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Rethinking the Mood and Anxiety Disorders:
A Quantitative Hierarchical Model for *DSM-V*

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Abstract

DSM-IV (American Psychiatric Association, 1994) groups disorders into diagnostic classes based on the subjective criterion of “shared phenomenological features”. We now have sufficient data to eliminate this rational system and replace it with an empirically-based structure that reflects the actual similarities among disorders. The existing structural evidence establishes that the mood and anxiety disorders should be collapsed together in an overarching class of emotional disorders, which can be decomposed into three subclasses: the bipolar disorders (bipolar I, bipolar II, cyclothymia), the distress disorders (major depression, dysthymic disorder, generalized anxiety disorder, posttraumatic stress disorder) and the fear disorders (panic disorder, agoraphobia, social phobia, specific phobia). The optimal placement of other syndromes (e.g., obsessive-compulsive disorder) needs to be clarified in future research.

Rethinking the Mood and Anxiety Disorders:
A Quantitative Hierarchical Model for *DSM-V*

A large body of research has sought to explicate the underlying structure of the mood and anxiety disorders. This research has been stimulated by two key taxonomic problems: comorbidity and heterogeneity. Comorbidity can be broadly defined as the co-occurrence of different disorders within the same individual (see Lilienfeld, Waldman, & Israel, 1994; Mineka, Watson, & Clark, 1998); it reflects the empirical overlap between constructs that are hypothesized to be distinct. Therefore, when comorbidity occurs at greater than chance levels in the population, it raises the more general issue of discriminant validity. Evidence of significant—often substantial—comorbidity among *DSM* disorders has led to the development of increasingly sophisticated models that attempt to account for these patterns of co-occurrence (see Mineka et al., 1998).

In contrast, heterogeneity results when phenomena that ordinarily are collapsed together are found to be sufficiently distinctive to warrant their separation; in the nosological context, this frequently results in the creation of diagnostic subtypes, which is an acknowledgment that an existing taxonomic category is too heterogeneous to be maximally informative (Watson, 2003b). This type of evidence has stimulated research into the symptom dimensions underlying many of the anxiety and mood disorders, including posttraumatic stress disorder (PTSD), obsessive compulsive disorder (OCD), and specific phobia (see Watson, Gamez, and Simms, in press).

In this paper, I will review this structural evidence and explore its broader implications for the classification of the mood and anxiety disorders in *DSM-V*. I begin by explicating the logic of the current *DSM-IV* (American Psychiatric Association [APA], 1994) classification scheme for these disorders; to highlight significant problems with the current taxonomy, I will recast it as a quantitative structural model. I then review earlier structural models of this domain and discuss how they attempted to address the problems of comorbidity and heterogeneity. Finally, I will examine key problems with the existing

DSM nosology and propose an alternative hierarchical model that more accurately reflects the existing structural evidence.

Methodological Considerations

Robustness of the Structural Data

In the sections that follow, I will rely primarily on correlational and factor analyses that typically involve self-report and/or interview-based scores. I will supplement these results with other types of data (e.g., genetic analyses from twin studies) wherever possible, but corroborating evidence frequently is either sparse or completely absent. Fortunately, the currently available data have established several important structural principles that are highly robust and generalizable across a broad range of conditions. I will highlight three points in particular. First, genotypic and phenotypic results are broadly consistent with one another and generally lead to similar structural conclusions. For instance, after reviewing evidence from several twin studies, Mineka et al. (1998) concluded that “depression is genetically indistinguishable from GAD, moderately related to panic, and more modestly related to the phobias” (p. 391). This pattern was paralleled in the phenotypic results, which indicated that depression was more strongly comorbid with GAD than with the other anxiety disorders. Similarly, analyses of the mood, anxiety, and substance use disorders in both phenotypic (Cox, Clara, & Enns, 2002; Krueger, 1999; Vollebergh et al., 2001) and genetic data (Kendler, Prescott, Myers, and Neale, 2003) have revealed the same basic three-factor structure; I will explore this model in detail subsequently.

Second, structural analyses yield highly consistent results across different types of respondents, including student, general adult, and patient samples. Most notably, O’Connor (2002) conducted a meta-analytic investigation of 37 personality and psychopathology inventories to determine whether there are important structural differences in the data produced by clinical and nonclinical respondents. He concluded that

The results indicate that the same numbers of dimensions or components exist in the correlation matrices for contemporary assessment instruments for clinical and nonclinical populations. Furthermore, statistical assessments of the nature of these components revealed that the factors essentially are the same for the two kinds of populations...More generally, differences between the clinical and nonclinical data were rare, and one would be hard pressed to argue against the overall pattern of similarity. (p. 974)

I subsequently present additional evidence to corroborate this key point.

Third, structural models show impressive cross-cultural replicability. Krueger, Chentsova-Dutton, Markon, Goldberg, and Ormel (2003) examined relations among seven common psychopathological syndromes in 14 different countries across five continents: Europe (e.g., France, Italy), Asia (e.g., China, India), Africa (Nigeria), South America (Brazil, Chile), and North America (United States). Their results indicated that a two-factor model—reflecting a basic distinction between internalizing versus externalizing syndromes—“provided a good fit to data from each country.” (p. 443). Along these same lines, I will show that the same basic structural patterns (such as the strong comorbidity between GAD and the mood disorders) have emerged in large national samples from the United States (Krueger, 1999), The Netherlands (Vollebergh et al., 2001), and Australia (e.g., Hunt, Issakidis, & Andrews, 2002).

Limitations of Diagnosis-based Analyses

At the same time, however, I also must acknowledge one major limitation of this literature, namely, that much of the available evidence is based on dichotomous indicators (i.e., presence versus absence) of *DSM* diagnoses. Although these types of analyses are important and informative, researchers face several problems when conducting analyses based solely on formal *DSM* diagnoses. I briefly note four of these problems here (see also Brown, Campbell, Lehman, Grisham, & Mancill, 2001; Brown, DiNardo, Lehman, & Campbell, 2001). First, many *DSM* disorders are relatively rare and show very low

base rates in nonclinical samples, thereby rendering statistical analyses problematic or impossible (among other things, extremely low base rates will attenuate the magnitude of observed correlations with other variables). For instance, bipolar disorder has been excluded from structural analyses of both the National Comorbidity Survey (NCS; Krueger, 1999) and the Netherlands Mental Health Survey and Incidence Study (NEMESIS; Vollebergh et al., 2001) because of its very low base rate in the general population. Similarly, although OCD symptomatology is quite common in the general population (see Watson, Wu, & Cutshall, 2004), the full syndrome shows a low prevalence rate and often is excluded from structural analyses (e.g., Vollebergh et al., 2001).

Second, diagnosis-based analyses are complicated by hierarchical exclusion rules; these are criteria stipulating that certain disorders should not be diagnosed if they are judged to occur only during the course of a co-existing disorder (one that occupies a higher, more privileged place in the hierarchy; see Brown & Barlow, 1992; Clark, Watson, & Reynolds, 1995). This has proven to be a particular problem in the mood and anxiety disorders. *DSM-III* (APA, 1980) included an extensive set of exclusionary criteria that had the general effect of making it difficult to diagnose anxiety disorders in the presence of significant depression (see Mineka et al., 1998). Many of these exclusion rules subsequently were dropped in *DSM-III-R* (APA, 1987) and *DSM-IV* (APA, 1994), but some remain in modified form, and they have been applied inconsistently across different studies. Brown, Campbell et al. (2001), for example, discuss the problem that a *DSM-IV* diagnosis of GAD should not be assigned if its features occur exclusively within the course of a mood disorder. Because GAD is highly comorbid with the mood disorders (e.g., Brown, Campbell et al., 2001; Brown, Chorpita, & Barlow, 1998; Mineka et al., 1998), the strict application of this exclusion rule will drastically lower the base rate of GAD in a sample and attenuate its associations with syndromes such as major depression.

