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COMORBIDITY OF ANXIETY AND UNIPOLAR MOOD DISORDERS

Susan Mineka

Department of Psychology, Northwestern University, Evanston, Illinois 60208; e-mail: mineka@nwu.edu

David Watson and Lee Anna Clark

Department of Psychology, The University of Iowa, Iowa City, Iowa 52242-1407

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ABSTRACT

Research on relationships between anxiety and depression has proceeded at a rapid pace since the 1980s. The similarities and differences between these two conditions, as well as many of the important features of the comorbidity of these disorders, are well understood. The genotypic structure of anxiety and depression is also fairly well documented. Generalized anxiety and major depression share a common genetic diathesis, but the anxiety disorders themselves are genetically hetergeneous. Sophisticated phenotypic models have also emerged, with data converging on an integrative hierarchical model of mood and anxiety disorders in which each individual syndrome contains both a common and a unique component. Finally, considerable progress has been made in understanding cognitive aspects of these disorders. This work has focused on both the cognitive content of anxiety and depression and on the effects that anxiety and depression have on information processing for mood-congruent material.

CONTENTS

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MEANINGS AND IMPLICATIONS OF ANXIETY-DEPRESSION COMORBIDITY

Background

Comorbidity is currently one of the "hot" topics in psychopathology research (Kendall & Clarkin 1992). According to Klerman (1990), a neo-Kraeplinian belief in the existence of discrete mental disorders clearly reemerged in the Third Edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-III*) (American Psychiatric Association 1980), fueled in part by the success of research into diagnostic classification afforded by such developments as the Research Diagnostic Criteria (RDC) (Spitzer et al 1978). Consistent with this belief, the framers of *DSM-III* included extensive exclusionary criteria. However, criticism of these exclusionary rules soon followed on both conceptual and empirical grounds (First et al 1990), and they were largely eliminated in *DSM-III-R* (American Psychiatric Association 1987). Freed from these exclusionary rules, ensuing research documented extensive comorbidity across the entire spectrum of psychopathology (Clark et al 1995, Kendall & Clarkin 1992) and sparked numerous discussions on the meaning and implications of comorbidity in psychopathology.

With regard to anxiety and depression, the modern history of their classification (Clark 1989, Clark & Watson 1991a,b) and the surrounding controversies (Cole et al 1997, Feldman 1993, King et al 1991, Lonigan et al 1994) have been reviewed previously. Throughout the century, the anxiety and depressive disorders have been treated as separate diagnostic classes in official nosologies; many researchers have argued that these disorders are distinct entities (Akiskal 1985, Cox et al 1993). Nonetheless, other researchers have asserted that they represent a single underlying dimension, or that together they form a more general class of mood disorders (Feldman 1993, Hodges 1990). This latter, unitary construct,

view has been particularly prominent in Europe where even the term "mood disorders" subsumes both anxiety and depression. In turn, the former, dual construct, view may reflect a more general tendency on the part of American psychiatry toward diagnostic "splitting" rather than "lumping" (Frances et al 1990).

In *DSM-III* exclusion rules limited co-diagnosis both within and across the anxiety and depressive disorders, but as mentioned, early research that ignored these exclusion rules revealed extensive comorbidity. Moreover, research based on the *DSM-III-R* and *DSM-IV* (American Psychiatric Association 1994), in which the exclusion rules were largely eliminated, documented that the anxiety and depressive disorders were among the most notable examples of overlapping disorders (Clark et al 1995). Ironically, insofar as this research is leading some researchers to argue that diagnostic splitting has gone too far and that certain disorders should be recollapsed into a single category, it may serve ultimately to justify the view of the *DSM-III* framers that certain coexisting syndromes actually reflect the same underlying disorder and should not be diagnosed separately.

More generally, researchers gradually have begun to realize that the controversy over the unitary versus dual models is both unnecessary and unproductive. These models increasingly are being replaced by a more nuanced view in which anxiety and depression are posited to have both shared, common components and specific, unique components (Clark & Watson 1991b). We discuss these developments in a subsequent section.

The Meaning of Comorbidity: Current Controversies

The term *comorbidity* was coined in the context of chronic disease (Feinstein 1970) to refer to "any distinct additional clinical entity that has existed or that may occur during the clinical course of a patient who has the index disease under study" (pp. 456–57). Attempts to extend this definition of comorbidity to psychiatric disorder, however, quickly run into difficulties in delineating the concept of a "distinct clinical entity" (Lilienfeld et al 1994). If the presence of Disorder A significantly increases the likelihood of the occurrence of Disorder B, various reasons may explain this increased co-occurrence. Kaplan & Feinstein (1974) described several types of comorbidity in medical disorders in which the development of Disorder B was specifically related to the presence of Disorder A. For example, Disorder A may be a risk factor for B (prognostic comorbidity), or Disorder B may be a secondary complication of A (pathogenic comorbidity) (for discussions, see Lilienfeld et al 1994, Maser & Cloninger 1990a, Spitzer 1994). But what if Disorder A [(e.g. major depressive disorder (MDD)] is frequently comorbid not only with B [e.g. general anxiety disorder (GAD)] but also with Disorders C, D, E, F, G, and H (e.g. the other anxiety and mood disorders) and further, what if these disorders themselves sub-

stantially co-occur? Given these conditions, the concept of a distinct clinical entity is decidedly fuzzy; yet data from the National Comorbidity Survey (NCS) reveal precisely this pattern (e.g. Kessler 1997).

Discussions of this topic have been wide ranging. Frances et al (1990) present a series of methodological issues that affect determination of the degree of comorbidity. For example, they note that higher rates of comorbidity tend to be found in more recent studies using structured interviews. To discount the phenomenon of comorbidity based on this fact, however, would be to blame the messenger for the message. Another issue commonly raised is that the presence of the same symptom in two or more diagnoses (e.g. sleep disturbance in MDD and GAD] artifactually raises the co-occurrence of the disorders (e.g. Caron & Rutter 1991). A key word here is "artifactually." Certainly no one would argue that the presence of fever in the diagnosis of chicken pox and measles artifactually raises their co-occurrence, or that "headaches" should be removed from the list of symptoms of either brain tumors or concussions in order to improve differential diagnosis; nevertheless, similar arguments have been made for overlapping psychological symptoms, perhaps reflecting differential levels of knowledge about the etiology of medical versus mental disorders.

Excessive diagnostic splitting also has been noted as an artifactual "cause" of comorbidity, usually with regard to highly similar disorders (e.g. overanxious disorder and GAD in children; Caron & Rutter 1991). However, the separation of phenotypically diverse syndromes (e.g. MDD and GAD) into distinct categories may reflect the same phenomenon. Thus, the greatest challenge that the extensive comorbidity data pose to the current nosological system concerns the validity of the diagnostic categories themselves—do these disorders constitute distinct clinical entities? Even when one psychological syndrome temporally precedes another (e.g. GAD preceding MDD), the earlier syndrome may represent a prodromal manifestation of the latter.

Lilienfeld et al (1994) advocate avoiding the term "psychiatric comorbidity" because it implies greater knowledge about disorders than currently exists and will lead to reification of the *DSM* syndromes. Others, however, argue that the term is less important than the phenomena it reveals (Robins 1994). Certainly, the challenge is to make sense of surface-descriptive comorbidity data, and to recognize that more extensive information about underlying mechanisms and causal relationships is needed for this purpose (Frances et al 1990).

Impact of Comorbidity

Numerous studies have documented the negative effects of anxiety-depression comorbidity on various aspects of psychopathology, such as course, chronicity, recovery and relapse rates (Brown et al 1996, Rief et al 1995; however, a few studies report no effects, e.g. Hoffart & Martinsen 1993), treatment seek-

ing (Lewinsohn et al 1995, Sartorius et al 1996), and psychosocial functioning (Lewinsohn et al 1995, Reich et al 1993). Insofar as comorbidity rates tend to be substantially higher in individuals with more severe conditions (Kendall et al 1992, Kessler et al 1994), and severity of disorder is also a negative prognostic indicator (Keller et al 1992), it may be impossible at this stage of our knowledge to disentangle the issues of comorbidity and severity of disorder (Clark et al 1995). We next illustrate the negative impact of comorbidity involving anxiety and depression by reviewing evidence related to suicide potential.

