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Structural Relationships Among Dimensions of the *DSM–IV* Anxiety and Mood Disorders and Dimensions of Negative Affect, Positive Affect, and Autonomic Arousal

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Using outpatients with anxiety and mood disorders ($N = 350$), the authors tested several models of the structural relationships of dimensions of key features of selected emotional disorders and dimensions of the tripartite model of anxiety and depression. Results supported the discriminant validity of the 5 symptom domains examined (mood disorders; generalized anxiety disorder, GAD; panic disorder; obsessive–compulsive disorder; social phobia). Of various structural models evaluated, the best fitting involved a structure consistent with the tripartite model (e.g., the higher order factors, negative affect and positive affect, influenced emotional disorder factors in the expected manner). The latent factor, GAD, influenced the latent factor, autonomic arousal, in a direction consistent with recent laboratory findings (autonomic suppression). Findings are discussed in the context of the growing literature on higher order trait dimensions (e.g., negative affect) that may be of considerable importance to the understanding of the pathogenesis, course, and co-occurrence of emotional disorders.

Over the past few decades, the number of diagnostic categories has increased markedly with each edition of the major classification systems for mental disorders (e.g., the *Diagnostic and Statistical Manual of Mental Disorders, DSM*, and the *International Classification of Diseases*, World Health Organization, 1993). For instance, with regard to the anxiety disorders alone, only 3 relevant categories existed in the second edition of the *DSM* (*DSM–II;* American Psychiatric Association, 1968), compared with the 12 categories that currently exist in the fourth edition of this system (*DSM–IV;* American Psychiatric Association, 1994). This increase could be viewed as signifying greater precision in the classification of disorders. However, many researchers (e.g., Andrews, 1996) have expressed concern that the expansion of our nosologies has come at the expense of less empirical consideration of shared or overlapping features of emotional disorders that, relative to unique features of specific disorders, may have far greater significance in the understanding of the prevention, etiology, and course of disorders, and in predicting their response to treatment.

Of further concern is the possibility that our classification systems have become overly precise to the point that they are now erroneously distinguishing symptoms and disorders that actually reflect inconsequential variations of broader, underlying syndromes. Findings indicating that a variety of *DSM* disorders respond similarly to the same drug or psychosocial treatment have been offered in support of this position (e.g., Hudson & Pope, 1990; Tyrer et al., 1988). Moreover, consistent findings of high comorbidity among anxiety and mood disorders (T. A. Brown & Barlow, 1992), as well as emerging data that comorbid diagnoses often remit after psychosocial treatment of another anxiety disorder (Borkovec, Abel, & Newman, 1995; T. A. Brown, Antony, & Barlow, 1995), may also be reflective of poor discriminant validity of current classifications. However, conclusions about the validity of current classification systems cannot be drawn from the descriptive evidence of high rates of co-occurrence and covariation among disorders, given the multiple conceptual explanations for diagnostic comorbidity (cf. Blashfield, 1990; Frances, Widiger, & Fyer, 1990). Indeed, these explanations are sufficiently wide-ranging to either support or invalidate present nosologies (e.g., supportive explanation: two disorders co-occur because they share the same diathesis or because the features of one disorder act as risk factors for another disorder; nonsupportive explanation: high comorbidity is due to artificial separation of a broader syndrome or unnecessary overlap in definitional criteria).

Of studies that bear on the validation of the classification of anxiety and mood disorders, the majority have been conducted at the diagnostic level (e.g., family and twin studies; Andrews, Stewart, Morris-Yates, Holt, & Henderson, 1990; Kendler, Neale, Kessler, Heath, & Eaves, 1992) or have examined dimensional

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features within a diagnostic category (e.g., psychometric evaluations of constituent features within a *DSM* disorder; Marten et al., 1993). As we have discussed at length elsewhere (T. A. Brown, 1996; T. A. Brown & Chorpita, 1996), the categorical approach to analysis has many limitations (cf. Livesley, Schroeder, Jackson, & Jang, 1994). For instance, studies conducted at the diagnostic level (e.g., comorbidity, genetic or familial aggregation, across-diagnosis comparisons) are restricted by their adherence to the disorders defined by the classification system; that is, by using diagnoses as the units of analysis, researchers are implicitly accepting or are bound to the nosology they are evaluating. Moreover, in view of evidence that anxiety and depression symptoms operate on a continuum, analyses at the diagnostic level rely largely on data that do not reflect the dimensional nature of these features.[1] Categorization of dimensional variables usually forfeits meaningful information by artificially (and often erroneously) collapsing variability above and below an arbitrary threshold (e.g., presence vs. absence of a *DSM–IV* disorder). Conversely, if assessment were performed at the dimensional level, the interrelationships among symptoms and syndromes could be examined, as could the extent to which the latent structure of these features corresponds to the structure forwarded by major classification systems such as *DSM–IV.*

Although this form of analysis has occurred in some areas of psychopathology (e.g., personality disorders; Moldin, Rice, Erlenmeyer-Kimling, & Squires-Wheeler, 1994), research of this nature for the anxiety and mood disorders has been sparse. However, a recent example in the area of anxiety and mood disorders is a study by Zinbarg and Barlow (1996). In this study, an exploratory factor analysis of various questionnaires of features of anxiety disorders produced a factor structure that was largely consistent with the *DSM–III–R* (*DSM,* 3rd ed., rev.; American Psychiatric Association, 1987) nosology (i.e., Social Anxiety, Generalized Dysphoria, Agoraphobia, Fear of Fear, Obsessions and Compulsions, Simple Fears). Support for *DSM–III–R* was also provided by discriminant function analyses indicating that selected diagnostic groups (defined by principal diagnoses established by structured interviews) evidenced characteristic profiles in factor scores generated from a higher order factor analysis. Although encouraging, as noted by Zinbarg and Barlow, these findings were limited by the preponderant use of self-report measures (e.g., method variance could account, in part, for the structure observed) and the poor representation of mood disorders (e.g., depressive symptoms were assessed by a single measure with a scale under psychometric development). The latter limitation is noteworthy given evidence that mood disorders (i.e., major depression, dysthymia) may pose greater boundary problems for certain anxiety disorders than do other anxiety disorders (cf. T. A. Brown, Marten, & Barlow, 1995; T. A. Brown, Anson, & DiBartolo, 1996).

Unfortunately, specific disorders are often evaluated in isolation (Watson, Clark, & Harkness, 1994). For instance, the associated symptom criterion for *DSM–IV* generalized anxiety disorder was revised (i.e., autonomic arousal symptoms were eliminated) without empirical consideration of how this might further obfuscate its boundary with the mood disorders (T. A. Brown, Barlow, & Liebowitz, 1994). Comprehensive evaluations of the structure and construct validity of anxiety disorders should entail features of both anxiety and depression given the strong relationship and potential overlap of these domains (cf. Kendall & Watson, 1989).

