffering from separation anxiety who experiences parental divorce or other loss might be especially susceptible to the development of depression. Or, a generally anxious child might easily feel overwhelmed by school and peer challenges, developing depressive experiences of hopelessness and helplessness as well as low self-esteem. Of course, the sequence could also be the reverse: a depressed child has a heightened negative outlook on the future, becoming fearful, overwhelmed, and generally anxious by his or her perceived shortcomings in dealing with threatening or challenging circumstances.

There are additional ways in which the syndrome of depression might itself cause other problems. For example, substance use disorders might follow the maladaptive coping with depression, or eating disorders might represent an attempt to cope with a depression-exaggerated concern with weight and appearance. This suggests that clinical interviews should attempt to determine as best as possible the temporal sequencing of the onset of various symptoms and disorders. This would involve careful lifetime history assessment, and the integration of data from multiple sources including child, parent, teacher, school, and mental health records.

Thus, in addition to temporal associations between different symptom patterns, their functional relationships should be explored as well. Such efforts further move us beyond a static, disease model notion of depression, to view it as a syndrome with profound consequences for the ways in which individuals live their lives (e.g., Hammen, 1991, 1992). Accordingly, treatment needs to address problems stemming from the consequences of the disorders as well as the reduction of symptoms.

### *Correlated Risk Factors*

Another etiological implication of comorbidity is the possibility that the disorders truly are distinct but that they occur together because their separate risk or etiological factors occurred together. The operation of risk factors may be complex; not only might their occurrence be correlated but also a single risk factor might be associated with various, rather than specific, manifestations of distress and disorder. Furthermore, a risk factor may contribute to the development of one disorder, which in turn leads to the development of depression (or the reverse).

Some of the ways in which risk factors might be correlated, predicting covarying symptoms, involve biological, psychological, and social factors. In the biological realm, for instance, family studies have suggested that children of depressed parents are at increased risk for both depression and anxiety disorders (Weissman, 1990), suggesting covariation within a single method of genetic transmission. Another example is that correlated genetic risk factors for separate disorders may be transmitted to a youngster, as in the well-known phenomenon of *assortative mating*. A common pattern, for instance, is for women with affective disorders to marry men with antisocial personalities and/or substance use disorders (e.g., Hammen, 1991; Merikangas & Spiker, 1984; Rutter & Quinton, 1984). To the extent that both disorders have a heritable component, the offspring would be at risk for both. At the neuroendocrine level, a model of stress-induced dysregulation of HPA processes that includes their linkage with neurotransmitter functioning would appear to suggest that both depression and anxiety might result from dysregulation (Gold, Goodwin, & Chrousos, 1988).

Correlated risk factors in the cognitive realm involve the acquisition of cognitive schemas that influence perceptions and their emotional consequences. Depression, anxiety, and anger, for example, are hypothesized to arise from somewhat different cognitions

about the worth and competence of the self, perceived danger, and attributions of blame for negative events. Nevertheless, there are both theoretical and empirical reasons for suspecting that different cognitive themes may co-occur, giving rise to mixed patterns of distress and disorder (e.g., Alloy, Kelly, Mineka, & Clements, 1990; Beck, Brown, Steer, Eidelson, & Riskind, 1987; Quiggle et al., 1992).

Studies of social processes, such as the experience of stressors and family interaction patterns may also suggest examples of correlated risk factors associated with covariation in symptoms. Although studies of the effects of stressful life events have clearly established their association with depressive moods and disorders (Compas, Grant, & Ey, in press), studies that have also included measures of other symptoms have found stressors to be related to those symptoms as well. Although there may be specific links between types of stressors and specific vulnerability for depression (e.g., loss), it is also possible that some stressors elicit multiple symptoms.

The realm of dysfunctional family or parent–child interactions raises various possibilities of correlated risks for multiple outcomes. A well-known example is the co-occurrence of parental depression and marital discord, potentially leading to children's depression and conduct disorder (e.g., Downey & Coyne, 1990). Another example is disturbances of mother–infant attachment; insecure (anxious) attachment might be expressed as both depression and anxiety; also, various maternal behaviors eventuating in disorganized forms of insecure attachment might predict the child's expressions of both depressive and hostile/antisocial behaviors.