SUICIDE POTENTIAL Multiple studies in a range of settings have found increased rates of suicidal ideation, suicide attempts, and completed suicide in cases with comorbid anxiety and depression compared to those with a single disorder. In general, the risk of suicide is greater in patients with depression than any other single diagnosis (Wilson et al 1996) with the possible exception of substance abuse (Conwell 1996). However, the increased risk associated with comorbidity cannot be attributed simply to the presence of depression (Clark et al 1995). For instance, the co-presence of anxiety in patients with depression, or vice versa, increases the risk of suicide over the risk associated with pure depression (Bronisch & Wittchen 1994, Reich et al 1993). Moreover, the direction of causality is not simply from comorbidity to increased suicide risk: Newly abstinent substance abuse patients were more likely to develop a comorbid anxiety disorder if they had a history of suicidal ideation (Westermeyer et al 1995).

Increased rates of suicide attempts in individuals with comorbid depression and anxiety disorders have been reported in community samples (Angst 1993, Bronisch & Wittchen 1994, Lewinsohn et al 1995, Schneier et al 1992) as well as patient samples (Fawcett et al 1993, Reich et al 1993). Many studies have also examined comorbidity of anxiety and depression with other disorders and found increased suicide risk regardless of the comorbid disorder (e.g. personality disorder, Zisook et al 1994). Similarly, age does not appear to be a factor: Similar findings have been reported in children (Shafii et al 1988), adolescents (Lewinsohn et al 1995), and the elderly (Kunik 1993).

SYMPTOM CO-OCCURRENCE AND DIAGNOSTIC COMORBIDITY

Symptom Co-occurrence

The term "comorbidity" probably should be reserved to designate co-occurring disorders (or at least syndromes), but investigation of anxiety-depression comorbidity begins with the observation that key symptoms that define these theoretically distinct syndromes or disorders often co-occur. These symptoms can be divided into those that are unique to each type of disorder (e.g. panic at-

tacks versus feelings of worthlessness) and those that are shared (e.g. difficulty concentrating). However, few of these symptoms clearly differentiate patients with one type of disorder versus the other (Clark 1989). When rated by clinicians, panic attacks, agoraphobic avoidance, and overall autonomic symptoms (but, surprisingly, not anxious mood) tend to be found more frequently in anxiety disorder patients, whereas depressed mood, anhedonia, psychomotor retardation, suicidal behavior, early-morning wakening, and pessimism (but not loss of libido, loss of appetite, feelings of worthlessness, or guilt) are generally found to be more frequent in depressed patients. However, when self-ratings are compared, depressed patients tend to report more symptoms of both types than do those with anxiety disorders (Clark 1989).

A similar picture is obtained at the syndromal level. The psychometric properties of measures assessing syndromal depression and anxiety are generally good in terms of convergent validity for both self- and clinician ratings, but the discriminant validity of self-ratings is poor in both adults (Clark & Watson 1991b) and children (Brady & Kendall 1992). Clinician ratings are notably more discriminating, suggesting that clinicians give more weight to factors that distinguish anxiety from depression than do patients. It is unclear, however, whether this represents (*a*) sensitivity to subtle cues that patients discount or are unaware of, or (*b*) rating biases on the part of clinicians. However, ratings of anxiety and depression in children by clinicians, teachers, and parents also show poor discrimination: Analyses of behavioral and observational rating scales typically yield a single anxiety-depression factor in children. It is unclear whether (*a*) the syndromes are less differentiated in children or (*b*) the scales used to assess them are less adequate than those available for use with adults (Brady & Kendall 1992).

Diagnostic Comorbidity

ANXIETY AND DEPRESSION As noted, lifetime diagnoses of anxiety and depression show extensive comorbidity. In Clark's (1989) meta-analysis, depressed patients had an overall rate of 57% for any anxiety disorder. The NCS found a remarkably similar 58% lifetime prevalence rate (Kessler et al 1996) and an only slightly lower (51.2%) 12-month prevalence rate. In general, the likelihood of a particular anxiety disorder co-occurring with depression mirrors the base-rate prevalence of the anxiety disorder (Kessler et al 1994). That is, social and simple phobias have both higher overall base rates and the highest rates of occurrence in depressed individuals, whereas panic disorder has the lowest rate in both cases. Nonetheless, the odds ratio (OR) in all cases far exceeds co-occurrence due simply to base rates (overall OR = 4.2; Kessler et al 1996). Moras et al (1996) reported a similar phenomenon but at notably lower levels for strictly defined *current* comorbidity across 10 studies.

As for depression comorbid with anxiety, the overall average is the same (e.g. 56% in Clark's 1989 meta-analysis), but with rates widely varying by diagnosis: 67% in panic/agoraphobia, 33% in *DSM-III* GAD, 20% in social and simple phobias. The rates obtained for *DSM-III-R* GAD are considerably higher, however, in both the NCS data (62.4% for MDD, 39.5% for dysthymia; Wittchen et al 1994) and the one study (73% MDD) reported in Clark (1989), which reflects the major changes in GAD criteria between the two versions. Again, Moras et al (1996) reported lower rates for *current* comorbidity of a mood disorder with a principal anxiety disorder (24%, ranging from 15% for simple phobia to 66% for obsessive-compulsive disorder). The various anxiety disorders are also highly comorbid with each other (Brown & Barlow 1992, Brown et al 1997), but anxiety disorders in the NCS were as—or more—comorbid with depression (*M* OR = 6.6) as among themselves (*M* OR = 6.2) (Kessler 1997).

The literature on children yields similar results. Angold & Costello's (1993) review reported that comorbidity with anxiety disorders was high, ranging from 30% to 75%. Data from adolescents (e.g. DB Clark et al 1994) and from other countries (King 1990) lead to similar conclusions.

Anxiety and depression with other disorders Our focus is on the comorbidity between anxiety and depressive disorders, but these disorders also show extensive comorbidity with other types of psychopathology. For example, anxiety and/or depressive disorders have been found to be strongly comorbid with substance-use disorders (e.g. Kendler et al 1995, Kessler 1997, Mulder 1991, Westermeyer et al 1995, Wittchen et al 1994), hypochondriasis, somatization and other somatoform disorders (e.g. Rief et al 1995), eating disorders (Braun et al 1994, Castonguay et al 1995, Herpertz-Dahlmann et al 1996), conduct and attention deficit disorders (Angold & Costello 1993, Jensen et al 1993, Loeber & Keenan 1994, Milberger et al 1995), and personality disorder (Farmer & Nelson-Gray 1990, Flick et al 1993, Mulder 1991, Shea et al 1992). This sampling of published research includes both epidemiological and clinic-based studies; draws from populations of children, adolescents, adults, and the elderly; and focuses only on the major classes of disorders for which comorbidity with anxiety and depression have been studied. Throughout this review, therefore, it is important to keep in mind that the data focused on anxiety and depression represent but one piece of a puzzle.

OTHER IMPORTANT FEATURES OF COMORBIDITY

In 1990, Alloy & Mineka and colleagues identified three "tentative" phenomena that any comprehensive theory of comorbidity in anxiety and depression should ideally be able to explain: (*a*) the sequential relationship between anxi-

ety and depression, (*b*) the differential comorbidity of depression with various anxiety disorders, and (*c*) the relative infrequency of pure depression compared with pure anxiety (Alloy et al 1990). These phenomena were labeled "tentative" because knowledge about comorbidity was relatively sparse at that time. We now examine the current status of these phenomena.

The Sequential Relationship of Anxiety and Depression Both Within and Across Episodes

The sequential relationship between anxiety and depression has been observed both within episodes and across the lifetime. Within a single episode of illness, anxiety symptoms are more likely to precede depressive symptoms than the reverse (Alloy et al 1990). Such observations stemmed initially from both human and nonhuman primate research examining infants' responses to separation and loss, where there is typically a biphasic response of protest followed by despair or depression (e.g. Bowlby 1973, Mineka & Suomi 1978). Bowlby (1980) later argued that this initial protest response is a prototype for anxiety in adults, whereas the despair response is a prototype for depression. He noted that adults show a similar pattern in response to loss, starting with a brief period of numbness and disbelief, followed by a phase of searching and yearning (cf intense anxiety), and then often by a phase of disorganization and despair (cf severe depression). The experimental literature on response to uncontrollable aversive events also suggests that anxiety symptoms are much more likely to precede depressive symptoms than vice versa (Alloy et al 1990).