Third, the diagnostic criteria for many disorders change significantly with each succeeding edition of the *DSM*; some of these changes may affect the strength of their associations with other syndromes. For instance, to improve differential diagnosis vis-à-vis panic disorder, symptoms of autonomic arousal (e.g., accelerated heart rate, shortness of breath) were eliminated from the *DSM-IV* criteria for GAD (see Brown, DiNardo, et al., 2001). The net effect of these changes was to enhance the general distress component in GAD, which in turn has led to greater comorbidity with major depression (see Brown, DiNardo, et al., 2001; Mineka et al., 1998). More generally, substantial changes in the symptom criteria make it somewhat hazardous to collapse findings across *DSM-III*, *DSM-III-R*, and *DSM-IV* diagnoses for the mood and anxiety disorders.

Fourth, diagnosis-based analyses fail to account for the significant heterogeneity that exists within many of the current *DSM* disorders (see Watson, 2003b). As noted earlier, some of the current anxiety disorders can be decomposed into several distinct symptom groups. For example, structural analyses consistently have identified multiple symptom dimensions within OCD (e.g., Summerfeldt, Richter, Antony, & Swinson, 1999; Watson & Wu, in press; Wu & Watson, 2003), PTSD (see Simms et al., 2002), and specific phobia (e.g., Arrindell, Pickersgill, Merckelbach, Ardon, & Cornet, 1991; Cutshall & Watson, in press). These symptom dimensions may correlate quite differently with other syndromes, thereby complicating structural analyses and hampering the creation of an adequate taxonomy.

Reconceptualizing *DSM-IV* as a Quantitative Hierarchical Model

Basic Assumptions

In *DSM-IV*, psychopathology is organized into (a) general diagnostic classes, (b) specific disorders, and (c) diagnostic subtypes. As noted earlier, I will begin by translating this *DSM-IV* nosology into a quantitative hierarchical structure. This exercise will help to explicate various problems with the existing taxonomy that are easily correctable in *DSM-V*. In translating *DSM-IV* in this manner, I make three basic

assumptions. First, disorders that fall in different diagnostic classes (e.g., major depression of the mood disorders vs. OCD of the anxiety disorders) are assumed to be empirically unrelated; this is because the current *DSM* system does not formally recognize any such links by placing them together in a common group. Second, I assume that disorders falling within the same diagnostic class are significantly related; for example, OCD should empirically covary with GAD, because both are classified as anxiety disorders. Third, measures of diagnostic subtypes are assumed to be more strongly correlated than indicators of different disorders. For instance, animal phobia should correlate more strongly with blood-injection-injury phobia (another subtype of specific phobia) than with social phobia.

It could be argued that this translation process fundamentally misrepresents *DSM-IV*, because it was not necessarily meant to reflect patterns of empirical covariation. Most notably, the current diagnostic classes are not designed to model overlap/comorbidity per se; rather, “The organizing principle...is to group disorders based on their shared phenomenological features in order to facilitate differential diagnosis.” (*DSM-IV*, p. 10) In other words, the current organization is rationally-based: Disorders are grouped together if they “seem to belong together” because they share phenotypically similar symptoms. What are these “shared phenomenological features”? According to *DSM-IV* (following closely on the logic of both *DSM-III* and *DSM-III-R*), major depression and the other mood disorders “have a disturbance of mood as the predominant feature.” (p. 317). *DSM-IV* does not include an explicit statement regarding the shared features that bind the anxiety disorders together in a common class. According to *DSM-III-R*, however, “The characteristic features of this group of disorders are symptoms of anxiety and avoidance behavior.” (p. 235)

I believe that we now have sufficient knowledge to eliminate this rationally-based system and replace it with an empirically-based structure that reflects the actual—not the apparent—similarities among different disorders. Put differently, we can facilitate

differential diagnosis much better by grouping together disorders that are strongly comorbid and that truly represent significant diagnostic problems. Indeed, I subsequently will establish that the current nosological distinction between (a) mood disturbance versus (b) anxiety/avoidance is fundamentally unsound and fails to provide an optimal arrangement of these disorders.

This quantitatively-based taxonomy offers several advantages over the current rational scheme. First, unlike the current classification scheme, it models comorbidity directly. That is, a structural model of this sort effectively communicates observable patterns of covariation/comorbidity in an easily understandable form. This is because in a quantitative structure of this type, strongly correlated syndromes are located together within the same group, whereas more weakly related disorders are placed in different classes. This, in turn, would make it easier for clinicians and researchers to incorporate important comorbidity data into their thinking, thereby enhancing both (a) differential diagnosis and (b) the design of basic psychopathology research. Second, by using comorbidity as the primary basis for classification, a quantitative model would stimulate further research into the nature and causes of this important diagnostic problem. Finally, as was discussed earlier, genotypic (e.g., twin studies) and phenotypic results generally parallel one another and typically lead to similar structural conclusions. Accordingly, a quantitatively-based model would facilitate the ultimate development of a taxonomy based on underlying etiological processes (i.e., disorders that covary empirically also are very likely to reflect shared etiological factors, such as common genetic diatheses).

The DSM-IV Taxonomy

With this in mind, the current *DSM-IV* structure is schematically depicted in Figures 1 (mood disorders) and 2 (anxiety disorders). These figures capture the major disorders within each diagnostic class, but exclude residual categories (e.g., depressive disorder not otherwise specified) and disorders linked to specific causes (e.g., substance-induced anxiety disorder). These structures also include officially recognized diagnostic subtypes

(e.g., the four subtypes of specific phobia), but do not model the specifiers that add considerable complexity to certain parts of the system (e.g., major depression with melancholic features).

Two aspects of Figure 2 merit additional comment. First, following previous structural analyses of this domain (e.g., Cox et al., 2002; Kendler et al., 2003; Krueger, 1999; Vollebergh et al., 2001), I have modeled panic disorder and agoraphobia as separate syndromes. According to the text of *DSM-IV*, however, “In clinical settings, almost all individuals (over 95%) who present with Agoraphobia also have a current diagnosis (or history) of Panic Disorder.” (p. 403) Accordingly, one could argue that agoraphobia is more accurately represented as a subtype or secondary complication of panic disorder, rather than as a distinct syndrome.

Second, *DSM-IV* does not recognize formal subtypes of PTSD. However, the PTSD criteria include three distinct clusters of symptoms, each of which must be present for the disorder to be diagnosed: Criterion B (intrusions and persistent re-experiencing of the trauma), Criterion C (numbing and avoidance of stimuli associated with the trauma), and Criterion D (symptoms of increased arousal). This tripartite organization has generated an extensive literature on the structure of PTSD symptoms (e.g., Anthony, Lonigan, & Hecht, 1999; Simms et al., 2002; Taylor, Kuch, Koch, Crockett, & Passey, 1998). Although the results have varied substantially across studies, most analyses have failed to support the 3-factor structure implied in *DSM-IV* (see Simms et al., 2002, for a review). Thus, the organization of symptoms within PTSD represents another unresolved structural problem that needs to be addressed in future research.

Previous Structural Models

The Problem of Comorbidity

As discussed previously, much of the structural work in this area was stimulated by accumulating evidence regarding diagnostic comorbidity. Comorbidity now is widely recognized to be a pervasive problem throughout the *DSM* (Clark et al., 1995; Widiger &

Clark, 2000), including the mood and anxiety disorders (Mineka et al., 1998). It is useful to distinguish three basic types of comorbidity evidence. First, the mood disorders are strongly comorbid with the anxiety disorders, and vice versa. For example, in analyses of lifetime *DSM-III-R* diagnoses in the NCS data, Kessler et al. (1996) reported that 58% of individuals with major depression also met criteria for a comorbid anxiety disorder; the comorbidity rate was only slightly reduced to 51.2% when 12-month diagnoses were used. Conversely, most individuals with diagnosed anxiety disorders also meet criteria for depression, although comorbidity rates vary widely across disorders (Mineka et al., 1998).

Second, the various anxiety disorders are highly comorbid with each other (e.g., Brown, Campbell, et al., 2001; Brown & Barlow, 1992). For instance, using lifetime diagnoses in the NCS data, Magee, Eaton, Wittchen, McGonagle, and Kessler (1996) reported that 74.1% of those with agoraphobia, 68.7% of those with simple phobia, and 56.9% of those with social phobia also met criteria for another anxiety disorder.