Indeed, although anxiety and depression have historically been regarded as distinct at the conceptual level, dimensional measures of these constructs have evidenced considerable overlap (L. A. Clark & Watson, 1991; Kendall & Watson, 1989). Such findings, as well as data on the high co-occurrence at the syndromal level, have led investigators to question whether clinical anxiety and depression are in fact empirically distinct phenomena. On the basis of a review of the literature, L. A. Clark and Watson (1991) concluded that although anxiety and depression share a significant nonspecific component encompassing general affective distress and other common symptoms, the two constructs can be distinguished by certain unique features. L. A. Clark and Watson proposed a tripartite structure of anxiety and depression consisting of general distress or negative affect (shared by anxiety and depression), physiological hyperarousal (specific to anxiety), and an absence of positive affect (specific to depression). Sophisticated studies have begun to emerge in support of the tripartite structure (e.g., Joiner, Catanzaro, & Laurent, 1996; Watson, Clark, et al., 1995), though most of this work has been in analogue samples or clinical samples in which anxiety and mood disorders were not highly represented (Joiner, 1996; Watson, Weber, et al., 1995).

Nevertheless, in addition to explicating the shared and distinctive features of anxiety and depression, the tripartite model may have considerable importance in the understanding of the pathogenesis of anxiety and mood disorders. For instance, the dimension of negative affect may represent a key vulnerability factor for the development of both anxiety and depression (L. A. Clark, Watson, & Mineka, 1994; Watson & Clark, 1984; Watson et al., 1994). The collective evidence indicates that negative emotionality and related constructs (e.g., neuroticism) are heritable and temporally stable (e.g., Costa & McCrae, 1988; Tellegen et al., 1988; Watson & Clark, 1984). Ultimately, the construct of negative affect may prove empirically consistent with conceptual models (e.g., Barlow, Chorpita, & Turovsky, 1996) and genetic evidence (e.g., Kendler et al., 1992) suggesting that although differentiation among anxiety and mood disorders is warranted (e.g., the disorders differ on important dimensions that have implications for treatment), these syndromes are closely related because they share common diatheses (i.e., biological or trait vulnerabilities). Moreover, these positions provide a compelling account for the high rates of comorbidity observed among anxiety and mood disorders (i.e., the disorders co-occur because of the influence of the same underlying, causal factors). Similarly, low positive affect may operate as a vulnerability dimension specific to depression, although existing data are less compelling relative to the accumulated knowledge on

[1] Nevertheless, the issue of the qualitative versus quantitative nature of emotional disorders continues to be debated in the literature (cf. Flett, Vredenburg, & Krames, 1997). Moreover, although considerable consensus exists for the conclusion that measures of psychopathological symptoms operate dimensionally, expression of symptoms along a continuum alone cannot be taken to confirm or refute the absence of an underlying taxon (e.g., quantitative indicators can have dichotomous latent influences such as the presence or absence of a gene; cf. Meehl, 1995).

negative affect. Current data suggest that the third component of the tripartite model, autonomic arousal, may not be reflective of a trait vulnerability dimension of emotional disorders, although further research is needed (L. A. Clark, Watson, & Mineka, 1994).

With these issues in mind, the present study had several aims. Using a large sample of patients with anxiety and mood disorders (N = 350) who were assessed with a variety of self-report and clinician rating measures, it was predicted that confirmatory factor analysis would support a factor structure that corresponded to the *DSM–IV* typology of selected disorders (panic disorder, generalized anxiety disorder, social phobia, obsessive–compulsive disorder, and mood disorder) over models in which disorders were collapsed. After conducting analyses that were expected to confirm a factor structure consistent with the tripartite model, we comparatively evaluated structural models of the relationships among the *DSM–IV* disorders and tripartite factors. Of these models, it was predicted that the best fitting would entail the following: (a) significant paths from a higher order factor, Negative Affect, to each of the *DSM–IV* disorder factors, in accord with theory and evidence that negative affect represents a vulnerability dimension common to the anxiety and mood disorders (cf. Watson et al., 1994); (b) a significant path from the higher order factor, Positive Affect, to *DSM–IV* Depression, on the basis of evidence that low positive affect is a feature specific to depression and may act as a diathesis to mood disorders; and (c) paths from the *DSM–IV* anxiety disorder factors, but not from *DSM–IV* Depression, to the lower order factor of Autonomic Arousal that are significant and associated with good model fit (consistent with the prediction of the tripartite model that although autonomic arousal is not likely to be a trait vulnerability dimension, it distinguishes the anxiety disorders from the mood disorders; cf. L. A. Clark, Watson, & Mineka, 1994).

Method

Participants

Participants were 350 patients presenting for assessment and treatment at the Center for Stress and Anxiety Disorders, University at Albany, State University of New York. Women constituted the larger portion of the sample (61.7%); the average age of the sample was 35.56 years (SD = 10.96, range = 18–64).[2] Diagnoses were established with the Anxiety Disorders Interview Schedule for *DSM–IV:* Lifetime Version (ADIS-IV-L; Di Nardo, Brown, & Barlow, 1994), a structured interview designed to comprehensively evaluate current and lifetime *DSM–IV* anxiety, mood, and substance use disorders, as well as selected somatoform disorders (e.g., hypochondriasis); it was also designed to be a screen for the presence of other major disorders (e.g., psychosis). Findings of an initial study (N = 72) of the diagnostic reliability of the ADIS-IV-L for principal *DSM–IV* anxiety and mood disorders (i.e., calculated on the basis of two independent interviews) indicated good to excellent levels of interrater agreement (Di Nardo, Brown, Lawton, & Barlow, 1995; κ = .93 for panic disorder and panic disorder with agoraphobia, 1.00 for specific phobia, .83 for generalized anxiety disorder, .90 for obsessive–compulsive disorder, .64 for social phobia, .85 for mood disorder, either major depression or dysthymia). When the patient was deemed to have met criteria for two or more diagnoses, the principal diagnosis was the one that received the highest ADIS-IV-L clinical severity rating (CSR; a 9-point scale ranging from 0, *none,* to 8, *very severely disturbing/disabling*) that indicated the diagnostician's judgment of the degree of distress and interference in functioning associated with the diagnosis. Patients' *DSM–IV* principal diagnoses were as follows: panic disorder with or without agoraphobia (n = 120), generalized anxiety disorder (n = 30), social phobia (n = 52), specific phobia (n = 30), obsessive–compulsive disorder (n = 22), mood disorder (collapsed across major depressive disorder and dysthymia, n = 26), other (e.g., posttraumatic stress disorder, anxiety disorder not otherwise specified, coprincipal diagnoses; n = 70).