Regarding lifetime comorbidity, Alloy et al (1990) also reviewed evidence that an anxiety disorder is significantly more likely to precede a mood disorder than the reverse. This phenomenon is now well established. For example, in the International WHO/ADAMHA CIDI field trials, of the 242 individuals with comorbid anxiety and mood disorders, 59% had had the onset of the first anxiety disorder at least a year before the onset of the first mood disorder, only 15% had experienced the onset of a mood disorder before the onset of the first anxiety disorder, and another 26% experienced the onset of both within the same year (Lepine et al 1993).

Similarly, in the NCS data, Kessler et al (1997) found that all the anxiety disorder diagnoses are associated with an elevated risk of a later diagnosis of minor or major depression. The ORs were especially high for severe major depression (7–9 symptoms), ranging from 2.86 for social phobia to 12.87 for GAD. Kessler (1997) also reported that anxiety disorders were more likely to be temporally primary. For example, nearly 83% of patients with one or more lifetime anxiety disorders reported that one of these was their first disorder; in contrast, only about 44% of those with a mood disorder reported that it was

their first disorder. In fact, most cases (roughly 62%) of lifetime major depression are secondary to other *DSM-III-R* disorder(s), with anxiety disorders being the most common primary conditions (about 68% of cases with secondary depression are associated with a temporally primary anxiety disorder) (Kessler et al 1996). The elevated risk of depression in those with a temporally primary anxiety disorder endures for many years without changing in magnitude.

Similarly, recent reanalyses of the ECA study (approximately 18,000 adults) indicated that of the participants with comorbid depression and social phobia, the latter was temporally primary for nearly 71% (Schneier et al 1992). Lewinsohn et al (1997) reported similar findings in a study of over 10,000 high school students. Finally, Kovacs et al (1989) reported that anxiety tends to precede depression in children (reviewed in Brady & Kendall 1992).

The Differential Comorbidity Between Depression and Different Anxiety Disorders

Alloy et al's (1990) early review also suggested that individuals who received the diagnoses of panic disorder, agoraphobia, obsessive-compulsive disorder, and post-traumatic stress disorder were more likely to experience depression than were those with generalized anxiety disorder, social phobia, or simple phobia. The NCS data generally confirmed this conclusion through the use of OR that take into account the large base-rate differences for the different disorders (Kessler et al 1996). Specifically, lifetime comorbidity data for major depression revealed OR of 2.9 for social phobia, 3.1 for simple phobia, 3.4 for agoraphobia, 4.0 for panic disorder and post-traumatic stress disorder, and 6.0 for GAD (results for obsessive-compulsive disorder are not presented). Thus, the one exception to the pattern observed by Alloy et al (1990) was that GAD was the anxiety disorder *most* likely to co-occur with MDD. Lepine et al (1993) also largely replicated these results, except that social phobia was nearly as highly associated with MDD as were agoraphobia and panic disorder. Finally, Brown & Barlow (1992) reported results from a clinical sample in which obsessive-compulsive disorder and severe agoraphobia were most highly associated with MDD and dysthymia, whereas simple phobia was least associated, and social phobia and GAD were intermediate.

Thus, results seem to confirm that obsessive-compulsive disorder, panic disorder and agoraphobia, and post-traumatic stress disorder are highly associated with MDD and that simple phobia is either less or not associated with it. The results for social phobia and GAD are less consistent, with more recent data suggesting a significantly stronger association with depression. The inconsistent results for GAD likely reflect the major changes in diagnostic criteria made between *DSM-III* and *DSM-III-R*.

The Relative Infrequency of Pure Depression

Alloy et al (1990) also summarized evidence that cases of pure depression without concomitant anxiety were rarer than cases of pure anxiety without concomitant depression. This pattern can be observed at the purely symptomatic level: Individuals with a diagnosis of MDD typically show levels of anxiety on self-report or clinical rating scales that are as high as—or higher than—those of patients with a diagnosis of GAD or panic disorder (e.g. DiNardo & Barlow 1990). It also occurs at the diagnostic level: The likelihood that someone with a mood disorder also will receive an anxiety-disorder diagnosis (either concurrently or subsequently) appears to be greater than the reverse. In the ECA study, 43% of individuals with a mood disorder also received an anxiety diagnosis at some time in their lives, whereas only 25% of those with an anxiety disorder also received a lifetime mood disorder diagnosis (Regier et al 1990). In the NCS data, 58% of those with a lifetime diagnosis of a depressive disorder also had an anxiety disorder (Kessler et al 1996). Finally, in eight of the nine studies of children and adolescents reviewed by Angold & Costello (1993), the rates of mood disorder in those with an anxiety disorder were substantially lower than vice versa.

Theoretical Perspectives on These Important Features of Comorbidity

THE HELPLESSNESS/HOPELESSNESS PERSPECTIVE Alloy, Mineka, and colleagues proposed a helplessness/hopelessness perspective to clarify these important features of anxiety-depression comorbidity (Alloy et al 1990, see also Garber et al 1980). Their model integrates Abramson et al's (1989) hopelessness theory of depression with extensive research documenting the importance of perceived uncontrollability in the etiology of anxiety (e.g. Barlow 1988, Barlow et al 1996, Mineka 1985, Mineka & Kelly 1989, Mineka & Zinbarg 1996). Uncontrollable, stressful life events were originally implicated as playing a central etiological role in depression (e.g. Seligman 1974). However, later reformulations of the helplessness theory of depression deemphasized uncontrollability per se and focused on the central role that key cognitive variables play in determining vulnerability to depression following negative life events (Abramson et al 1978). In a later reformulation, expectations of hopelessness were hypothesized to serve as proximal and sufficient causal factors for the onset of depression (Abramson et al 1989). Concurrently, anxiety researchers—based in large part on animal research—were recognizing the central role that perceptions of uncontrollability play in creating vulnerability to anxiety as well as depression (e.g. Barlow 1988, Mineka 1985, Mineka & Kelly 1989).

Alloy et al (1990) proposed that the interplay of three cognitive components of helplessness and hopelessness may determine, at least in part, whether a person experiences pure anxiety, a mixed anxiety-depression syndrome, or hopelessness depression. Specifically, they proposed that the interplay of helplessness expectancies, negative-outcome expectancies, and the certainty of these expectancies influences which of these states will be experienced. Individuals uncertain about their ability to control important outcomes would be most likely to experience pure anxiety. In contrast, those who are more certain about their helplessness—but still uncertain about whether a negative outcome will actually occur—will experience a mixed anxiety-depressive state. Finally, individuals who are certain of both their helplessness and the occurrence of a negative outcome will experience hopelessness depression, characterized by despair, loss of interest, and suicidality. Thus, anxiety and depression share expectations of helplessness but differ in negative-outcome expectancies (see also Garber et al 1980). Numerous other theorists have also argued that anxiety is characterized by helplessness (e.g. Beck & Emery 1985, Mandler 1972), whereas depression is centered on hopelessness (e.g. Beck 1967, Brown & Harris 1978).

Alloy et al (1990) argued that this perspective is useful in accounting for the three important features of comorbidity discussed. The sequential relationship between anxiety and depressive symptoms is explained by noting that expectancies of helplessness (certain or uncertain) are likely to precede the development of certain negative-outcome expectancies when efforts to exert control may all fail. The across-episode sequential relationship between anxiety and depression may occur because prior experiences with uncertain helplessness (and anxiety) may increase one's vulnerability to more certain helplessness and even hopelessness (and hopelessness depression) in the face of future severe stressors, perhaps especially when an individual has a pessimistic attributional style (i.e. attributing negative outcomes to stable and global causes). This perspective is also consistent with findings that anxiety is associated with anticipated threat or danger, whereas depression is often preceded by a major loss event (e.g. Brown et al 1993, Monroe 1990). Loss events are more likely to lead to hopelessness and depression given that a bad outcome has occurred; threat events have an inherent uncertainty and are more likely to lead to a sense of helplessness and therefore anxiety.