Finally, the mood and anxiety disorders both show extensive comorbidity with other types of psychopathology, such as the substance use disorders, eating disorders, somatoform disorders, and personality disorders (Mineka et al., 1998; Widiger & Clark, 2000). These data indicate that a comprehensive structural scheme should not be restricted to the mood and anxiety disorders, but also would need to model syndromes that currently are grouped in other diagnostic classes. I will return to this issue subsequently.

The Two-Factor Affective Model

The “Big Two” dimensions of affect. Why do we see such extensive comorbidity between the mood and anxiety disorders? My colleagues and I originally developed an explanatory model that drew on key findings from the basic mood literature. Extensive evidence indicates that there are two dominant dimensions of emotional experience: Negative Affect and Positive Affect (e.g., Watson & Tellegen, 1985; Watson, Wiese,

Vaidya, & Tellegen, 1999). Negative Affect is a general dimension of subjective distress and dissatisfaction. It subsumes a broad range of specific negative emotional states, including fear, anger, sadness, guilt, and disgust. Its emergence in analyses of affect ratings indicates that these various negative emotions significantly co-occur both within and across individuals. Thus, an individual who reports feeling sad is also likely to report substantial levels of anger, guilt, fear, and so on. In parallel fashion, the general Positive Affect dimension reflects important co-occurrences among positive mood states; for instance, an individual who reports feeling happy and joyful also will report feeling interested, excited, confident and alert.

Influence of the general Negative Affect factor. Extrapolating from these data, we argued that this general Negative Affect dimension was largely responsible for the substantial overlap/comorbidity between depression and anxiety (see Watson, Clark, & Carey, 1988). Put differently, this higher order factor produces strong correlations among different types of negative emotion, including sad/depressed affect and fearful/anxious affect.

To demonstrate this key point, Table 1 reports correlations between the 6-item Fear scale (*afraid, frightened, jittery, nervous, scared, shaky*) and the 5-item Sadness scale (*alone, blue, downhearted, lonely, sad*) from the Expanded Form of the Positive and Negative Affect Schedule (PANAS-X; Watson & Clark, 1999) in 14 different samples. Eleven of these samples were described in Watson and Clark (1997). Eight data sets were collected from students at Southern Methodist University (SMU); they differed only in the rated time frame that the respondents used to describe their mood (e.g., Moment, Today, General). Two additional samples consisted of University of Iowa students who rated themselves using Past Few Days and General instructions. The 11th sample was composed of Dallas-area adults who rated their mood using Past Week instructions. The final three samples consist of (a) friendship dyads, (b) dating couples, and (c) married couples who rated each other (using General trait instructions) on the PANAS-X (see

Watson, Hubbard, & Wiese, 2000); only the other-rating correlations (e.g., the wife's ratings of her husband's fear and sadness) are reported here.

Replicating a very robust finding from the broader mood literature, the Fear and Sadness scales are strongly related in every sample, with correlations ranging from .50 to .69. It is noteworthy, moreover, that this same strong association emerges across (a) different time frames [including both state and trait ratings], (b) different types of respondents [i.e., college students versus adults], and (c) both self- and other-ratings of affect. These data have important structural implications for *DSM-V*, in that they establish a strong link between sad/depressed mood (a core, defining element of the mood disorders) and fearful/anxious mood (a key feature of the anxiety disorders).

The role of Positive Affect. How, then, can depression and anxiety be distinguished? Findings from the mood literature establish that the Positive Affect factor has stronger and more consistent (negative) associations with depression than with anxiety (although I will consider an important exception to this pattern subsequently). This pattern also is amply illustrated in Table 1, which reports correlations between Fear and Sadness and the 8-item PANAS-X Joviality scale (*cheerful, delighted, energetic, enthusiastic, excited, happy, joyful, lively*). Across the 14 samples, Joviality is weakly related to Fear, with correlations generally ranging between -.05 and -.20. In contrast, Joviality is more substantially associated with Sadness, with coefficients generally falling in the -.30 to -.45 range.

The two-factor model of affect. On the basis of these data, we proposed that low levels of positive affectivity (i.e., anhedonia) were a specific feature of depression that distinguishes it from the anxiety disorders (Watson et al., 1988). Thus, in this two-factor model, negative affectivity represents a nonspecific factor common to depression and anxiety, whereas low positive affectivity is a specific factor that is related primarily to depression. In support of this model, indicators of depression and anxiety both are found to be strongly related to measures of general negative affectivity; in contrast, measures of

positive affectivity are consistently negatively correlated with depressed mood and symptomatology and are more weakly related to anxious mood and symptomatology (e.g., Jolly, Dyck, Kramer, & Wherry, 1994; Mineka et al., 1998; Watson et al., 1988).

The Tripartite Model

Basic elements of the tripartite model. Clark and Watson (1991) subsequently extended this two-factor model by proposing a second specific factor—physiological hyperarousal—that is relatively specific to anxiety. They therefore argued that a “tripartite model” more accurately captures this domain. This model classifies symptoms of anxiety and depression into three basic subgroups. First, many symptoms are strong indicators of the general distress/negative affectivity dimension; this nonspecific group includes both anxious and depressed mood (consistent with the earlier two-factor model), as well as other symptoms (e.g., insomnia, poor concentration) that are prevalent in both the mood and anxiety disorders. In addition, however, each syndrome also is characterized by its own cluster of symptoms: somatic tension and hyperarousal (e.g., shortness of breath, dizziness) are unique to anxiety, whereas anhedonia/low positive affectivity (e.g., loss of interest, feeling that nothing is enjoyable) are specific to depression.

Clarifying the nature of the general factor. This tripartite structure has received support in a number of studies (e.g., Brown et al., 1998; Joiner, 1996; see Mineka et al., 1998, for a review). Much of the relevant evidence has been collected using the Mood and Anxiety Symptom Questionnaire (MASQ; Watson & Clark, 1991), a self-report symptom instrument that was designed to test key aspects of the model (see Watson, Clark, Weber, Assenheimer, Strauss, & McCormick, 1995; Watson, Weber, Assenheimer, Clark, Strauss, & McCormick, 1995). For our purposes, it is especially informative to consider results obtained with two of the MASQ scales, General Distress: Anxious Symptoms (GD: Anxiety) and General Distress: Depressive Symptoms (GD: Depression). The 11-item GD: Anxiety scale includes several indicators of anxious mood

(e.g., felt nervous, felt afraid), as well as other symptoms of anxiety disorder that were hypothesized to be relatively nonspecific in the tripartite model (e.g., inability to relax, diarrhea). Similarly, the 12-item GD: Depression scale contains several items reflecting sad, depressed mood (felt depressed, felt sad, felt hopeless), along with other nonspecific symptoms of mood disorder (e.g., feelings of disappointment and failure, self-blame, pessimism).

Table 2 presents correlations between these scales in a large number of samples, including (a) several of our own data sets and (b) results published by other investigators. To further document the important point that structural findings tend to replicate well across clinical versus nonclinical populations, the results have been subdivided according to whether they were obtained in non-distressed samples (i.e., respondents that would be expected to report relatively normal levels of distress; these groups mostly consist of college students) versus distressed samples (i.e., respondents that would be expected to report elevated levels of distress, such as psychiatric patients). Supporting a key prediction of the tripartite model, these MASQ scales are very strongly correlated in both types of respondents, with weighted mean correlations of .68 and .74 in the non-distressed and distressed samples, respectively. Together with the results shown in Table 1, these data have important structural implications for *DSM-V*, in that they clearly demonstrate a very strong association between core symptom features of the mood and anxiety disorders.