Per guidelines of ADIS-IV-L administration and scoring, disorders that met or surpassed the threshold for a formal *DSM–IV* diagnosis were assigned CSRs of 4 (*definitely disturbing*/disabling) or higher (i.e., clinical diagnoses). When the key features of a disorder were present but were not extensive or severe enough to warrant a formal *DSM–IV* diagnosis (or for *DSM–IV* disorders in partial remission), CSRs of 1 to 3 were assigned (subclinical diagnoses). When no features of a given disorder were present, CSRs of 0 were assigned. For the five disorders examined in the present study, the total frequency of their occurrence at the clinical level was as follows: mood disorder (n = 137), generalized anxiety disorder (n = 93), panic disorder/agoraphobia (n = 166), obsessive–compulsive disorder (n = 36), and social phobia (n = 115).

Model Indicators

ADIS-IV-L ratings and questionnaires were collected and analyzed as indicators for the latent variables examined in the structural and measurement models. Latent factors were the *DSM–IV* disorders of Depression (DEP), Generalized Anxiety Disorder (GAD), Panic Disorder/Agoraphobia (PD/A), Obsessive–Compulsive Disorder (OCD), and Social Phobia (SOC); tripartite model latent factors were Negative Affect (NA), Positive Affect (PA), and Autonomic Arousal (AA).

ADIS-IV-L. ADIS-IV-L CSRs were used as indicators for each of the five disorders evaluated: DEP (collapsed across major depression, dysthymia, and depression not otherwise specified), GAD, PD/A, OCD, and SOC. In many sections of the ADIS-IV-L, diagnosticians made dimensional ratings (0–8 scales) of the key features of the disorder in question, irrespective of whether the disorder was ultimately assigned. These ratings were available for all patients as indicators for the latent variables GAD, OCD, and SOC. In the GAD section of the ADIS-IV-L, clinicians made excessiveness ratings for seven common worry spheres (e.g., minor matters, family, finances), using a 0 (*no worry*) to 8 (*constantly worried*) scale. In the OCD section, nine obsessions (e.g., doubting, contamination, nonsensical impulses) were rated on a 0 (*never/no distress*) to 8 (*constantly/extreme distress*) persistence and distress scale. In this section, the frequency of six common compulsions (e.g., counting, checking, washing) was also rated on a 0 (*never*) to 8 (*constantly*) scale. In the SOC section, patients' fear of 13 social situations was rated using a 0 (*no fear*) to 8 (*very severe fear*) scale. For each set of ratings, average scores were used as indicators in the various models.

Depression Anxiety Stress Scales (DASS; S. H. Lovibond & P. F. Lovibond, 1995). The DASS is a 42-item instrument measuring current (over the past week) symptoms of depression, anxiety, and stress. The three psychometrically distinct scales consist of 14 items each, which are rated on a scale from 0 (*did not apply to me at all*) to 3 (*applied to me very much, or most of the time*); the range of scores for each scale is 0–42. The DASS–Depression and DASS–Anxiety scales were used as indicators for the latent variables *DSM–IV* DEP and AA, respectively. The DASS–Depression scale consists of items emphasizing dysphoria, hopelessness, self-deprecation, lack of interest and involvement,

[2] The samples used in the current study and in Zinbarg and Barlow's (1996) study did not overlap.

and so forth. The DASS–Anxiety scale consists of items assessing autonomic arousal and fearfulness. Large-sample studies of clinical and nonclinical participants have provided strong support for the psychometric properties of the DASS (e.g., T. A. Brown, Chorpita, Korotitsch, & Barlow, 1997; P. F. Lovibond & S. H. Lovibond, 1995).

Beck Anxiety and Depression Inventories (BAI, BDI; Beck & Steer, 1987, 1990). The BAI and BDI are widely used measures of current (over the past week) anxiety and depression. Both scales consist of 21 items, which are responded to on a 0–3 scale. Total scores range from 0 to 63. The BDI and BAI were used as indicators for the latent variables of *DSM–IV* DEP and AA, respectively. Guided by results of a factor analysis (T. A. Brown et al., 1996) indicating a two-factor solution (Cognitive–Affective, Nonspecific–Somatic), the BDI was scored using only the 10 items that loaded on the factor deemed to be specific to the key features of mood disorders (i.e., the Cognitive–Affective factor: Items 1–9 and 13; e.g., depressed mood, sense of hopelessness, feelings of failure). Thus, this rescoring eliminated nonspecific items of general distress and negative affect (e.g., irritability, sleeplessness, fatigability) in line with our objective to use indicators of key features of the selected *DSM–IV* constructs under study.

Albany Panic and Phobia Questionnaire (APPQ; Rapee, Craske, & Barlow, 1995). The APPQ is a 27-item measure of situational and interoceptive fear. Patients responded to items using a 0 (*no fear*) to 8 (*extreme fear*) scale, on the basis of how much fear they would expect to experience if they encountered the situation or activity during the next week. These items form three subscales: Agoraphobia (APPQ-A), composed of 9 items reflecting common agoraphobic situations (e.g., "going long distances from home alone"); Social Phobia (APPQ-S), composed of 10 items representing situations that can cause social phobia (e.g., "meeting strangers"); and Interoceptive (APPQ-I), composed of 8 items measuring fear of activities that cause physical sensations (e.g., "playing a vigorous sport"). Evidence from clinical samples supports the reliability, factor structure, and convergent and discriminant validity of the APPQ (Rapee et al., 1995). The APPQ-A and APPQ-I scales were used as indicators of the latent variable *DSM–IV* PD/A. The APPQ-S scale was used as an indicator of the latent variable *DSM–IV* SOC.

Anxiety Sensitivity Index (ASI; Peterson & Reiss, 1992). The ASI is a widely used measure of the construct of anxiety sensitivity—fear of the symptoms of anxiety. It consists of 16 items, which are responded to on a 0–4 scale, yielding a possible range of scores between 0 and 64. The ASI was used as an indicator for the latent variable *DSM–IV* PD/A.

Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990). The PSWQ is a widely used measure of the trait of worry. It consists of 16 items, which are rated on a 1–5 scale; total scores range from 16 to 90. Evidence from clinical and nonclinical samples supports the reliability, unidimensional structure, and convergent and discriminant validity of the PSWQ (T. A. Brown, Antony, & Barlow, 1992; Meyer et al., 1990). The PSWQ was used as an indicator of the latent variable *DSM–IV* GAD.

Worry Domains Questionnaire (WDQ; Tallis, Eysenck, & Mathews, 1992). The WDQ is a 25-item measure assessing the extent to which a person worries about various content areas (e.g., relationships, finances). Items are rated on a 0 (*not at all*) to 4 (*extremely*) scale. In addition to a total score, the items of the WDQ can be scored into five subscales: Relationships, Lack of Confidence, Aimless Future, Work, and Financial (5 items each). Although initial evidence, primarily from nonclinical samples, indicates that the WDQ has favorable psychometric properties (Tallis, Davey, & Bond, 1994), recent data from our laboratory (T. A. Brown et al., 1996) have indicated that some WDQ items have low convergent and discriminant validity (e.g., items from the Aimless Future scale are more strongly associated with hopelessness and DEP than with GAD worry). On the basis of these findings, only one subscale (Work) was selected from the WDQ as an indicator of the latent variable *DSM–IV* GAD.