Regarding the differential comorbidity of depression with the different anxiety disorders, Alloy et al (1990) hypothesized that those anxiety disorders characterized by more chronic and pervasive feelings of helplessness would show higher rates of comorbidity with depression. They therefore argued that panic disorder and agoraphobia, obsessive-compulsive disorder, and posttraumatic stress disorder should show a particularly strong degree of overlap

with depression, which has been supported by more recent evidence. Alloy et al further argued that the more pervasive feelings of helplessness associated with these disorders might stem, in part, from the intrusive and terrifying nature of many of their symptoms (e.g. panic attacks, obsessions, compulsions, flashbacks, nightmares, etc). To the extent that such symptoms are perceived as uncontrollable, over prolonged periods a state of certain helplessness would be expected to emerge, which would then lead to depression. Individuals with a pessimistic attributional style who have these anxiety disorders (Heimberg et al 1989, Mineka et al 1995) may be particularly prone to developing depression in response to the experience of uncontrollable anxiety symptoms.

Social phobia and simple phobia, by contrast, should be expected to be associated with a more circumscribed sense of helplessness, perhaps accounting for the lower rates of comorbidity of these disorders with depression. Individuals with generalized social phobia, as opposed to specific social phobia, should show higher levels of depression given that their lives are far more restricted in terms of the social situations they fear and avoid. Holt et al (1992) and Turner et al (1992) both reported such findings.

However, Alloy et al (1990) did not predict the recent findings of high rates of comorbidity between GAD and MDD, given that chronic worry and other generalized anxiety symptoms would not generally be expected to lead to as pervasive a sense of uncontrollability as would panic disorder, obsessive-compulsive disorder, and post-traumatic stress disorder. However, below we review evidence for a common genetic diathesis for GAD and MDD that may be the important key to this relationship rather than any factor posited by Alloy et al's perspective.

Finally, the relative infrequency of pure depression is explained by the observation that the cognition of hopelessness is a subset of helplessness cognitions. That is, people who are hopeless also perceive that they are helpless but the reverse is not necessarily true. This could account for why pure anxiety is much more likely to be observed than is pure depression.

BOWLBY'S ATTACHMENT-OBJECT LOSS THEORY Bowlby wrote extensively about the relationship between anxiety and depression from the vantage point of attachment theory (1973, 1980) and later incorporated ideas from cognitive psychology into his perspective (1980). Attachment theory views attachment behavior in evolutionary/functional terms, with a young infant's attachment behavior serving to create a secure base from which to explore the environment. When separation from the attachment object occurs, a biphasic protest-despair response occurs. Early experiences with separation and loss (as well as the nature of the attachment relationships per se) are postulated to have major effects on adult attachment relationships as well as on response to adulthood

stressors. The effects of these early experiences are thought to be mediated through the development of cognitive schemata that later affect an adult's response to disruptions in attachment relationships.

Bowlby's perspective can explain various aspects of comorbidity. First, the sequential relationship between anxiety and depressive symptoms (within episode) is explicitly predicted; indeed, his observations of children and adults in response to separation and loss are key findings documenting this phenomenon (Bowlby 1973). Second, pure depression is infrequent because people first become anxious about the threat of a loss (or as an initial response to an actual loss); however, if the loss does not occur, or does not persist, or if a substitute attachment occurs, then depression may never occur. In this regard, the importance of social support in buffering against depression in the face of major life stressors has received considerable support in the literature (e.g. Brown et al 1993). Bowlby (1980) also noted, however, that once the depression phase occurs, it is usually interspersed with phases of anxiety and longing for the lost attachment object (mixed anxiety-depression). Finally, Bowlby's perspective (1973, 1980) explicitly predicts the high levels of comorbidity between panic disorder/agoraphobia and depression, in that disordered attachment relationships (early in life and/or in adulthood) play a central role in both conditions, and both are often precipitated by a major loss.

STRUCTURAL MODELS OF ANXIETY AND DEPRESSION

As mentioned, the unitary versus dual construct debate has gradually given way to more sophisticated views of the anxiety-depression relationship. Investigators have used a variety of multivariate techniques to isolate common and unique features of the two constructs and have developed structural models to conceptualize the findings that emerged. We now review the evolution of structural models at both the genotypic and phenotypic levels.

The Genotypic Structure of Depression and Anxiety

SYMPTOM LEVEL ANALYSES Beginning in the 1980s, numerous studies have examined the genetic links between anxiety and depression, primarily investigating whether a common genetic diathesis renders certain individuals vulnerable to the development of both types of disorder. The first major analysis investigated self-reported anxious and depressive symptoms in a large, community-based sample of Australian twins (Jardine et al 1984). These analyses indicated that the observed phenotypic covariation between the two types of symptoms was largely due to a single common genetic factor (see also Kendler et al 1987). Moreover, this same genetic factor was shared with neuroticism, a broad personality trait that reflects individual differences in subjective distress

and dissatisfaction (see Kendler et al 1993a, Watson et al 1994). Thus, anxiety, depression, and neuroticism could be linked to a single genetic diathesis that apparently represents an underlying vulnerability to subjective distress and negative affectivity.

Subsequent analyses of these same data, however, revealed that symptoms of a panic attack (e.g. breathlessness or heart pounding) reflected unique genetic variance that differentiated them from other symptoms of depression and anxiety (Kendler et al 1987) and from neuroticism (Martin et al 1988). Thus, these symptom-level data revealed an important distinction between the cognitive/affective versus somatic symptoms of anxiety, with the former being more closely related to depression and neuroticism than the latter.

DIAGNOSTIC ANALYSES More recently, a number of genetic studies have examined the issue at the diagnostic level and again have revealed evidence of both commonality and specificity, with the level of overlap varying systematically across the individual anxiety disorders. At one extreme, analyses consistently have found that major depression and GAD are genetically indistinguishable; that is, the genetic correlation between these disorders is essentially unity, indicating that they reflect a single, common genetic diathesis (Kendler 1996, Kendler et al 1992, Roy et al 1995). Moreover, replicating results at the symptom level, Kendler et al (1993a) found that this common genetic diathesis was also strongly linked to neuroticism, suggesting that this shared genetic factor represents a general tendency to respond poorly to stress and, therefore, to experience frequent and intense episodes of distress and negative affect (Kendler et al 1992, 1995).

In contrast, anxiety disorders other than GAD are more modestly related to depression. Panic disorder, for example, is genetically distinguishable from both GAD and depression (Kendler 1996, Kendler et al 1995, Woodman 1993), a finding that replicates the symptom-level results. Similarly, Kendler et al (1993b) found that major depression shared a moderate amount of genetic variance with agoraphobia, social phobia, and animal phobia but was genetically unrelated to situational phobias (e.g. fears of tunnels, bridges, heights). Other evidence indicates that obsessive-compulsive disorder is genetically distinct from major depression, GAD, panic, and the phobias (Pauls et al 1994); in fact, unlike these other syndromes, obsessive-compulsive disorder appears to have a strong genetic link to Tourette's syndrome (Pauls 1992, Pauls et al 1995).

To date, Kendler et al (1995) have reported the most comprehensive analysis of the genetic architecture of depression and the anxiety disorders, examining the associations among major depression, GAD, panic, and the phobias. They found evidence of two significant genetic factors. One factor was primar-

ily defined by major depression and GAD (loadings of .64 and .47, respectively); panic disorder also had a moderate loading (.35) on this factor, but the phobias were largely unrelated to it (a loading of only .14). In contrast, the second factor was defined primarily by panic disorder and the phobias (loadings of .58 and .57, respectively).

SUMMARY AND IMPLICATIONS The genetic evidence has important implications for our understanding of the mood and anxiety disorders. First, it is clear that the anxiety disorders themselves are genetically heterogeneous (Kendler 1996, Kendler et al 1995). Thus, one cannot posit a single invariant relation between depression and the anxiety disorders; rather, the nature of the relation necessarily will depend on the type of anxiety that is examined. Consistent with this view, the accumulating data indicate that depression is genetically indistinguishable from GAD, moderately related to panic, and more modestly related to the phobias (which are themselves heterogeneous) (Kendler et al 1993b, 1995). In addition, major depression and GAD both are closely linked to the broad personality trait of neuroticism (LA Clark et al 1994, Jardine et al 1984, Kendler et al 1993a). Taken together, these data suggest that the observed covariation between depression and anxiety is largely attributable to a shared genetic factor that reflects general individual differences in subjective distress and negative affectivity. However, the precise nature of this shared genetic factor remains unclear (for discussions, see Carey & DiLalla 1994, Neale & Kendler 1995, Roy et al 1995).