The Integrative Hierarchical Model

Limitations of the tripartite model. Although key elements of the tripartite model have received extensive support, the accumulating data also exposed two significant problems. First, this model fails to account for the heterogeneity within the anxiety disorders. Most notably, Brown et al. (1998) 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 (see also Zinbarg et

al, 1994); subsequent evidence also has linked anxious arousal to PTSD (see Brown, Campbell, et al., 2001). Second, several studies have demonstrated that the Positive Affect dimension is not uniquely linked to depression, but also shows consistent negative associations with indicators of social phobia (e.g., Brown et al., 1998; Watson et al., 1988; Watson, Gamez, & Simms, in press).

The integrative hierarchical model. In light of this evidence, Mineka et al. (1998) proposed an integrative hierarchical model that incorporated elements from both the tripartite model and Barlow's (1991; Zinbarg & Barlow, 1996) hierarchical organization of the anxiety disorders. In this expanded scheme, each individual syndrome is hypothesized to contain both a common and a unique component. Consistent with these earlier models, the shared component represents broad individual differences in general distress and negative affectivity; it is a pervasive higher order factor that is common to both the anxiety and mood disorders and primarily is responsible for the comorbidity problem that was discussed earlier. In addition, each disorder also includes unique features that differentiate it from all of the others. Thus, anxious arousal no longer is viewed as broadly characteristic of the anxiety disorders, but instead assumes a more limited role as a specific element in syndromes such as panic disorder.

Mineka et al. (1998) discussed three additional points that are worth noting. First, they summarized a range of evidence indicating that the size of these general and specific components differs markedly across disorders. Specifically, major depression and GAD both are distress-based disorders that clearly contain an enormous amount of this general factor variance; in contrast, most of the other anxiety disorders (such as social phobia and specific phobia) contain a more modest component of nonspecific negative affectivity (for a further elaboration of this point, see Watson, Gamez, & Simms, in press). Second, consistent with the comorbidity data reviewed previously, Mineka et al. (1998) argued that this general negative affectivity dimension was not restricted to the anxiety and mood disorders, but also characterized many other types of psychopathology. Finally, they

asserted that specificity must be viewed in relative terms, arguing that “It is highly unlikely that any group of symptoms will be found to be unique to a single disorder across the entire *DSM*” (p. 398). Consistent with this argument, they concluded that low positive affectivity was not unique to depression, but also characterized schizophrenia, social phobia, and other disorders.

Summary

Basic implications of the data. In summary, these structural models have three broad implications for the mood and anxiety disorders. First, they have identified a higher order dimension—general distress or negative affectivity—that is common to both the mood and anxiety disorders. This has particularly important implications for *DSM-V*, as it suggests that these disorders should be linked together into a more general category of distress-related syndromes, rather than remaining as entirely distinct diagnostic classes. Second, it now is clear that some disorders have a much larger distress component than others; this fact will play a key role in the quantitative hierarchical scheme I present later. Third, given the ubiquity of this general negative affectivity dimension, it seems likely that our structural models ultimately will need to be extended to incorporate a much broader array of psychopathology, including syndromes currently placed in other diagnostic classes.

Failure to account fully for comorbidity. Finally, although the integrative hierarchical model represents an improvement over its predecessors, it also has limitations of its own. This model posits that a single general factor—that is, general distress or negative affectivity—is largely responsible for observed patterns of comorbidity. Specifically, it predicts (a) a high level of comorbidity between two disorders that have strong components of negative affectivity, but (b) a weaker level of comorbidity between syndromes containing less of this general factor variance. This former proposition has received substantial support in the literature. Most notably, consistent with this scheme, major depression and GAD—two disorders that are strongly

saturated with general factor variance—are highly comorbid with one another (e.g., Krueger, 1999; Mineka et al., 1998; Vollebergh et al., 2001). The latter prediction has proven to be much more problematic, however. For instance, specific phobia and social phobia—disorders containing a lesser amount of nonspecific variance—also are strongly comorbid (e.g., Krueger, 1999; Magee et al., 1996; Vollebergh et al., 2001). These data establish that more than one nonspecific factor is required to model comorbidity adequately.

In this regard, the model's ability to capture observed comorbidities can be improved by taking into account the influence of the positive affectivity and anxious arousal dimensions. For instance, the shared component of low positive affectivity likely contributes to the comorbidity between major depression and social phobia, whereas a common element of anxious arousal may be partly responsible for the observed covariation between panic disorder and PTSD (Brown, Campbell, et al., 2001; Brown et al., 1998). Even so, however, the model still fails to account fully for the overlap between lower distress disorders such as specific phobia and social phobia. Of course, the model could be further enhanced through the identification of additional dimensions, which would enable it to provide a better fit to the empirical evidence. It is simpler, however, to analyze the comorbidity data directly and then create a structural model that captures these covariations as accurately as possible. This is the strategy I will follow to articulate a quantitative structural model for *DSM-V*.

A Quantitative Hierarchical Model for *DSM-V*

Rethinking the Diagnostic Classes

Necessity of a higher order factor. This review of the structural/comorbidity data has revealed some important findings that need to be incorporated into *DSM-V*. To facilitate the creation of a quantitative hierarchical structure that more accurately captures the empirical data, I will highlight two basic problems with the existing *DSM-IV* nosology depicted in Figures 1 and 2. The first problem is both obvious and easily

corrected, namely, that this organizational scheme fails to capture the fact that the mood disorders—or, at least, the unipolar syndromes of major depression and dysthymic disorder—are substantially related to the anxiety disorders. This problem can be addressed simply and easily by adding a general higher order factor that is linked to both types of disorder; in this revised model, the current mood and anxiety disorders would be reconceptualized as subclasses within a still broader diagnostic class. This broader class clearly would need to be given a nonspecific label, such as “emotional disorders” or “internalizing disorders”.

The problem of GAD. The second problem is much more serious and has profound implications for *DSM-V*. Simply put, the two current diagnostic classes (i.e., the mood disorders and the anxiety disorders) lack structural coherence and cannot be maintained as basic organizing elements in a quantitative hierarchical structure. The most glaring manifestation of this problem involves the current placement of GAD within the anxiety disorders. As noted earlier, GAD is very strongly linked to the unipolar mood disorders, both phenotypically and genotypically (see Kendler et al., 2003; Mineka et al., 1998). For instance, analyses of twin data consistently have found that major depression and GAD are genetically indistinguishable; that is, the genetic correlation between them essentially is unity, indicating that they reflect a single, common genetic diathesis (Kendler, 1996; Kendler, Neale, Kessler, Heath, & Eaves, 1992; Roy, Neale, Pedersen, Mathé, & Kendler, 1995). It is noteworthy, moreover, that antidepressant drugs have been shown to be effective in the treatment of GAD (e.g., Gorman, 2002; Rivas-Vasquez, 2001).

At the phenotypic level, Krueger (1999) reported that a lifetime diagnosis of GAD had tetrachoric correlations of .64 and .59 with dysthymia and major depression, respectively, in the NCS data; corresponding values (based on 12-month diagnoses) in the Australian National Survey of Mental Health and Well-Being were .66 and .69, respectively (T. Slade, personal communication, July 27, 2004). Similarly, analyses of

the NEMESIS data (again based on 12-month diagnoses) revealed that GAD correlated .68 and .67 with major depression and dysthymia, respectively, in Wave 1; the corresponding values in Wave 2 were .70 and .70, respectively (W. A. Vollebergh, personal communication, December 15, 2003). Finally, Brown, DiNardo, et al. (2001) reported that GAD diagnostic disagreements very frequently involved the mood disorders. They noted that this finding is consistent with other data indicating that “the mood disorders pose a more significant boundary issue for GAD than do other anxiety disorders.” (p. 55)

Indeed, the structural data indicate that GAD actually is more strongly related to the unipolar mood disorders than to other anxiety disorders. For instance, Brown et al. (1998) used confirmatory factor analysis to estimate the latent correlations among major depression and various anxiety disorders. GAD correlated more strongly with depression ($r = .63$) than with OCD ($r = .52$), panic/agoraphobia ($r = .50$), and social phobia ($r = .37$).