Maudsley Obsessive–Compulsive Inventory (MOCI; Hodgson & Rachman, 1977). The MOCI is a widely used measure of obsessive–compulsive symptoms. It consists of 30 items that are responded to using a true–false scale. The MOCI provides five scores: Total Obsessional Score, Checking, Washing, Slowness–Repetition, and Doubting–Conscientiousness. However, some items contribute to more than one subscale (e.g., some items from the Slowness–Repetition scale are also used in the Checking and Washing scales). In the present study, the Checking scale (MOCI-C, 9 items) and the Doubting–Conscientiousness scale (MOCI-D, 7 items) were used as indicators of the latent variable *DSM–IV* OCD, because these scales do not possess overlapping items (item overlap would artificially enhance loading on the same factor) and because these subscales evidence favorable psychometric properties (Emmelkamp, 1988).

Social Interaction Anxiety Scales (SIAS; Mattick, Peters, & Clarke, 1989). The SIAS is a 20-item measure of social interaction anxiety (i.e., distress when initiating and maintaining conversations with friends, strangers, potential mates, etc.). Items are rated using a 0 (*not at all characteristic or true of me*) to 4 (*extremely characteristic or true of me*) scale. Total scores range from 0 to 80. Several studies have provided evidence attesting to the sound psychometric properties of the SIAS (e.g., E. J. Brown et al., 1997; Mattick et al., 1989). The SIAS was used as an indicator of the latent variable *DSM–IV* SOC.

Self-Consciousness Scale (SCS; Fenigstein, Scheier, & Buss, 1975). The SCS is a 23-item measure that yields three subscale scores: (a) Private Self-Consciousness, (b) Public Self-Consciousness, and (c) Social Anxiety. Items are rated on a 0 (*extremely uncharacteristic*) to 4 (*extremely characteristic*) scale. Although all three scales are widely used and possess favorable psychometric qualities (e.g., Fenigstein et al., 1975; Hope & Heimberg, 1988), only the 6-item Social Anxiety scale was selected for use in the present study, given its appropriateness as an indicator for the latent variable *DSM–IV* SOC.

Positive and Negative Affect Scales (PANAS; Watson, Clark, & Tellegen, 1988). The PANAS is a 20-item measure of two primary dimensions of mood: Positive Affect (PANAS-P, 10 items) and Negative Affect (PANAS-N, 10 items). Items are rated on a 1 (*very slightly or not at all*) to 5 (*extremely*) scale (total scores range from 10 to 50). The PANAS can be administered with various instructional sets reflecting different time frames (e.g., state vs. trait versions); in the present study, patients responded to PANAS items on the basis of how they felt in general. The PANAS-P and PANAS-N were used as indicators for the latent variables PA and NA, respectively.

Approach to Structural Modeling

The sample variance–covariance matrix of the aforementioned indicators was analyzed using a linear structural relations program and a maximum-likelihood solution (LISREL 8.12a; Jöreskog & Sörbom, 1993). In models involving more than one latent *X* variable (e.g., the five-factor model of *DSM–IV* disorders), the *X* factors were permitted to be intercorrelated. Goodness-of-fit was evaluated using the following: the comparative-fit index (CFI; Bentler, 1990), the incremental-fit index (IFI; Bollen, 1989), the root mean square error of approximation (RMSEA; Steiger, 1990), and the goodness-of-fit index (GFI; Bentler & Bonett, 1980). Multiple indices were selected because they provide different information for evaluating model fit (i.e., absolute fit, fit adjusting for model parsimony, fit relative to a null model); used together, these indices provide a more conservative and reliable evaluation of the various models (cf. Jaccard & Wan, 1996).

When competing models were nested (e.g., various measurement models of the *DSM–IV* anxiety and mood disorders), comparative fit was evaluated using nested chi-square tests. When competing models were not nested, comparative fit could not be evaluated using significance testing.[3] Therefore, the following criteria were used in the identification of best model fit: (a) overall fit (e.g., CFI, IFI); (b) the lowest chi-square with the most model parsimony (i.e., fewest number of paths), as quantified by Akaike's information criterion (AIC; Akaike, 1987); and (c) interpretability and strength of the various parameter estimates.

Results

Measurement Models for the DSM–IV Disorder Factors

Confirmatory factor analysis was performed to test the hypothesis that a five-factor model (corresponding to the *DSM–IV* disorders DEP, GAD, PD/A, OCD, and SOC) would provide an acceptable fit for the data, in comparison to a unifactorial model representing a general anxious–depressive syndrome and a two-factor model corresponding to depression and anxiety disorders (i.e., GAD, PD/A, OCD, and SOC collapsed under a single factor). Each *DSM–IV* latent variable was associated with three or more indicators, with at least one clinician rating and one questionnaire indicator (see Table 1). In all models, the theta-delta matrix was programmed to estimate correlated error among the ADIS-IV-L ratings within a given *DSM–IV* disorder (e.g., correlated error between ADIS-IV-L CSR-GAD and ADIS-IV-L Worry) on the basis of the expectation that individual symptom ratings for a disorder would influence the CSR that was ultimately assigned to it.

First, the five-factor model was fitted to the data. Fit indices indicated that this model provided an acceptable fit to the data, $\chi^2(174), N = 350) = 475.18$, CFI = .92, IFI = .92, RMSEA = .070, GFI = .89 ($N = 350$ for all chi-square analyses reported). However, inspection of modification indices and standardized residuals suggested that model fit could be improved if correlated error was estimated between the MOCI-C and MOCI-D scales, and among the three APPQ scales (highest modification index = 24.88). Thus, the five-factor model was refitted to the data with these adjustments (specifying correlated method variance among subscales). Fit indices for the revised model indicated improved fit, $\chi^2(170) = 401.20$, CFI = .94, IFI = .94, RMSEA = .062, GFI = .90. This was confirmed by a statistically significant decrease in the chi-square value, $\chi^2_{\text{diff}}(4) = 73.98$, $p < .001$. Factor loadings (completely standardized estimates from the lambda-X matrix) are presented in Table 1. All of these loadings, as well as the specified correlated errors (which ranged from .11 to .51), were statistically significant.

Next, the one-factor model was fitted to the data; this model included the same error theory (correlated error among certain indicators) used in the revised five-factor model. This model fit the data poorly, $\chi^2(180) = 1{,}623.65$; a nested chi-square test indicated that this model degraded fit significantly, $\chi^2_{\text{diff}}(10) = 1{,}222.45$, $p < .001$. Whereas the two-factor model improved fit relative to the one-factor model, $\chi^2_{\text{diff}}(1) = 478.87$, $p < .001$, it too fit poorly and was inferior to the revised five-factor model, $\chi^2_{\text{diff}}(9) = 743.58$, $p < .001$.