Finally, these data have potentially important implications for the classification of these disorders within the *DSM*, in that they indicate that GAD is more closely linked to major depression than to the other anxiety disorders (Kendler et al 1995). It is noteworthy, moreover, that comorbidity and structural-modeling data demonstrate this same pattern at the phenotypic level (Brown et al 1995, 1997). Therefore, maximum clarity in this area might be achieved by rearranging the mood and anxiety disorders to place greater emphasis on the close affinity between distress-based disorders such as major depression and GAD.

The Phenotypic Structure of Depression and Anxiety

TWO-FACTOR AFFECTIVE MODEL Paralleling the genetic data, phenotypic models increasingly have emphasized that depression and anxiety are characterized by both common and distinctive features. An early example of this approach was a two-factor model based on the seminal work of Tellegen (1985) that emphasized the role of basic dimensions of affect. Extensive research has demonstrated that affective experience is characterized by two general factors:

Negative Affect and Positive Affect (Tellegen 1985, Watson & Clark 1997, Watson & Tellegen 1985). Negative Affect reflects the extent to which a person is experiencing negative mood states such as fear, sadness, anger, and guilt, whereas Positive Affect reflects the extent to which one reports positive feelings such as joy, enthusiasm, energy, and alertness.

These two general dimensions are differentially related to depression and anxiety. Specifically, depression and anxiety both are strongly related to measures of general Negative Affect. In contrast, measures of Positive Affect are consistently negatively correlated with depressed mood and symptomatology but are largely unrelated to anxious mood and symptomatology (Dyck et al 1994, Jolly et al 1994, Tellegen 1985, Watson et al 1988). Thus, in this two-factor model Negative Affect represents a *nonspecific* factor common to depression and anxiety, whereas Positive Affect is a *specific* factor that is related primarily to depression.

TRIPARTITE MODEL Clark & Watson (1991b) extended this model by proposing a second specific factor—physiological hyperarousal—that is relatively specific to anxiety. They therefore argued that a "tripartite model" offered a more accurate characterization of anxious and depressive phenomena. In this model, symptoms of depression and anxiety can be grouped into three basic subtypes. First, many symptoms are strong indicators of a general distress or Negative Affect factor. This nonspecific group includes both anxious and depressed mood, as well as other symptoms (insomnia, poor concentration, etc) that are prevalent in both types of disorder. However, each syndrome also is characterized by its own cluster of symptoms: somatic tension and hyperarousal (e.g. shortness of breath, dizziness and lightheadedness, dry mouth) are relatively specific to anxiety, whereas manifestations of anhedonia and the absence of Positive Affect (e.g. loss of interest, feeling that nothing is interesting or enjoyable) are relatively specific to depression.

BARLOW'S THREE-FACTOR MODEL Recently, Barlow and his colleagues have articulated a very similar three-factor model (Barlow et al 1996, Chorpita et al 1997). This model emphasizes that the mood and anxiety disorders are fundamentally disorders of emotion (Barlow 1988, 1991) and so relates these disorders to processes associated with the three basic emotions of anxiety (or "anxious apprehension"), fear, and depression. At the symptom level, Barlow and colleagues argue that (*a*) general distress and Negative Affect are manifestations of *anxiety* (anxious apprehension), (*b*) autonomic arousal is an expression of *fear*/*panic*, and (*c*) anhedonia, low Positive Affect, and hopelessness are indicators of *depression*. Paralleling the tripartite model of Clark & Watson (1991b), autonomic arousal and anhedonia/low Positive Affect are viewed as unique symptom clusters that are relatively specific to the anxiety and mood

disorders, respectively, whereas general distress and Negative Affect are relatively nonspecific symptoms that are strongly characteristic of both types of disorder.

TESTS OF THE THREE-FACTOR MODELS The formulation of these three-factor models has stimulated a new wave of research into the phenotypic structure of anxious and depressive symptoms. Several early studies subjected existing psychometric instruments to traditional exploratory factor analyses. Consistent with the tripartite model, these studies found clear evidence of three factors: a specific anxiety factor, a specific depression factor, and a general factor that subsumed both types of phenomena (DA Clark et al 1990, Jolly & Dykman 1994, Jolly & Kramer 1994).

One limitation of these studies is that they relied on measures that were laden with items assessing general distress/Negative Affect, and that covered the two hypothesized specific symptom groups (i.e. somatic hyperarousal and anhedonia/low Positive Affect) less satisfactorily. This meant that the nonspecific factor tended to be quite large relative to the two specific factors; moreover, the paucity of good hyperarousal and/or anhedonia items (particularly the latter) made it difficult to identify specific factors that closely matched the predictions generated by the three-factor models. Nevertheless, several investigators obtained structures that offered strong support for these models (DA Clark et al 1994; Steer et al 1994, 1995), with the exception that the third somatic factor was sometimes broader than expected (Dyck et al 1994).

To address these measure-based limitations, Watson & Clark (1991) created the Mood and Anxiety Symptom Questionnaire (MASQ). Watson et al (1995b) tested the prediction that symptoms of somatic arousal and anhedonia offer the best differentiation of anxiety and depression using two specific scales—Anhedonic Depression and Anxious Arousal—composed of items assessing anhedonia/low Positive Affect and somatic arousal, respectively. Consistent with the tripartite model, these specific scales provided the best differentiation of the constructs in each of five samples, with correlations ranging from .25 to .49 (M = .34). In contrast, measures of anxious and depressed mood—which, according to the tripartite model, should demonstrate much poorer specificity—consistently showed a much higher level of overlap, with correlations ranging from .61 to .78 (M = .69).

Watson et al (1995a) subjected the 90 MASQ items to separate factor analyses in each of five samples (three student, one adult, one patient). Consistent with the model, three large and replicable dimensions could be identified: a nonspecific general distress factor, a bipolar dimension of positive mood versus anhedonia, and a factor defined largely by somatic manifestations of anxiety. However, the specific anxiety factor was somewhat broader than expected

and included several somatic symptoms that do not clearly reflect sympathetic arousal (e.g. nausea, diarrhea). Consequently, this third factor is better characterized as "somatic anxiety" rather than the hypothesized dimension of "anxious arousal." Importantly, the same three factors emerged in each of the five data sets, indicating that symptom structure in this domain is reasonably robust across diverse samples.

Several recent studies have broadened the empirical support for the tripartite model in notable ways. For instance, Joiner et al (1996) showed that the three hypothesized factors emerged in a sample of child and adolescent psychiatric inpatients, thereby extending the model to younger respondents. Joiner (1996) explicitly tested the viability of the model in college students using confirmatory factor analysis; he found that the hypothesized three-factor structure provided a good fit to the observed data, whereas one- and two-factor structures did not. Chorpita et al (1997) replicated and extended these findings in a clinical sample of children and adolescents, conducting confirmatory factor analyses on both self- and parent ratings; again, the hypothesized three-factor model best fit the data. Similarly, Brown et al (1997) reported evidence supporting the tripartite model in LISREL analyses using both self-report and interview-based data.

Psychophysiological analyses offer further support for the tripartite model by demonstrating that the three hypothesized symptom groups reflect highly distinctive patterns of brain activity. Specifically, individuals reporting elevated levels of general Negative Affect consistently show augmented base startle reactivity (Cook et al 1991, Lang et al 1993); this exaggerated basal startle response is thought to be mediated by the bed nucleus of the stria terminalis (Cuthbert et al 1996, Davis et al 1997) and can be distinguished from the cue-based reactivity characteristic of phobics (Cuthbert & Melamed 1993, McNeil et al 1993), which appears to be mediated by the base nucleus of the amygdala (Cuthbert et al 1996, Davis et al 1997). Other evidence has linked heightened levels of Negative Affect to increased activity in the right frontal cortex (Bruder et al 1997, Tomarken & Keener 1997).