There are numerous additional possibilities of correlated risk factors or of risk factors associated with nonspecific outcomes. Further studies of comorbidity of symptom patterns in children might help to shed further light on such etiological mechanisms.

## SUMMARY AND CONCLUSIONS

The unmasking of depression in youngsters has stimulated enormous efforts to understand and treat childhood and adolescent mood disorder. Yet there is concern that we have implicitly assumed that such comorbid depression is the same depression as in depressed-only children or that mixed conditions are more similar to each other than different. The extent and patterns of comorbidity and covariation of symptoms of depression and other problems in children and adolescents raise critical issues for our field. The fact that comorbidity appears to be the rule, rather than the exception, calls into question our very conceptualization of depression, as well as our understanding of its causes and consequences.

The discovery that depression can be diagnosed in children and adolescents using the same criteria as applied to adults has sparked a great deal of research with important theoretical and practical implications. Nevertheless, it may have contributed to the illusion that we thereby understand depressive phenomena in youngsters. We conclude with the following provocative comments, intended to stimulate further thinking and research.

Much of what we thought we knew or understood about the syndrome of depression in children and the course of the disorder, may be based not just on the depression but on the nature and extent of concurrent disorders. As Caron and Rutter (1991) noted, we may have misattributed to depression some of the characteristics and correlations that more accurately concern co-existing conditions.

This is a profoundly complex matter. It is essential to explore the natural course of disorder, including not only the influence of different kinds of symptom expressions on each other and the course of disorder but also the influence of symptoms on the child's

development and role functioning and how such adaptations or dysfunctions influence the expression of symptoms.

Correspondingly, we have little information about characteristics that are specific to the depression experience rather than that which might be characterized as nonspecific distress or mixed with various internalizing and externalizing disorders. There is, indeed—for both child and adult depression—relatively little research demonstrating the specificity of particular models and theories for depression alone.

A related concern is that researchers and clinicians may make the assumption that depressive experiences in children and adolescents are the same, regardless of whether the depression co-occurs with other disorders or not. We suspect that the truth is far different and that there are important etiological, prognostic, and treatment implications of different depressive profiles.

A related difficulty is the extent of limitations in our understanding of depression as mood, syndrome, and diagnostic entity in youngsters and how these different expressions differ from each other or relate to one another. Whereas the continuity of mood and diagnosed depression in adults may be called into question because of the typically transitory and normal experience of depressed mood (Gotlib & Hammen, 1992), we may lose a good deal of information about child and adolescent depression by failing to explore and understand nonclinical depressive phenomena.

The prevalence of comorbidity/covariation of depression with other conditions poses fundamental questions about its origin and meaning. For instance, comorbidity might arise from artifactual sources such as definitional and assessment matters, from developmental processes in which depression might be viewed as an outcome or a consequence of earlier dysfunction or from covariation in risk factors, such as family process, genetic, biological, and psychological patterns.

The nature of samples, methods, and assessments needed to address some of the fundamental definitional, developmental, and course of disorder issues is greatly affected by recognition of the comorbidity issue. For example, clinical samples are more likely to show comorbidity but are less representative of the disorder as a whole, whereas nonclinical samples may present with mild versions of depression whose continuity with clinical conditions needs to be proven rather than assumed. Instruments measuring the depressive symptoms have been emphasized to the relative neglect of measuring children's behaviors and adaptations—themselves likely to affect the expression of mood symptoms and related behaviors.

The risk factors—biological, psychological, and social—present a formidable challenge to the goal of determining specificity. Moreover, often risk factors are themselves correlated (or nonorthogonal, Walker, Downey, & Nightingale, 1989), such as genetic, marital distress, cognitive and social factors. Nevertheless, progress cannot be made by ignoring covariation in children's disorders.

Treatment of childhood depression is itself a relatively undeveloped topic of research often yielding inconclusive evidence of effectiveness. Comorbidity of depression with other disorders requires elaboration of complex treatment strategies aimed at different target symptoms as well as their consequences in family, social, and academic life. The course and outcomes of mixed disorders are likely very different from those of depression alone, and challenge us to develop strategies that take into account increasing knowledge of the meaning of comorbid depressions.

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