Krueger (1999) conducted an extensive series of confirmatory factor analyses of *DSM-III-R* diagnoses in the NCS data. He found that a three-factor model—consisting of Externalizing (alcohol dependence, drug dependence, antisocial personality disorder), Anxious-Misery (major depression, dysthymia, GAD), and Fear (panic disorder, agoraphobia, social phobia, simple phobia)—best fit the data; the latter two factors were strongly correlated and so defined a higher order “Internalizing” dimension. For our purposes, the crucial finding is GAD formed part of the Anxious-Misery dimension with major depression and dysthymia, rather than defining a pure anxiety factor with the other assessed anxiety disorders (Krueger, 1999).

It is noteworthy, moreover, that this model (a) replicated across both lifetime and 12-month diagnoses and (b) fit the data better than a structure based on the traditional *DSM* classification, in which the anxiety disorders marked one factor, and depression and dysthymia defined the other. Krueger’s results subsequently were replicated by

Vollebergh et al. (2001) in confirmatory factor analyses of the NEMESIS data. Once again, the model fits were better when GAD was grouped with depression and dysthymia than with the other anxiety disorders. Finally, structural analyses of diagnostic data from the Australian National Survey of Mental Health and Well-Being again established that GAD defined a common anxious-misery factor with depression and dysthymia, rather than loading with other anxiety disorders (T. Slade, personal communication, July 27, 2004). Thus, this same basic structural pattern now has been identified in large national epidemiological samples in the United States, The Netherlands, and Australia.

Furthermore, Kendler et al. (2003) used data from the Virginia Twin Registry to examine major sources of genetic risk that give rise to common psychiatric disorders. They concluded that “The structure of these genetic risk factors bears a conspicuous resemblance to the phenotypic structure of adult psychiatric disorders proposed by Krueger et al. and Vollebergh et al.” (p. 935) Their proposed structural model of these genetic risk factors again reflected the presence of three underlying dimensions: Externalizing (defined by alcohol dependence, other drug abuse or dependence, adult antisocial behavior, and conduct disorder), Anxious-Misery (marked by major depression and GAD, with a lesser link to panic disorder), and Fear (defined by situational phobia, animal phobia, and panic disorder) (see Kendler et al., 2003, Figure 3).

An Alternative Arrangement of the Disorders

Initial Considerations. The factor structure originally identified by Krueger (1999)—and that subsequently has been replicated in both phenotypic (e.g., Vollebergh et al., 2001) and genotypic (Kendler et al., 2003) analyses—provides a valuable starting point for the creation of a quantitative hierarchical scheme for *DSM-V*. As just noted, a major change in this alternative structure is that GAD needs to be placed in a new cluster with major depression and dysthymic disorder, rather than retaining its traditional classification with the other anxiety disorders. This new diagnostic group was labeled “Anxious-Misery” by previous investigators (Kendler et al., 2003; Krueger, 1999;

Vollebergh et al., 2001), but I will refer to these syndromes here as the “distress disorders” to emphasize the fact that they all involve the experience of pervasive subjective distress. Indeed, these disorders all contain a very large component of non-specific negative affectivity (see Mineka et al., 1998; Watson, Gamez, & Simms, in press).

Krueger (1999) and Vollebergh et al. (2001) term the other higher order dimension—which is defined by panic, agoraphobia, social phobia, and specific phobia in their data—as “fear”. This appears to be an accurate label, so I will use the term “fear disorders” to describe this second cluster.

Extending the model to other disorders. My review thus far has established that the current *DSM-IV* nosology can be improved by (a) adding an overarching diagnostic category of emotional (or internalizing) disorders and (b) transferring GAD from the anxiety disorders to the distress disorders. Although this is a promising start, these earlier structural analyses failed to examine the full range of mood and anxiety disorders. In order to develop a comprehensive taxonomy for *DSM-V*, it obviously is necessary to consider the placement of other key disorders, including (a) PTSD (and the related syndrome of acute stress disorder), (b) OCD, and (c) the bipolar disorders. I therefore turn to an examination of the available evidence regarding each of these disorders. This review will reveal additional problems with the current nosology that need to be addressed in *DSM-V*.

Locating PTSD in the Taxonomy

Acute stress disorder. Acute stress disorder (ASD) was newly added in *DSM-IV*. ASD reflects initial stress reactions during the first month following a traumatic event. Its inclusion in *DSM-IV* proved to be controversial, however. One major objection was the emphasis on dissociative symptoms (e.g., derealization, depersonalization, dissociative amnesia) in the diagnostic criteria, which was based more on theoretical considerations than on empirical evidence (Harvey & Bryant, 2002). Moreover, the

diagnosis itself has proven to be problematic. Brewin, Andrews, and Rose (2003) reported a very high level of comorbidity between ASD and PTSD: Specifically, 28 of the 30 individuals (93.3%) who met criteria for ASD also met criteria for PTSD. Based on these results, Brewin et al. concluded: “The high level of overlap between acute stress disorder and PTSD calls into question whether, as presently formulated, they represent distinct diagnoses.” (p. 783) Similarly, after reviewing the literature, Harvey and Bryant (2002) concluded that “Our review suggests that the theoretical and empirical support for the ASD is flawed and challenges the basis for its continued use.” (p. 886) Finally, this literature has generated very little structural data. Therefore, it will not be considered further.

PTSD. The classification of PTSD presents intriguing possibilities because of the marked heterogeneity of its symptom criteria. Cox et al. (2002), for instance, point out that it is comprised of elements that appear to characterize both the distress disorders (e.g., Criterion C symptoms such as markedly diminished interest in significant activities) and the fear disorders (e.g., Criterion D symptoms of autonomic arousal). How, then, should PTSD be classified in this alternative *DSM-V* taxonomy?

The available data are sparse, because PTSD was not included in the analyses of Krueger (1999), Vollebergh et al. (2001), or Kendler et al. (2003). Fortunately, Cox et al. (2002) reanalyzed the NCS data using a large subsample ($N = 5,877$) on whom PTSD diagnostic information was available. Their results indicated that PTSD should be grouped with the distress disorders rather than with the fear disorders, and I will follow that classification here. I must acknowledge, however, that PTSD was a relatively weak marker of this dimension: Whereas the factor loadings for the other three distress disorders ranged from $-.64$ to $-.83$, the loading for PTSD was only $-.39$ (see Cox et al., 2002, Table 2). It is noteworthy, moreover, that factor analyses of the Australian National Survey of Mental Health and Well-Being data have revealed the same basic pattern: That is, PTSD again was a significant marker of the Distress/Anxious-Misery

dimension that also was defined by major depression, GAD, and dysthymia; as in Cox et al., however, it loaded less strongly on this factor than these other disorders (T. Slade, personal communication, July 27, 2004).

Thus, the Australian and NCS data both suggest some additional complexities in the placement of PTSD within the hierarchy: Although it loads on the Anxious-Misery factor, it is not as strong a marker of this dimension as the other distress disorders. As I suggested earlier, the classification of this disorder is further complicated by the distinctive qualities of the individual PTSD symptom dimensions. In this regard, Simms et al. (2002) conducted a series of confirmatory factor analyses in samples of deployed Gulf War veterans ($N = 1,896$) and nondeployed controls ($N = 1,799$). The same 4-factor structure—consisting of Intrusions, Avoidance, Dysphoria, and Hyperarousal—provided the best fit in both samples. These factors then were used to create symptom scales.

The Dysphoria scale has especially interesting structural implications. Simms et al. (2002) established that this scale is a nonspecific measure of subjective distress. In the deployed veteran sample, for instance, it correlated .80 and .63 with scales assessing depression and generalized anxiety, respectively; moreover, it actually correlated much more strongly with depression than with the other three PTSD symptom scales (r s ranged from .51 to .61). Simms, Watson, and Doebbeling (2003) found similar evidence of nonspecificity in a six-year follow-up assessment: For instance, Dysphoria had the strongest prospective associations with current diagnoses of major depression ($r = .31$) and GAD ($r = .22$). On the basis of these data, it seems reasonable to conclude that this Dysphoria dimension has a substantial influence on the overlap between PTSD and distress disorders such as major depression and GAD. This, in turn, suggests that it may be primarily responsible for PTSD's significant loading on the Anxious-Misery factor in the NCS and Australian data.