The two hypothesized specific factors—anhedonia and anxious arousal—show markedly different patterns of psychophysiological activity. Anxious arousal consistently has been linked to *hyper*activation of the right parietotemporal region; in sharp contrast, anhedonia and low Positive Affect are associated with *hypo*activation of this same region (Bruder et al 1997, Heller 1993, Heller et al 1995), as well as hypoactivation of the left prefrontal area (Larsen et al 1995, Tomarken & Keener 1997). It is noteworthy that formerly depressed patients who were currently euthymic also showed left frontal hypoactivation (Henriques & Davidson 1990, see also Allen et al 1993), as did asymptomatic adolescent children of depressed mothers (Tomarken & Keener 1997).

On the basis of these data, Davidson, Tomarken, and their colleagues have argued that left frontal hypoactivation reflects a characterological deficit in a reward-oriented approach system that clinically manifests itself as a loss of interest and motivation (i.e. anhedonia; see Henriques & Davidson 1991, Tomarken et al 1992, Tomarken & Keener 1997). Further evidence links this frontal asymmetry to activity in the mesolimbic dopaminergic system, which also has been related to individual differences in positive emotionality versus anhedonia (Depue et al 1994, Tomarken & Keener 1997).

CLARIFYING THE RELATION AMONG THE THREE FACTORS Although an impressive array of evidence demonstrates the existence of three separable factors, the nature of the relations among these factors remains an unresolved and somewhat controversial issue. Investigators in some of the earlier studies extracted highly correlated lower order factors corresponding to depression and anxiety, which then gave rise to a second order dimension of general distress or Negative Affect (e.g. DA Clark et al 1994, Steer et al 1995). These results suggest a hierarchical three-factor model in which the traditional syndromes of anxiety and depression represent narrow, lower order constructs that are highly interrelated; in this hierarchical model, the Negative Affect dimension emerges as a broader, more general construct that represents the strong degree of overlap between the lower order syndromes. In other studies, however, general distress, anhedonia/low Positive Affect, and somatic arousal have emerged as three separable first order factors. In some analyses, the somatic anxiety and Negative Affect factors are moderately to strongly interrelated (e.g. Brown et al 1997, Chorpita et al 1997, Joiner 1996), but in other cases all three factors are largely independent of one another (Joiner et al 1996, Watson et al 1995b). These data suggest a nonhierarchical model in which the three hypothesized symptom factors exist at the same basic level of generality.

To a considerable extent, these apparently inconsistent findings reflect the types of variables that were included in these structural analyses. That is, studies using traditional assessment instruments, instruments laden with items tapping general Negative Affect have tended to support a hierarchical model with a dominant higher order factor. In contrast, analyses using carefully selected items that are explicitly linked to the tripartite model have yielded greater support for a nonhierarchical arrangement (see Joiner 1996, Joiner et al 1996). Still, these factors alone cannot account for all of the reported inconsistencies, and further investigations of this issue (e.g. using techniques such as LISREL) are needed. To date, Brown et al (1997) reported the most compelling analysis. They tested various alternative structural models and found evidence of two higher order factors; one of these (Positive Affect) was specifically related to depression, whereas the other (Negative Affect) was nonspecific. The third

component of the tripartite model—anxious arousal—emerged as a specific lower order factor.

An Integrative Hierarchical Model of Anxiety and Depression

HETEROGENEITY OF THE ANXIETY DISORDERS It has become increasingly apparent that at both the genotypic and phenotypic levels, the anxiety disorders are heterogeneous and subsume a diverse array of symptoms (Brown et al 1997, Kendler 1996, Kendler et al 1995, Zinbarg & Barlow 1996). This heterogeneity has important implications for structural models. First, as reviewed earlier, the individual anxiety disorders clearly are differentially related to depression, with certain types of anxiety showing much greater overlap than others. Second, the individual anxiety disorders are differentially related to one another; for example, panic disorder is much more highly related to GAD than to obsessive-compulsive disorder (Brown et al 1997). Third, it is increasingly clear that a single specific factor—such as the anxious arousal or somatic anxiety component of the tripartite model—is insufficient to account fully for the diversity of symptoms subsumed by the anxiety disorders.

BARLOW'S HIERARCHICAL MODEL OF THE ANXIETY DISORDERS To address this last problem, Barlow and colleagues have proposed a hierarchical model of the anxiety disorders (Barlow 1991, Brown & Barlow 1992, Zinbarg & Barlow 1996). They assert that each of the individual anxiety disorders contains a shared component that represents the higher order factor in a two-level hierarchical scheme. In earlier treatments of the model, this higher order factor usually was described as "anxious apprehension" (e.g. Barlow 1991, Brown & Barlow 1992); in more recent papers, however, Barlow and colleagues have acknowledged that it essentially represents the general Negative Affect component of the tripartite model (Brown et al 1997, Zinbarg & Barlow 1996). Accordingly, this higher order factor not only is common across the anxiety disorders but is shared with depression; therefore, it primarily is responsible for the observed overlap both among the individual anxiety disorders and between depression and anxiety. In addition to this shared component, however, each of the anxiety disorders also contains a specific, unique component that distinguishes it from all the others (for a parallel analysis of the childhood anxiety disorders, see Spence 1997).

Several recent structural analyses, which use both self-report and interview-based data, have yielded strong support for this hierarchical view (Brown et al 1997, Spence 1997, Zinbarg & Barlow 1996). The results of Brown et al (1997) are especially noteworthy: They found that the anxious arousal component of the tripartite model was not generally characteristic of the anxiety disorders but instead represented the specific, unique component of panic disorder.

AN INTEGRATIVE HIERARCHICAL MODEL OF ANXIETY AND DEPRESSION We suggest that a more accurate and comprehensive structural model, one that is consistent with both the genotypic and phenotypic data, is one that integrates key features of Clark & Watson's (1991b) tripartite model with Barlow's (1991, Zinbarg & Barlow 1996) hierarchical model of the anxiety disorders. In this integrative, hierarchical model, each individual syndrome can be viewed as containing both a common and a unique component. The shared component represents broad individual differences in general distress and Negative Affect; it is a pervasive higher order factor that is common to both the mood and anxiety disorders and is primarily responsible for the overlap among these disorders. In addition, each disorder also includes a unique component that differentiates it from all the others. For instance, anhedonia, disinterest, and the absence of Positive Affect comprise the specific, unique component of depression.

Thus far, all of these propositions are fully consistent with the original tripartite model. The major change, which is necessitated by the marked heterogeneity of the anxiety disorders, is that anxious arousal no longer is viewed as broadly characteristic of all anxiety disorders; rather, it assumes a more limited role as the specific component of panic disorder (see Brown et al 1997). Note that each of the other anxiety disorders—with the possible exception of GAD, which clearly contains an enormous amount of general distress variance (e.g. Brown & Barlow 1992, Brown et al 1997)—includes its own unique component that is differentiable from anxious arousal. An important task for future research is to specify the nature of these unique components more precisely. In this regard, Brown et al (1997) have demonstrated that one obtains a clearer and more accurate view of these specific components after the influence of the general Negative Affect factor is eliminated.

Both the scope and the precision of this integrative model can be enhanced with three additional considerations. First, the size of the general and specific components differs markedly across the various anxiety disorders. For example, the genetic and phenotypic data both establish that depression and GAD are distress-based disorders containing an enormous amount of variance attributable to general Negative Affect; in contrast, obsessive-compulsive disorder, social phobia, and specific phobia all appear to contain a more modest component of general distress (e.g. Brown et al 1997, Kendler 1996, Kendler et al 1995). Thus, future research must move beyond the simple truism that each disorder is characterized by both a common and a unique component and specify the proportions of general and specific variance that are characteristic of each syndrome.

Second, it now is obvious that this general Negative Affect dimension is not confined solely to the mood and anxiety disorders, but is even more broadly re-

lated to psychopathology (e.g. Hinden et al 1997). Significant elevations in Negative Affectivity and neuroticism have been reported in a wide array of syndromes, including substance use disorders, somatoform disorders, eating disorders, personality and conduct disorders, and schizophrenia (e.g. Krueger et al 1996, Trull & Sher 1994, Watson & Clark 1994). Indeed, Widiger & Costa (1994) recently concluded that "neuroticism is an almost ubiquitously elevated trait within clinical populations" (p. 81). Thus, this integrative model clearly need not be confined to the mood and anxiety disorders, and we encourage future investigators to expand its scope to include a broad range of associated phenomena, such as the personality and somatoform disorders.