Table 2 presents the intercorrelations among the *DSM–IV* disorder factors from the revised five-factor model (i.e., completely standardized coefficients from the phi matrix). As can be seen in Table 2, the highest correlation between *DSM–IV* factors was for DEP and GAD (.63), consistent with previous evidence that generalized anxiety disorder may overlap more with the mood disorders than with its fellow anxiety disorders (cf. T. A. Brown et al., 1996; T. A. Brown, Marten, & Barlow, 1995). This finding raised the possibility that model fit would not be degraded (or would perhaps be improved) if DEP and GAD were collapsed into a single factor. Accordingly, a four-factor model (DEP–GAD, PD/A, OCD, and SOC) was fitted to the data. A nested chi-square test indicated that this four-

Table 1
Factor Loadings (Completely Standardized Estimates) for the DSM–IV Disorder Measurement Model

Latent factor and measure	Factor loading
DSM–IV Depression	
DASS–Depression	.86
Beck Depression Inventory (Items 1–9, 13)	.92
ADIS-IV-L CRS–Mood	.71
DSM–IV Generalized Anxiety Disorder	
Penn State Worry Questionnaire	.88
Worry Domains Questionnaire–Work	.61
ADIS-IV-L Worry	.59
ADIS-IV-L CSR-GAD	.41
DSM–IV Panic Disorder/Agoraphobia	
Anxiety Sensitivity Index	.86
APPQ–Interoceptive	.62
APPQ–Agoraphobia	.57
ADIS-IV-L CSR-PD/A	.61
DSM–IV Obsessive–Compulsive Disorder	
MOCI–Doubting	.87
MOCI–Checking	.94
ADIS-IV-L CSR-OCD	.41
ADIS-IV-L Obsessions	.45
ADIS-IV-L Compulsions	.46
DSM–IV Social Phobia	
Social Interaction Anxiety Scale	.91
APPQ–Social	.85
Self-Consciousness Scale–Social Anxiety	.83
ADIS-IV-L CSR-SOC	.64
ADIS-IV-L Social Fear	.79

Note. *DSM–IV* = *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.); DASS = Depression Anxiety Stress Scales; ADIS-IV-L = Anxiety Disorders Interview Schedule for *DSM–IV:* Lifetime version; CSR = clinical severity rating; CSR–Mood = ADIS-IV-L CSR of mood disorders (major depression, dysthymia, and depression not otherwise specified); ADIS-IV-L Worry = average ADIS-IV-L rating of seven worry spheres; CSR-GAD = ADIS-IV-L CSR of generalized anxiety disorder; APPQ = Albany Panic and Phobia Questionnaire; CSR-PD/A = ADIS-IV-L CSR of panic disorder/agoraphobia; MOCI = Maudsley Obsessive–Compulsive Inventory; CSR-OCD = ADIS-IV-L CSR of obsessive–compulsive disorder; ADIS-IV-L Obsessions = average ADIS-IV-L rating of nine obsessions; ADIS-IV-L Compulsions = average ADIS-IV-L rating of six compulsions; CSR-SOC = ADIS-IV-L CSR of social phobia; ADIS-IV-L Social Fear = average ADIS-IV-L rating of fear of 13 social situations.

[3] The chi-square difference test is inapplicable in situations where the structures of competing models vary substantially (e.g., in models where latent factors vary between being X and Y variables) and models are not nested (i.e., a nested model is one that has a subset of the free parameters of a parent model).

Table 2
Zero-Order Intercorrelations Among the Five DSM–IV Disorder Latent Factors

Latent factor	DEP	GAD	PD/A	OCD	SOC
DEP	—				
GAD	.63	—			
PD/A	.44	.50	—		
OCD	.43	.52	.29	—	
SOC	.39	.37	.22	.30	—

Note. Correlations are based on the results of the revised five-factor measurement model. *DSM–IV* = *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.); DEP = depression (*DSM–IV* mood disorders); GAD = generalized anxiety disorder; PD/A = panic disorder with or without agoraphobia; OCD = obsessive–compulsive disorder; SOC = social phobia.

factor model produced a significant degradation in model fit, relative to the five-factor model, $\chi^2_{diff}(4) = 152.83$, $p < .001$.[4]

Measurement Models for the Tripartite Model Factors

Prior to examining the various structural models involving the *DSM–IV* disorder and tripartite model factors, the structure of the tripartite factors was first evaluated with confirmatory factor analysis. Because the latent variables NA and PA were assessed with single indicators, the PANAS-N and PANAS-P were randomly split-halved for inclusion in these analyses (because of the need for at least two indicators per latent variable to prevent model underidentification). As noted earlier, the BAI and DASS-A were used as indicators for the latent variable AA. Three models were evaluated: (a) the predicted three-factor model (NA, PA, and AA), (b) a two-factor model (NA–PA and AA, given the possibility that the PANAS-N and PANAS-P would cluster together because they were derived from the same instrument), and (c) a one-factor model. The one- and two-factor solutions were poor fitting, $\chi^2(9) = 434.48$ and $\chi^2(8) = 317.69$, respectively. Conversely, the hypothesized three-factor model provided an excellent fit for the data, $\chi^2(6) = 3.83$, CFI = 1.00, IFI = 1.00, RMSEA = 0, GFI = 1.00. Each of the six indicators had strong and statistically significant loadings (*p*s < .001) on their respective latent factors: NA = .90, .86; PA = .98, .81; AA = .91 (BAI), .86 (DASS-A). The intercorrelations among these factors (completely standardized estimates from the phi matrix) were −.35 (NA–PA), −.30 (PA–AA), and .77 (NA–AA; all *p*s < .001). To rule out the possibility that the strong correlation between the NA and AA factors indicated that these latent variables could be collapsed, a fourth model was fit to the data. This two-factor model (i.e., NA–AA and PA) resulted in a degradation in fit, thereby providing further support for the tripartite measurement model, $\chi^2_{diff}(3) = 117.51$, $p < .001$.

Structural Models Using the DSM–IV Disorder and Tripartite Factors

As noted earlier, of the various models involving structural relationships among the tripartite and *DSM–IV* disorder factors, it was predicted that the best fitting model would entail (a) significant paths from a higher order factor, NA, to each of the five *DSM–IV* disorder factors (and paths from NA to AA); (b) a significant path from a higher order factor, PA, to DEP; and (c) significant paths from GAD, PD/A, OCD, and SOC to AA ("hypothesized model"; see Figure 1). This model was viewed to be consistent with the following: (a) The general distress symptoms shared by the various anxiety and mood disorders are best conceptualized as trait NA, a chronic feature that represents a vulnerability dimension for the development of emotional disorders; (b) the influence of trait PA is specific to DEP; and (c) the anxiety disorders, but not DEP, influence AA (i.e., the absence of a path from DEP to AA would not strain model fit because AA is specific to anxiety disorders). Although the hypothesized model contained a path from GAD to AA, it was uncertain whether this path would be significant given evidence that GAD and worry may be associated with autonomic suppression (e.g., Borkovec, Lyonfields, Wiser, & Diehl, 1993; Hoehn-Saric, McLeod, & Zimmerli, 1989); however, other data indicate that patients with GAD endorse autonomic symptoms frequently; e.g., T. A. Brown, Marten, & Barlow, 1995).