In contrast, the other PTSD symptom dimensions are less clearly linked to the distress disorders. Indeed, as was suggested by Cox et al. (2002), they contain many

elements that appear to be more broadly characteristic of the fear disorders. For example, the Avoidance dimension contains symptoms (avoiding thoughts of trauma, avoiding reminders of trauma) that reflect classic responses to phobic stimuli. Similarly, the content of the Hyperarousal factor (“feeling jumpy or easily startled”; “being ‘superalert’ or watchful or on guard”) suggests that it basically taps anxious arousal, which has been found to be a core symptom feature of panic disorder (Brown et al., 1998; Mineka et al., 1998).

These symptom-based analyses suggest two important points. First, it would be informative to assess and model these symptom dimensions separately in future structural investigations. Differentiated analyses of this type might help to clarify whether PTSD truly is a distress disorder, a fear disorder, or some combination of the two. Second, any significant changes in the PTSD symptom criteria in *DSM-V* might well alter its optimal placement in a structural hierarchy. For instance, any changes that enhance the salience of this Dysphoria component can be expected to strengthen the links between PTSD and distress disorders such as major depression and GAD; conversely, modifications that diminish its importance are likely to weaken these links. This again illustrates my earlier point that structural analyses can be highly sensitive to changes in diagnostic criteria.

The Placement of OCD

Basic OCD analyses. As noted earlier, OCD shows a relatively low prevalence rate in general populations and, therefore, often is excluded from structural analyses (e.g., Vollebergh et al., 2001). Accordingly, structural data are relatively limited for this disorder. However, OCD was assessed in the Australian National Survey of Mental Health and Well-Being. Factor analyses of these data indicated that OCD was a significant marker of a Fear dimension that also was defined by panic disorder, agoraphobia, and social phobia (T. Slade, personal communication, July 27, 2004). Paralleling the earlier situation with PTSD, however, OCD loaded less strongly on this factor than these other disorders.

We have corroborating data that also suggest that OCD should be classified with the fear disorders. We investigated a sample of military veterans who served during the 1991 Gulf War. The participants in this follow-up study were selected from a larger initial sample (Doebbeling et al., 2002; Simms et al., 2002) to investigate three common problems among Gulf War veterans: cognitive dysfunction, chronic widespread pain, and depression (for more details, see Simms et al., 2003; Watson, Gamez, & Simms, in press). Current symptoms and diagnoses were obtained from all participants using the Structured Clinical Interview for DSM-IV (SCID; First, Spitzer, Gibbon, & Williams, 1997). I conducted analyses on 11 symptoms derived from the SCID. Nine of these symptoms were taken from the SCID Screening Module, which assesses key symptoms of selected disorders using a three-point scale (1 = absent/no, 2 = subthreshold, 3 = present/yes). Seven of these questions are symptoms that directly relate to various anxiety disorders (e.g., panic attacks, social anxiety, specific phobias); the other two reflect substance abuse (excessive drinking, drug use) and so should define the Externalizing dimension that repeatedly has emerged in previous structural analyses (e.g., Kendler et al., 2003; Krueger, 1999; Vollebergh et al., 2001). The final two symptoms (depressed mood, loss of interest or pleasure) were taken from the SCID current depressive episode module.

I computed polychoric correlations among these symptoms (for a discussion of the use of polychoric versus Pearson correlations, see Krueger, 1999; Watson & Tellegen, 1999) and subjected them to a principal factor analysis using EQS (Bentler & Wu, 1995). I extracted three factors, which then were rotated to oblique simple structure using oblimin. These loadings are presented in Table 3. It is noteworthy that the resulting structure closely replicates the 3-factor structure that has been reported in previous disorder-based analyses (Kendler et al., 2003; Krueger, 1999; Vollebergh et al., 2001). That is, the three factors in Table 3 clearly can be identified as Anxious-Misery (defined by both depressed mood and nervousness/anxiety), Externalizing, and Fear, respectively.

Consistent with previous research, the Anxious-Misery and Fear factors were strongly correlated ($r = .52$), which again demonstrates the presence of a general overarching dimension that subsumes both the mood and anxiety disorders; both of these factors were only weakly related to Externalizing, however, ($r_s = .12$ and $.16$ for Anxious-Misery and Fear, respectively). Finally, the most notable finding in Table 3 is that the two assessed indicators of OCD—compulsions and obsessive intrusions—both were clear markers of the Fear factor, along with symptoms reflecting panic, agoraphobia, social phobia, and specific phobia.

Unfortunately, however, these structural findings do not replicate in analyses of the NEMESIS data. Indeed, OCD failed to emerge as a clear marker of the Fear dimension at either Time 1 or Time 2 in both exploratory and confirmatory factor analyses of these data (Watson, de Graaf, Nolen, & Vollebergh, 2004). In light of these contradictory findings, it is impossible to specify the optimal placement of OCD at this time. This clearly represents a crucial task for future research.

Symptom dimensions of OCD. As with PTSD, the classification of OCD is complicated by the heterogeneity of this disorder. Recent structural analyses have documented the existence of at least four replicable symptom dimensions within existing measures of OCD. Leckman et al. (1997) first identified these symptom dimensions in exploratory factor analyses of the Yale-Brown Obsessive Compulsive Scale (Y-BOCS; Goodman et al., 1989) in a combined sample of nearly 300 OCD patients. Their analyses revealed four replicable factors: (1) obsessions and checking, (2) symmetry and ordering, (3) cleanliness and washing, and (4) hoarding. Summerfeldt et al. (1999) subsequently established that this four-factor model fit the data well in a series of confirmatory factor analyses in more than 200 OCD patients. Finally, it is noteworthy that these same four symptom factors—checking, washing, ordering, and hoarding—have been identified in analyses of the Obsessive-Compulsive Inventory (OCI; Foa, Kozak, Salkovskis, Coles, & Amir, 1998), the revised OCI (OCI-R; Foa et al., 2002), and the Schedule of

Compulsions, Obsessions, and Pathological Impulses (SCOPI; Watson & Wu, in press) (see Foa et al., 2002; Watson & Wu, in press; Wu & Watson, 2003).

Furthermore, these symptom dimensions have distinctive correlates and are differentially related to other types of psychopathology. For instance, ordering/symmetry symptoms show particularly strong associations with Tourette's syndrome (Baer, 1994; Eapen, Robertson, Alsobrook, & Pauls, 1997). In a related vein, Watson et al. (2004) found that dissociation measures correlated much more strongly with checking than with cleaning, ordering, and hoarding; moreover, this pattern replicated across both student and outpatient samples. More generally, checking symptoms appear to be somewhat more nonspecific and show greater overlap with neuroticism/negative affectivity (Watson, Gamez, and Simms, in press) and other types of psychopathology (including depression) than these other symptom dimensions (Watson et al., 2004; Wu & Watson, in press). Consequently, it may be helpful to assess and model these symptom dimensions separately in future structural investigations. Dimensional, symptom-based analyses would circumvent problems associated with the low-base rate of the full syndrome in general population samples and would clarify its placement within the structural hierarchy.

The Bipolar Disorders

As with OCD, structural investigations of the bipolar disorders have been hampered by their very low base rates in the general population (Krueger, 1999; Vollebergh et al., 2001). Structural analyses are further complicated by hierarchical exclusion rules that— if strictly applied—preclude the simultaneous diagnosis of most unipolar and bipolar mood disorders. Indeed, the *DSM-IV* text explicitly states that “A history of a Manic, Mixed, or Hypomanic Episode precludes the diagnosis of Major Depressive Disorder.” (APA, 1994, pp. 342-343)

Because of these problems, the bipolar disorders have not been modeled in any previous structural investigation of this domain. To get a preliminary sense of where they

might be located within the hierarchy, I conducted a structural analysis of the NCS data, using the large subsample ($N= 5,877$) who received diagnoses of PTSD (see Cox et al., 2002). Following the approach used by Krueger (1999), I computed tetrachoric correlations among lifetime *DSM-III-R* diagnoses, which were scored without the use of any hierarchical exclusion rules. I subjected these data to a principal factor analysis; I extracted three factors, which were rotated using oblimin. These rotated loadings are presented in Table 4. As with previous structural analyses of the NCS data (Cox et al., 2002; Krueger, 1999), the three extracted factors clearly can be identified as Anxious-Misery, Externalizing, and Fear, respectively. It is noteworthy that bipolar disorder fails to emerge as a clear marker of any of these dimensions: Indeed, it has weak and virtually identical loadings on Anxious-Misery (.33), Externalizing (.29), and Fear (.29).