Third, symptom specificity must be viewed in relative rather than absolute terms. It is highly unlikely that any group of symptoms will be found to be unique to a single disorder across the entire *DSM*, if for no other reason than that the current taxonomic system contains many problematic diagnoses with overlapping symptoms and unclear boundaries (see Clark et al 1995). Moreover, it is clear that symptoms do not conform neatly to existing diagnostic categories but instead tend to characterize clusters of related disorders. For instance, anhedonia and low Positive Affect are not confined solely to depression, but also characterize—to a lesser degree, perhaps—schizophrenia, social phobia, and other disorders (e.g. Brown et al 1997, Watson & Clark 1995, Watson et al 1988). This suggests that we need to move toward more complex, multilevel hierarchical models in which groups of symptoms are classified at varying levels of specificity. Furthermore, it may be best to view individual disorders as representing unique *combinations* of different types of symptoms, with each type showing varying degrees of nonspecificity and with no type being entirely unique to any single disorder.

COGNITIVE APPROACHES TO ANXIETY AND DEPRESSION

Beck's Cognitive-Content Specificity Approach

In 1976, Beck expanded his cognitive model of depression into a more general model of psychopathology, focusing especially on similarities and differences between the anxiety and mood disorders. The theory holds that these disorders are characterized by specific kinds of cognitions (Beck 1976, Beck & Emery 1985). The automatic thoughts of depressed people tend to focus on themes of self-depreciation and negative attitudes about the world and the future and are fueled by underlying depressogenic schemas that are organized around themes of loss, personal deficiency, worthlessness, and hopelessness. By contrast, the automatic thoughts of anxious individuals are focused on anticipated future

harm or danger and are fueled by schemas organized around themes of danger, uncertainty, and future threat.

Several studies have provided important tests of this cognitive-content specificity approach, as well as of its relationship to the tripartite model of anxiety and depression discussed earlier. An initial test of the theory (Beck et al 1987) involved the development of a Cognition Checklist (CCL) that measured automatic thoughts considered to be relevant to depression (e.g. "I'm worthless," "I don't deserve to be loved") and anxiety (e.g. "I might be trapped," "I am going to have an accident"). The CCL was administered to outpatients at the Center for Cognitive Therapy, together with the Hamilton Rating Scales and structured diagnostic interviews. As predicted, depressed patients had higher scores on the CCL-Depression scale, whereas anxious patients scored more highly on the CCL-Anxiety scale. Moreover, the CCL-Depression scale was significantly correlated with the Hamilton Depression scale, even when controlling for anxiety, and vice versa for the CCL-Anxiety scale.

DA Clark et al (1990) subsequently conducted a similar study of another large outpatient sample, using several additional self-report measures. This study also provided a preliminary test of the extent to which Beck's cognitive-content specificity approach to anxiety and depression was complementary (vs contradictory) to the two-factor affective model discussed previously. An initial factor analysis of the anxiety and depression measures revealed a large general factor, which could be interpreted as negative affectivity, that accounted for roughly 40% of the variance. However, a second analysis in which two factors were extracted, one that clearly represents depression and the other anxiety, provided a better fit to the data. Moreover, depressed patients reported significantly more hopelessness, lower self-worth, and more negative thoughts involving failure and loss; anxious patients had more thoughts of anticipated danger and harm. Not surprisingly, patients with comorbid anxiety-depression diagnoses showed a mixed cognitive profile.

Three recent studies from the same research group (one with 844 outpatients, one with 1000 outpatients, and one with 420 undergraduates) tested the complementarity of the cognitive-content specificity approach with the tripartite model (DA Clark et al 1994, Steer et al 1995). Separate analyses of the three samples yielded a very large second order factor in each case (accounting for approximately 40–50% of the variance), which clearly could be interpreted as the general distress/Negative Affect dimension of the tripartite model. However, each analysis also revealed specific first order factors corresponding to depression and anxiety. Consistent with Beck's model, cognitive content centered on personal loss and failure loaded strongly on the lower order depression factor and more modestly on the higher order Negative Affect factor. Specific motivational and behavioral symptoms of depression (e.g. fatigability, social

withdrawal) also were significant markers of the specific depression factor, although they were modestly related to the general distress factor as well. Moreover, consistent with the tripartite model, a measure of positive emotionality was strongly negatively correlated with scores on the specific depression factor (DA Clark et al 1990). Finally, as predicted by the tripartite model, physiological symptoms of anxiety were most strongly related to the first order anxiety factor. Interestingly, the cognitive symptoms of anxiety were more strongly related to the higher order distress factor than to the specific anxiety factor. These results parallel the genetic analyses described earlier, which also revealed cognitive/affective symptoms of anxiety to be more closely related to neuroticism than were somatic symptoms. Overall, analyses of all three samples yielded broad support for both the tripartite and cognitive-content specificity models and clearly establish the compatibility of these approaches.

Information Processing Approaches: Cognitive Biases for Emotion-Relevant Material

In addition to work comparing the thought *content* of anxious and depressed people, the past decade also has seen much interest in the effects that these disorders have on cognitive *processing*. Researchers have been particularly interested in the role that cognitive biases may play in the etiology and/or maintenance of these disorders. The term "cognitive bias" refers to any selective or nonveridical processing of emotion-relevant information (e.g. Mineka 1992, Mineka & Tomarken 1989). Although the idea that both anxiety and depression can disrupt efficient cognitive processing in a general way has a long history (MacLeod & Mathews 1991), the focus of this research has been to delineate how these disorders often selectively affect processing of *emotion-relevant* material. More than a decade of research has shown that anxiety and depression have prominent effects on different aspects of information processing; most of the research has focused on attentional and memory biases, with a smaller amount addressing judgmental biases. In the current context, it is of particular interest to determine the similarity vs distinctiveness of the biases associated with these disorders.

ATTENTIONAL BIASES Many studies show that anxiety is associated with an attentional bias for threatening information, such that anxious patients' attention is drawn toward threatening cues when both threatening and nonthreatening cues are available. Nonanxious individuals tend, if anything, to show the opposite bias. These studies have used a number of different paradigms, ranging from the emotional Stroop or interference tasks to various attentional probe tasks. The bias appears to occur preconsciously, that is, without awareness (for reviews, see MacLeod & Mathews 1991, Mathews & MacLeod 1994, Mineka &

Sutton 1992). Many researchers believe that this attentional bias for threat may play a role in the maintenance of anxiety. That is, having one's attention automatically drawn toward threatening information may help to maintain or exacerbate anxiety. Results from at least one experiment suggest that the tendency to exhibit this bias may serve as a risk factor for the development of emotional distress symptoms in response to a major stressor (MacLeod & Hagan 1992).

In sharp contrast to the anxiety data, there is little evidence for a similar attentional bias for negative information in depression. There are a few studies suggesting that depressed individuals may show interference on the emotional Stroop task (e.g. Gotlib & Cane 1987, Williams & Nulty 1986); however, a study that directly compared depression and generalized anxiety failed to find such interference in depression (Mogg et al 1993). Moreover, emotional Stroop interference itself is not generally considered to be a pure measure of attentional bias, and the studies that have reported the bias did not rule out the possibility that the depressed patients' elevated levels of anxiety may be responsible for the effect (Mathews & MacLeod 1994). In contrast, attentional probe tasks provide a purer assessment of attentional bias; studies using this paradigm have sometimes found a relative bias, with depressed patients attending to positive and negative material equally, whereas nondepressed individuals show a bias toward positive material (e.g. Gotlib et al 1988). Again, however, one cannot rule out the possibility that anxiety actually is responsible for any observed effects (see Mogg et al 1991).

There are two recent exceptions to this generally negative pattern. Mathews et al (1996) found evidence for attention to social threat information in depression using an attentional probe task, but primarily when the words were clearly visible, suggesting it was a strategic process rather than the automatic preconscious bias seen with anxiety (see also Mogg et al 1995). At present, it is unclear how these findings can be reconciled with the largely negative results of prior studies; moreover, to clarify their meaning, it will be important to replicate these findings using more of the same paradigms used to study attentional biases in anxiety.