The hypothesized model was compared with two competing models (see Figure 1): (a) In Competing Model 1, the five *DSM–IV* disorder factors were specified as higher order factors, and the tripartite factors were specified as first-order factors (i.e., NA and PA, as well as AA, were influenced by the *DSM–IV* disorders), suggesting that although the tripartite model constructs characterize the shared (NA) and unique (PA and AA) symptoms of anxiety and depression, none represent trait dimensions that influence these disorders; and (b) in Competing Model 2, NA, PA, and AA were each specified as higher order factors, and the *DSM–IV* disorders were specified as first-order factors, signifying that in addition to NA and PA, AA was a traitlike variable that could predict variability in the *DSM–IV* disorder factors (this model was included because of empirical and conceptual uncertainty about whether, along with NA and PA, AA represents a trait vulnerability dimension of emotional disorders; L. A. Clark, Watson, & Mineka, 1994).

In both competing models, the patterns of paths between the tripartite and *DSM–IV* factors were the same (e.g., the *DSM–IV* anxiety disorder factors each had paths to AA, or vice versa). In all three models, the latent factors NA and PA were specified with single indicators (the full-scale PANAS-N and PANAS-P, respectively); however, measurement error was modeled in these factors by constraining the theta-delta and theta-epsilon matrices to predetermined values (i.e., estimates of error variance were calculated using internal consistency estimates of the PANAS-

[4] Given the possibility that inclusion of ADIS-IV-L diagnosis severity ratings biased the results in favor of the five-factor model (because ADIS-IV-L CSRs reflect dimensional severity ratings based on the *DSM–IV* classification scheme), confirmatory factor analyses of the *DSM–IV* measurement models were reconducted excluding ADIS-IV-L CSRs. Once again, the five-factor model provided the best fit for the data, $\chi^2(89) = 183.83$, CFI = .97, IFI = .97, RMSEA = .055, GFI = .94, relative to the one-factor model, $\chi^2(99) = 1{,}085.90$, the two-factor model, $\chi^2(98) = 811.03$, and the four-factor model, $\chi^2(93) = 328.27$; for example, a five- versus a four-factor model: $\chi^2_{diff}(4) = 144.44$, $p < .001$.

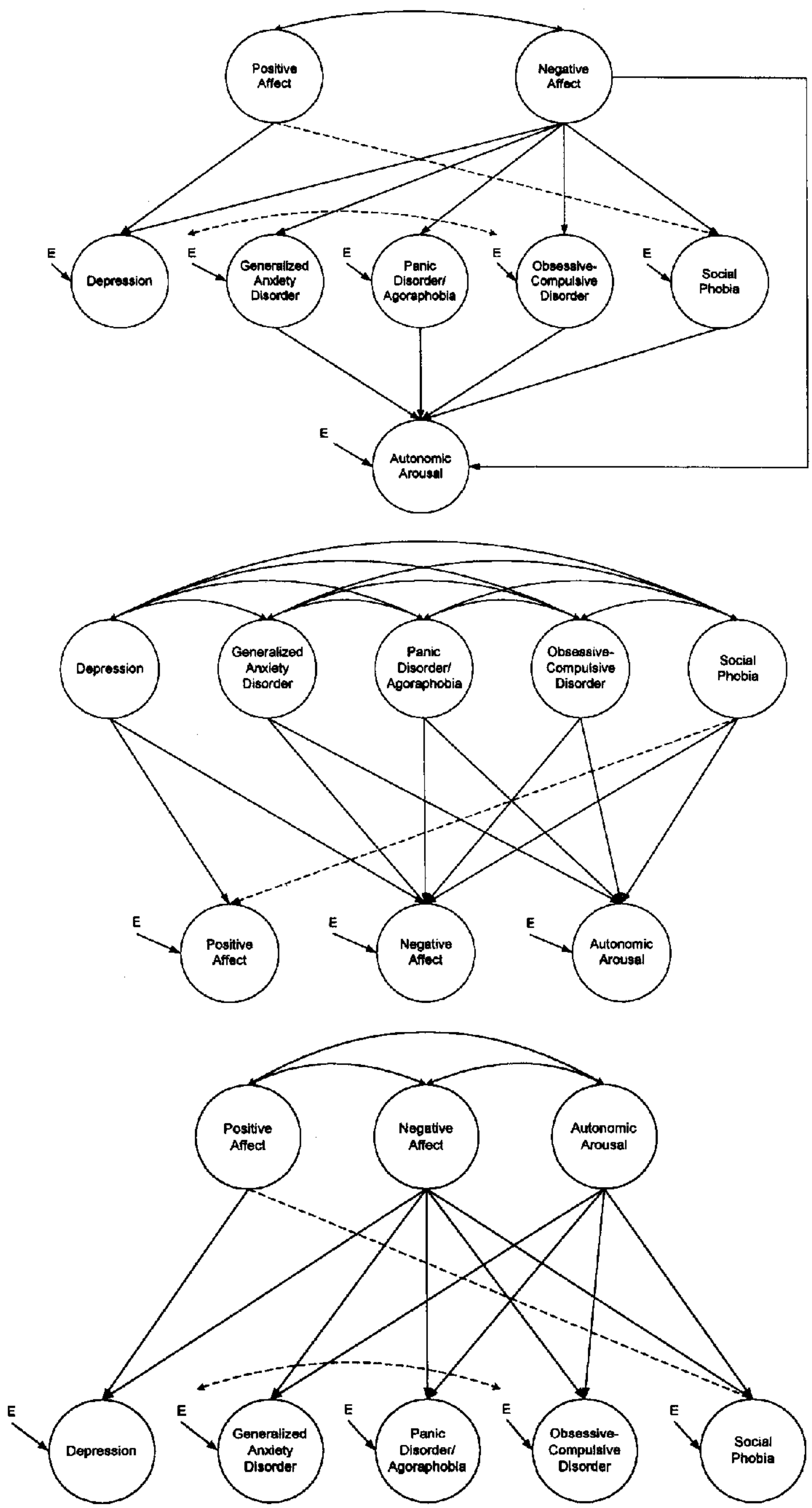

Figure 1. Structural models of *DSM–IV* disorder and tripartite model factors: hypothesized model (top), Competing Model 1 (middle), Competing Model 2 (bottom). Dotted lines indicate paths that were added after fit diagnostics of the initial model. *DSM–IV* = *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.); E = residual.