Consequently, these data provide no clear basis for locating the bipolar disorders within either Anxious-Misery or Fear. I therefore will keep them separate as a third diagnostic subclass within the broader category of the emotional disorders.

A Summary Model

Figure 3 presents a quantitative hierarchical arrangement of the current mood and anxiety disorders, incorporating all of the evidence I have reviewed. For reasons that I discussed earlier, this revised model does not include either ASD or OCD. It also excludes possible subtypes of existing disorders (such as specific phobia), which are beyond the scope of this paper and fully merit a separate review of their own. The remaining disorders are grouped into three broad subclasses: the distress disorders (major depression, dysthymic disorder, GAD, PTSD), the fear disorders (panic, agoraphobia, social phobia, specific phobia), and the bipolar disorders (bipolar I, bipolar II, cyclothymia). These subclasses, of course, are significantly interrelated (see Kendler et al., 2003; Krueger, 1999; Vollebergh et al., 2001); this, in turn, creates an overarching higher order construct, which is labeled “emotional disorders” in Figure 3. This alternative hierarchical scheme more accurately captures empirical patterns of

covariation/comorbidity than the current *DSM-IV* nosology. Although several aspects of this structure currently are unclear and require further investigation, this model provides a useful starting point for a revised organization of these diagnoses in *DSM-V*.

Final Considerations

Refining the Taxonomy

My review has identified several unresolved structural issues that need to be clarified in future research. For instance, structural data for PTSD and OCD still are relatively sparse. Accordingly, the classification of these disorders requires additional investigation in both phenotypic and genotypic analyses.

Moreover, the placement of these disorders is complicated by the heterogeneous nature of their symptoms. Both disorders consistently have been shown to be comprised of several distinguishable types of symptoms; furthermore, these symptom dimensions show distinctive patterns of correlations with other variables. For example, the classification of PTSD as a distress disorder may largely reflect the influence of its dysphoria subfactor; as discussed earlier, this symptom cluster is highly nonspecific and correlates strongly with depression and generalized anxiety (Simms et al., 2002, 2003). Once the influence of this nonspecific dimension is removed, the remaining PTSD symptom clusters may fall quite differently in the hierarchy. Similarly, the checking component of OCD appears to be more nonspecific and shows greater overlap with other types of psychopathology (including depression) than its other symptom dimensions (Watson et al., 2004; Wu & Watson, in press). These results highlight the importance of internal structural analyses of existing *DSM* syndromes. Further investigation of these symptom dimensions may play a key role in clarifying the placement of these disorders within the structural hierarchy.

Despite these problems and qualifications, we already have a good overall sense of the structure defined by the current anxiety and unipolar mood disorders (see Kendler et al., 2003; Krueger, 1999; Vollebergh et al., 2001). In contrast, good structural data are

lacking regarding the proper placement of the bipolar mood disorders. In Figure 3, I grouped these syndromes into a third diagnostic subclass within the broader category of the emotional disorders. In the absence of any compelling structural data, I believe this represents the most reasonable classification scheme, in that it preserves the traditional link between the unipolar and bipolar mood disorders (which have been components of the same diagnostic class since *DSM-III*). Obviously, however, much future work is needed to explicate the optimal taxonomic placement of the bipolar disorders.

Broadening the Taxonomy

As discussed earlier, the mood and anxiety disorders both show extensive comorbidity with other types of psychopathology, including the substance-use, eating, somatoform, attention-deficit, and personality disorders (Mineka et al., 1998; Widiger & Clark, 2000). These data establish that a comprehensive structural scheme should not be restricted to the mood and anxiety disorders, but should subsume and model syndromes that currently are placed in other diagnostic classes.

As one example, it now is clear that OCD shares important features with a range of related disorders. These overlapping features include similar symptoms, common etiological factors, and responsiveness to the same types of drug treatments. Collectively, this family of related syndromes has been labeled the “obsessive-compulsive spectrum disorders” (e.g., Black, 1998; Goldsmith, Shapira, Phillips, & McElroy, 1998; Hollander & Benzaquen, 1997). Hollander and Rosen (2000) suggest that these spectrum disorders can be classified into three broad subtypes: (1) disorders of impulse control (e.g., kleptomania, pathological gambling, sexual addictions), (2) syndromes characterized by an exaggerated preoccupation with body appearance or bodily sensations (e.g., body dysmorphic disorder, hypochondriasis), and (3) neurological disorders involving repetitive behaviors (e.g., autism, Tourette’s syndrome). Watson et al. (2004) extended this literature by establishing that OCD symptoms also are strongly related to individual differences in dissociation. These data raise the intriguing possibility that OCD actually

might form part of a separate diagnostic class with other types of psychopathology, such as the dissociative and impulse control disorders.

Dimensional versus Categorical Classification

A final issue that needs to be addressed is the underlying nature of this proposed taxonomy. The structure depicted in Figure 3 can be applied either categorically (i.e., as dichotomous diagnoses) or dimensionally (e.g., as continuous measures of psychopathology). Which approach is preferable?

The relative validity of these two approaches rests on a number of empirical considerations (for discussions, see Widiger & Clark, 2000; Widiger & Samuel, this issue). An especially important issue concerns the nature of the underlying distributions. On the one hand, evidence of a continuous distribution—with no clear demarcation between normality and abnormality—strongly supports the validity of a dimensional approach; on the other hand, data suggesting the existence of discrete, discontinuous types favors the utility of a categorical system. In recent years, a variety of sophisticated statistical techniques—including latent class analysis and taxometric analysis—have been used to address this basic issue (see Cole, 2004; Kraemer, Noda, & O’Hara, 2004). Although the relevant evidence still is relatively limited, the available findings tend to support a dimensional view of this domain. Thus, taxometric analyses of both chronic worry and posttraumatic stress have supported the presence of continuous dimensions (A. M. Ruscio, Borkovec, & Ruscio, 2001; A. M. Ruscio, Ruscio, & Keane, 2002). Analyses of depression have yielded mixed results, but generally have found evidence of continuity rather than discontinuity (Haslam & Beck, 1994; A. M. Ruscio & Ruscio, 2002; J. Ruscio & Ruscio, 2000; Solomon, Haaga, & Arnow, 2001; Whisman & Pinto, 1997).

Dimensional approaches also offer two important advantages that are worth noting briefly. First, dimensional schemes yield a greater amount of clinically-relevant information than simpler categorical models. In particular, dimensional systems allow one to model the severity of dysfunction, not simply its presence versus absence. This is

a key advantage, because severity has been found to be a significant predictor of a broad range of clinical phenomena, including both (a) comorbidity and (b) the course and chronicity of disorder (see Clark et al., 1995).

Second, continuous scores tend to be more stable over time and display higher levels of reliability than dichotomous measures (see Widiger, 1992; Widiger & Clark, 2000). This is because dimensional scores are largely unaffected by relatively minor shifts in psychopathology; in contrast, even modest changes can move an individual either above or below a dichotomous diagnostic threshold. As one simple example, suppose that a patient reported five symptoms of depression at Time 1, but only four at Time 2. In a categorical system, this individual would be completely discordant, meeting criteria for major depression in the first assessment but not in the second; in a dimensional system, however, this difference would be quantitatively indexed as a single point (i.e., a score of 5 at Time 1 and a score of 4 at Time 2).