MEMORY BIASES For the most part, the data on memory biases show a pattern opposite to that attentional biases. That is, a great deal of research demonstrates the presence of mood-congruent memory biases in depression, whereas the evidence for anxiety is relatively sparse and inconsistent (e.g. MacLeod & Mathews 1991, Mathews & MacLeod 1994, Mineka & Nugent 1995). Studies of mood-congruent memory in depression typically compare the performance of depressed and nondepressed participants on various memory tasks (free recall, recognition, autobiographical memory, etc), in which the materials to be remembered vary in affective content (positive vs negative vs neutral). A 1992

meta-analysis indicated that clinically depressed participants consistently show a significant bias to recall negative information, especially when it is self-referential (Matt et al 1992). Nondepressed individuals typically show a trend toward an opposite bias, favoring positive material. Teasdale (1988) argued that this memory bias creates a "vicious cycle of depression": When one is already depressed, having one's memory biased toward remembering negative events can only serve to perpetuate the depression. Results of several studies have demonstrated that such memory biases are indeed significant predictors of greater levels of depression three to seven months later, even when controlling for initial depression (e.g. Brittlebank et al 1993, Dent & Teasdale 1988).

More recent studies have also examined whether depression is associated with implicit or nonconscious memory biases for negative information. Unlike explicit memory tasks in which participants are asked specifically to reflect upon prior experiences, in implicit memory tasks the participants' memory is tested indirectly. Several more recent studies have provided convincing support for the presence of implicit memory biases (e.g. Bradley et al 1995, 1996; Watkins et al 1996). Such findings are of particular interest because they suggest a possible explanation for why depressed individuals so often have negative information entering into their consciousness without any conscious effort to recall such information.

In sharp contrast to the depression data, there is relatively little evidence indicating that similar biases are associated with anxiety (e.g. Mathews & MacLeod 1994, Mineka & Nugent 1995). Moreover, even when significant findings have been reported, they often have failed to replicate. Two studies did suggest the presence of an autobiographical memory bias in anxiety (Burke & Mathews 1992, Richards & Whitaker 1990), but these results are somewhat suspect because of significant methodological problems (Levy & Mineka 1997). Using improved methodology, Levy & Mineka (1997) found no evidence for an autobiographical memory bias in individuals high in trait anxiety.

Furthermore, although an early study suggested the presence of an implicit memory bias for threatening information in anxiety (Mathews et al 1989a), subsequent studies failed to replicate that finding (e.g. Bradley et al 1995, Mathews et al 1995, Nugent & Mineka 1994). One later study did report a significant implicit memory bias using a perceptual identification task, but no attempt was made to determine if the bias was because the anxious participants also had elevated depression levels (MacLeod & McLaughlin 1995). Overall, the pattern of largely negative findings for anxiety—using a wide range of memory bias paradigms—stands in rather striking contrast to the positive findings seen with depression.

Thus, although the results are not entirely consistent, the weight of the evidence suggests that anxiety and depression have different effects on cognitive

processing. Interesting theories have emerged to explain these apparent dissociations between the cognitive processes associated with the two syndromes (e.g. Oatley & Johnson-Laird 1987, Williams et al 1988), but none at present can account for all of the findings that have emerged (e.g. Bradley et al 1996, Mineka & Nugent 1995). Nevertheless, the different modes of cognitive operation associated with the different primary emotions may reflect the fact that these emotions evolved to meet different environmental demands (Oatley & Johnson-Laird 1987). Anxiety, like fear, is important for vigilance and preparation for impending danger; it therefore requires a cognitive system that facilitates a quick scanning for—and perception of—cues for danger (Mathews 1993). The attentional biases seen in anxiety seem well suited for this purpose. Depression, by contrast, involves a rather more reflective consideration of events that have led to perceived failure and loss; a cognitive system adept at remembering important information that might have led to such failures or losses certainly would facilitate such reflection. The mood-congruent memory biases we see in depression seem well suited for this purpose as well (Mathews 1993, Mineka 1992). In this context it is noteworthy that over a decade ago, Tellegen (1985) in a broad-ranging article specifically described anxiety as an engaged, "orienting" mode characterized by a focus on the future, and contrasted it with the disengaged, "oriented," past-focusing mode of depression.

JUDGMENTAL OR INTERPRETIVE BIASES Both anxiety and depression are associated with several forms of judgmental or interpretive biases. For example, relative to normal controls, anxious and depressed subjects show biased judgments of the likelihood that negative events will occur (Butler & Mathews 1983, Krantz & Hammen 1977). More recently, AK MacLeod & Byrne (1996) compared anxious and depressed individuals on their anticipation of both future positive and future negative experiences. Those with relatively pure anxiety showed greater anticipation of future negative experiences than controls, whereas those with depression (who also had elevated anxiety levels) showed both greater anticipation of negative experiences and reduced anticipation of positive experiences. The authors associated the heightened anticipation of future negative experiences with negative affect and the reduced anticipation of future positive experiences with low positive affect (Clark & Watson 1991a,b).

Anxious individuals also show an increased likelihood of interpreting ambiguous information in a negative manner. This occurs both with ambiguous homophones (e.g. die/dye, pain/pane) (Mathews et al 1989b) and with ambiguous sentences ("The doctor examined Little Emma's growth" or "They discussed the priest's convictions") (Eysenck et al 1991). The most elegant study to date involved a text comprehension paradigm; the results demon-

strated that these interpretive biases were occurring while the anxious individuals were reading the text rather than afterward (MacLeod & Cohen 1993).

SUMMARY Depression and anxiety both are associated with cognitive biases for emotion-relevant material. Judgmental and interpretive biases (which have received the least attention) occur with both conditions. Attentional biases for threatening information are more prominent in anxiety than in depression; this conclusion seems especially clear for preconscious automatic biases. Depression, by contrast, is more clearly associated with memory biases for mood-congruent information than is anxiety. From the evolutionary perspective of Oatley & Johnson-Laird (1987), these differences make some sense. However, from the perspective of the structural models and comorbidity viewed earlier, they are somewhat surprising. For example, in nearly all the studies of attentional bias in depression, the depressed individuals show very high levels of anxiety as well as of depression; nevertheless, they do not generally show the patterns of attentional bias seen with anxiety. What is it about the co-occurrence of depressed and anxious mood that seems to mask attentional biases? Future work in this area would profit from using measures with improved discriminant validity, such as the MASQ developed to test the tripartite model (Watson & Clark 1991).

CONCLUSIONS

Interest in the relationships between anxiety and depression has a long history but only began to receive serious attention from researchers and theoreticians in the mid to late 1980s. Since that time considerable progress has been made in documenting the similarities and differences between these two conditions. In addition, we now have a more sophisticated understanding of many of the important features of both the concurrent and the lifetime comorbidity of these disorders. Major progress also has been made in understanding the genotypic structure of anxiety and depression, with considerable evidence indicating that generalized anxiety and major depression share a common genetic diathesis but that the anxiety disorders themselves are genetically heterogeneous, reflecting multiple genetic diatheses. Phenotypic models also have become quite sophisticated, with data converging on an integrative hierarchical model of mood and anxiety disorders in which each individual syndrome contains both a common and a unique component. The shared component of Negative Affect is common to both the mood and the anxiety disorders. Anxious arousal, one of the components of the tripartite model, seems to be the specific component of panic disorder, whereas the other anxiety disorders have their own unique components that are differentiable from anxious arousal. Finally, considerable progress also

has been made in understanding the cognitive components of the anxiety and mood disorders. This work has focused on both the cognitive content of anxiety and depression and the effects that anxiety and depression have on information processing. The majority of evidence suggests that anxiety is associated with automatic attentional biases for emotion-relevant (threatening) material, that depression is associated with memory biases for emotion-relevant (negative) information, and that both anxiety and depression are associated with judgmental or interpretive biases. Much work remains to be done, however, to understand how this pattern of cognitive biases can be related to the hierarchical structural model of the anxiety and mood disorders that has emerged.

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CONTENTS