Table 3
Zero-Order Intercorrelations Among DSM–IV Disorder and Tripartite Model Latent Factors

Tripartite model factor	*DSM–IV* disorder factor				
	DEP	GAD	PD/A	OCD	SOC
NA	$.77_a$	$.74_a$	$.65_b$	$.43_c$	$.41_c$
PA	$-.53_a$	$-.27_c$	$-.23_{c,d}$	$-.16_d$	$-.39_b$
AA	$.60_b$	$.48_c$	$.89_a$	$.31_d$	$.31_d$

Note. Correlations were derived from the revised hypothesized structural model. Correlations sharing the same subscript letter do not differ in their relative magnitude as determined by the z test procedure (α = .05) presented by Meng, Rosenthal, and Rubin (1992); for example, although significantly ($p < .05$) different from PD/A, OCD, and SOC, the DEP and GAD factors do not differ in their strength of associations with NA. *DSM–IV* = *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.); DEP = depression (*DSM–IV* mood disorders); GAD = generalized anxiety disorder; PD/A = panic disorder with or without agoraphobia; OCD = obsessive–compulsive disorder; SOC = social phobia; NA = negative affect; PA = positive affect; AA = autonomic arousal.

N and PANAS-P in the present sample).[5] Moreover, each of the structural models was evaluated with the error theory used in the *DSM–IV* measurement models (e.g., correlated error among the APPQ scales and between the MOCI-C and MOCI-D), and the theta-delta-epsilon matrix was programmed to estimate correlated error between the DASS–Anxiety and DASS–Depression scales (i.e., nonrandom measurement error attributable to subscales from the same measure).

Hypothesized model. Indices of overall fit indicated that the hypothesized model provided a good fit for the data, $\chi^2(255)$ = 579.36, CFI = .94, IFI = .94, RMSEA = .060, GFI = .89. Modification indices pertaining to the measurement aspect of the model indicated no points of ill fit due to the addition of the tripartite indicators to the five *DSM–IV* disorder factor model (e.g., despite a zero-order factor correlation between PD/A and AA of .89, see Table 3; lambda-*Y* modification indices for both AA indicators and the PD/A factor were 0.14). However, inspection of modification indices for various aspects of the structural model revealed two points of strain. First, these indices suggested that fit could be improved if a path were freed between GAD and OCD (modification index = 19.31 in both the beta and psi matrices). Second, a high modification index (21.53) was observed in the gamma matrix for the PA and SOC factors. Results suggesting an association between GAD and OCD were interpreted as consistent with earlier findings indicating that these syndromes may be neighboring disorders in the context of other anxiety disorders (e.g., because of the potential similarities or overlap in chronic worry and certain types of obsessions; cf. T. A. Brown, Moras, Zinbarg, & Barlow, 1993; Turner, Beidel, & Stanley, 1992). Although counter to our initial predictions, further review of the literature indicated that the second strain in model fit was in fact consistent with prior evidence that social phobia is associated with low PA (Amies, Gelder, & Shaw, 1983; Watson, Clark, & Carey, 1988). Thus, the hypothesized model was refitted to the data with the additions of a path estimating correlated residuals between the GAD and OCD factors and a path from PA to SOC.

The revised hypothesized model is presented in Figure 2. The zero-order factor correlation matrix for the tripartite and *DSM–IV* disorder factors is presented in Table 3, along with the results of tests of the differential magnitude of the associations between the *DSM–IV* disorder factors and the tripartite factors. This model provided a significantly improved fit for the data, $\chi^2_{\text{diff}}(2) = 43.76$, $p < .001$. Overall fit indices were $\chi^2(253)$ = 535.60, CFI = .95, IFI = .95, RMSEA = .057, GFI = .89. Inspection of modification indices and standardized residuals indicated no strains in the structural model. As shown in Figure 2, completely standardized paths from NA to the various *DSM–IV* disorder factors were of the expected relative magnitude (e.g., the highest paths from NA were to GAD and DEP), and all were statistically significant ($ps < .001$). As expected, a statistically significant ($p < .001$) path was observed from PA to DEP (completely standardized estimate = −.29). A statistically significant path ($p < .001$) was also observed from PA to SOC (−.28). Consistent with the tripartite model, modification indices from the gamma matrix involving potential paths from PA to GAD, PD/A, and OCD were uniformly low (range = 0 to 0.47), which is indicative of a lack of relationship between PA and these disorders.

As suggested by the initial model, the residuals between the GAD and OCD factors were correlated significantly (.18, $p < .001$). Strong and statistically significant paths from PD/A and NA to AA were obtained (path coefficients = .67 and .50, respectively; $ps < .001$). It is interesting that a statistically significant ($p < .001$) path from GAD to AA was also observed; however, this path was negative (−.22), indicating that an increase in GAD was associated with a decrease in AA, despite the fact that the zero-order factor correlation between GAD and AA was positive (.48, see Table 3). Thus, a suppressor effect was operative in this aspect of the model: Specifically, the strong association between the predictors NA and GAD (zero-order factor correlation = .74) masked the true association between GAD and AA (cf. Cohen & Cohen, 1983). In other words, when variance from NA was removed, the true relationship between GAD and AA was elucidated (in this instance, the direction of the association was reversed).[6]

As shown in Figure 2, nonsignificant paths to AA were obtained for both OCD and SOC, a finding that was counter to expectation. However, consistent with our prediction and the tripartite model, results indicated that fit would not be improved

[5] Internal consistencies (Cronbach's alphas) for the PANAS-N and PANAS-P in the present sample were .88 and .90, respectively. The single indicator approach was selected over a split-half approach because measurement error could be modeled more accurately (i.e., Cronbach's alpha represents an average of all possible split-halves).

[6] To examine the possibility that we concluded erroneously that NA was chiefly responsible for the suppressor effect observed in the relationship between GAD and AA, a simplified model was evaluated with LISREL: NA was specified as a predictor with paths to the GAD and AA factors, and a path was specified from GAD to AA. This model provided an excellent fit to the data, $\chi^2(11) = 31.25$, CFI = .98, IFI = .98, RMSEA = .073, GFI = .98, and the same suppressor effect was obtained (the completely standardized path from GAD to AA was −.15).