This difference between categorical and dimensional assessment becomes particularly striking when the underlying distribution is continuous. Indeed, a large body of evidence indicates that the artificial dichotomization of continuous measures leads to substantial losses in reliability and validity (Cohen, 1983; Watson, 2003a; Widiger, 1992). For instance, I investigated the temporal stability of dissociation measures across a two-month interval (Watson, 2003a). A continuous measure of dissociation produced a retest correlation of .62 across this time span; its stability was dramatically reduced (correlations ranged between .27 and .34), however, when it was artificially dichotomized in various ways (see Watson, 2003a, Table 3).

Despite these advantages, it may be argued that categorical constructs are more conducive to clinical decision-making than are continuous dimensions, given that these decisions often are dichotomous in nature (e.g., whether or not to treat a patient with an anti-depressant drug). Although it may seem plausible at first glance, I believe this argument is unsound. Just as one can easily move from (a) the quantitative assessment

of systolic and diastolic blood pressure to (b) the dichotomous classification of hypertension, one always has the option of developing cut scores that transform continuous dimensions into categorical judgments, should that prove to be desirable (McFall & Treat, 1999). It should be kept in mind, moreover, that it is much easier to move from complexity to simplicity than vice versa: Once information is eliminated from a system, it typically is gone forever. Consequently, the best strategy is to start by obtaining the maximum amount of information possible (i.e., through quantitative dimensions), and then to simplify things as needed.

Conclusion

DSM-IV currently classifies the mood and anxiety disorders according to the subjective criterion of “shared phenomenological features.” In this paper, I have argued that we now have sufficient knowledge to eliminate this rationally-based system and replace it with an empirically-based structure that reflects the actual—not the assumed—similarities among different disorders. Although much work remains to be done, we have made substantial progress in creating a quantitative hierarchical model. My review of the evidence establishes that the current mood and anxiety disorders should be subsumed together in an overarching class of emotional disorders, which can be decomposed into three subclasses: the bipolar disorders (bipolar I, bipolar II, cyclothymia), the distress disorders (major depression, dysthymic disorder, GAD, PTSD) and the fear disorders (panic disorder, agoraphobia, social phobia, specific phobia). We now should commit ourselves to completing the task of explicating this model in its entirety; with this commitment, a fully articulated hierarchical structure is within our reach.

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Table 1

Correlations between the PANAS-X Fear, Sadness, and Joviality Scales in Various Samples

Sample	<i>N</i>	Fear- Sadness	Fear- Joviality	Sadness- Joviality
<i>Self-Ratings</i>				
SMU Students				
Moment	1,027	.55	-.07	-.34
Daily	1,007	.56	-.02	-.28
Past Few Days	289	.63	-.07	-.28
Past Week	1,278	.69	-.07	-.38
Past Few Weeks	678	.57	-.13	-.34
Past Month	1,006	.62	-.12	-.32
Past Year	315	.53	-.16	-.41
General	1,657	.57	-.03	-.30
<i>Weighted Mean r</i>	<i>7,257</i>	<i>.58</i>	<i>-.07</i>	<i>-.33</i>
Iowa Students				
Past Few Days	502	.63	-.26	-.48
General	598	.58	-.15	-.40
<i>Weighted Mean r</i>	<i>1,100</i>	<i>.60</i>	<i>-.20</i>	<i>-.44</i>
Dallas-area Adults				
Past Week	328	.58	-.23	-.48
<i>Other-Ratings (General)</i>				
Friendship Dyads	558	.50	-.07	-.37
Dating Couples	272	.59	-.23	-.44
Married Couples	148	.62	-.23	-.48
<i>Weighted Mean r</i>	<i>978</i>	<i>.54</i>	<i>-.14</i>	<i>-.41</i>

Note. PANAS-X = Expanded Form of the Positive and Negative Affect Schedule. SMU = Southern Methodist University.

Table 2

Correlations between the General Distress: Depression and General Distress: Anxiety Scales of the Mood and Anxiety Symptom Questionnaire (MASQ)

Study	Sample	<i>N</i>	Correlation
<i>Non-Distressed Samples</i>			
Clark, Cook & Snow (1998)	Medical Patients	71	.76
Gilbert et al. (2002)	College Students	193	.71
Nitschke et al. (2001)	College Students	783	.64
Ready & Keogh (1997)	College Students	98	.79
Watson, Suls & Haig (2002)	College Students	287	.74
	College Students	346	.66
Watson & Walker (1996)	Ex-Students	334	.69
Watson, Weber et al. (1995)	College Students	516	.68
	College Students	381	.67
	College Students	516	.61
	Adults	328	.69
Wu (2003)	College Students	419	.75
<i>Weighted Mean r</i>		4,272	.68
<i>Distressed Samples</i>			
Clark, Cook & Snow (1998)	Depressed Inpatients	51	.37
Gilbert et al. (2002)	Psychiatric Inpatients	81	.77
Marcus, Hamlin & Lyons (2001)	Male Inmates	142	.73
Watson, Simms & Gamez (2004)	Military Veterans	568	.73
Watson, Weber et al. (1995)	VA Patients	470	.78
Wu (2003)	Psychiatric Outpatients	102	.78
	OCD Patients	52	.78
Yovel & Mineka (2004)	Distressed Students	123	.69
<i>Weighted Mean r</i>		1,589	.74

Note. VA = Veterans Administration. OCD = obsessive-compulsive disorder.

Table 3

Oblimin-Rotated Factor Loadings of SCID-based Symptoms in the Gulf War Sample

Symptom	Factor 1	Factor 2	Factor 3
Depressed mood	<u>.88</u>	-.08	-.04
Loss of interest or pleasure	<u>.79</u>	-.03	.11
Nervousness/anxiety	<u>.69</u>	.10	.05
Excessive drinking	-.20	<u>.73</u>	.01
Drug Use	.19	<u>.67</u>	-.01
Specific phobias	-.15	-.04	<u>.64</u>
Compulsions	.08	.10	<u>.53</u>
Panic attacks	.31	.07	<u>.50</u>
Social anxiety	.26	-.17	<u>.49</u>
Agoraphobic fears	.36	.20	<u>.44</u>
Obsessive intrusions	.23	.10	<u>.43</u>

Note. N = 559. Loadings of |.40| and greater are highlighted.

Table 4

Oblimin-Rotated Factor Loadings of Lifetime DSM-III-R Diagnoses from the National Comorbidity Survey

Disorder	Factor 1	Factor 2	Factor 3
Dysthymia	<u>.80</u>	.01	-.13
Major Depressive Episode	<u>.75</u>	.00	.02
Generalized Anxiety Disorder	<u>.62</u>	-.01	.15
Posttraumatic Stress Disorder	<u>.40</u>	.15	.17
Alcohol Dependence	-.02	<u>.76</u>	-.05
Antisocial Personality Disorder	.01	<u>.75</u>	-.03
Drug Dependence	.01	<u>.74</u>	.04
Simple Phobia	-.02	-.04	<u>.74</u>
Agoraphobia	.10	-.04	<u>.68</u>
Social Phobia	-.09	.08	<u>.67</u>
Panic Disorder	.31	-.06	<u>.50</u>
Bipolar Disorder	.33	.29	.29

Note. N = 5,877. Loadings of |.40| and greater are highlighted.

Figure Captions

Figure 1. A schematic structural model of the *DSM-IV* mood disorders.

Figure 2. A schematic structural model of the *DSM-IV* anxiety disorders. GAD = generalized anxiety disorder. OCD = obsessive-compulsive disorder. PTSD = posttraumatic stress disorder. ASD = acute stress disorder. BII = blood-injection-injury. Natural Environ = natural environment. Sit = situational. Intrus = intrusions. Avoid/Numb = avoidance/numbing.

Figure 3. A revised structural model of the mood and anxiety disorders for *DSM-V*. BPI I = bipolar I disorder. BPD II = bipolar II disorder. CT = cyclothymia. MDD = major depressive disorder. DD = dysthymic disorder. GAD = generalized anxiety disorder. PTSD = posttraumatic stress disorder.