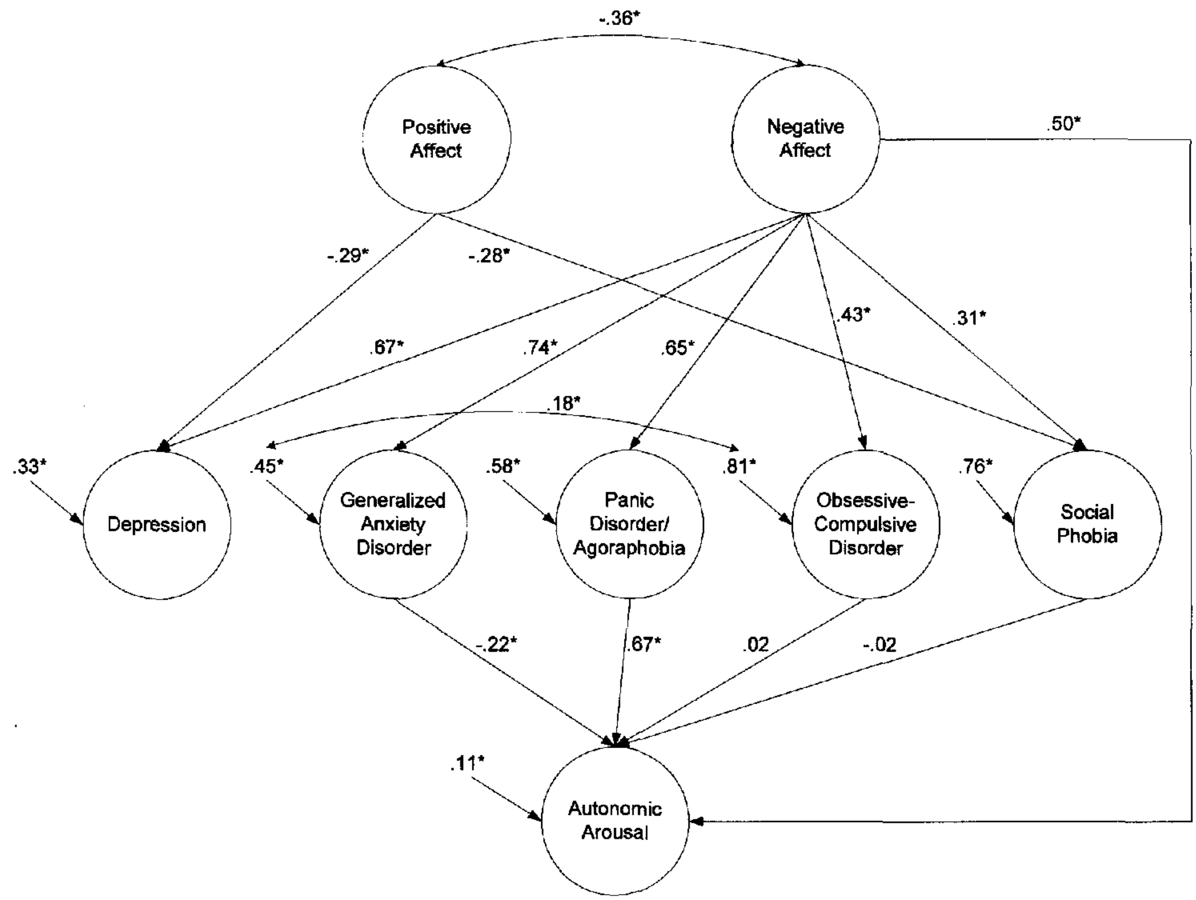

Figure 2. Completely standardized solution of hypothesized structural model—revised. $*p < .01$.

if a path was added from DEP to AA (beta modification index = 2.46; standardized expected change = 0).

Competing models. The two competing models were fitted to the data (incorporating the structural modifications suggested by the initial hypothesized model; e.g., the path linking PA and SOC) to determine whether the revised hypothesized model indeed best accounted for the interrelationships among the tripartite and *DSM–IV* disorder factors. Competing Model 1 (in which the *DSM–IV* disorder factors were predictors) generally fit the data well, $\chi^2(246) = 561.10$, CFI = .94, IFI = .94, RMSEA = .061, GFI = .89. However, although significance testing could not be used (i.e., the various models were not nested; see the Method section), it is interesting to note that relative to the hypothesized model, this model produced a higher chi-square value (561.10 vs. 535.60), despite having fewer degrees of freedom (246 vs. 253); AIC = 719.10 and 679.60 for Competing Model 1 and the hypothesized model, respectively. The reduction in degrees of freedom in Competing Model 1 was due to the specification of the *DSM–IV* disorder factors as predictors; thus, all correlations among these disorders (see Table 2) were estimated in the phi matrix. Therefore, it is surprising that Competing Model 1 produced an increase in chi square given that the intercorrelations among the *DSM–IV* disorder factors were fully modeled, especially in context of the more stringent hypothesized model, in which the intercorrelation among *DSM–IV* disorder factors could be accounted for only by their associations with NA and PA (and the correlated residuals of GAD and OCD). On the basis of these considerations, in tandem with the observation of nonsignificant paths between some *DSM–IV* disorder factors and NA (e.g., −.02 for OCD, .09 for SOC), Competing Model 1 was rejected because it was not equivalent or superior to the hypothesized model.

A similar result was obtained for Competing Model 2 (which specified all three tripartite dimensions as higher order factors to the *DSM–IV* disorder factors). Whereas this model generally fit the data well, $\chi^2(252) = 542.84$, CFI = .94, IFI = .94, RMSEA = .058, GFI = .89, compared with the hypothesized model it too produced a higher chi-square value (542.84 vs. 535.60) with fewer degrees of freedom (252 vs. 253); AIC = 688.84 and 679.60 for Competing Model 2 and the hypothesized model, respectively. Although associated with fewer counterintuitive path estimates than Competing Model 1 (though several poor estimates did exist; e.g., NA to PD/A = −.09), this model was also rejected because of its inferior interpretability and parsimony (i.e., higher chi-square and AIC values), relative to the hypothesized model.

Discussion

For the five *DSM–IV* anxiety and mood disorder constructs examined, confirmatory factor analysis of a variety of dimensional symptom measures provided empirical support for the discriminant validity of these disorders. While upholding the exploratory factor structure reported by Zinbarg and Barlow (1996), the present findings extend these prior results with the inclusion of model indicators assessed by multiple methods (questionnaires, clinician ratings) and the inclusion of a *DSM–IV* mood-disorder latent factor. As expected from these analyses, the highest factor correlation observed was between GAD and DEP (.63), supporting previous contentions that relative to the other anxiety disorders, the features of generalized anxiety disorder have the most overlap with the mood disorders (in fact, the correlation between GAD and DEP was higher than the correlations between GAD and PD/A, between GAD and OCD, and between GAD and SOC). It is also interesting to note that the relative magnitudes of other zero-order factor correlations generated from the five-factor solution (see Table 2) were in accord with previously observed or hypothesized points of overlap among these disorders (e.g., the OCD factor had its strongest correlation with GAD; cf. T. A. Brown et al., 1993). Nevertheless, in addition to its superiority to the one- and two-factor models, the five-factor model was significantly better fitting than a four-factor model (in which GAD and the mood disorders were collapsed as a single latent factor), thereby providing support for the distinction of these domains.

Although the five-factor *DSM–IV* disorder model fit the data well and was superior to competing models, these analyses could be viewed as a somewhat liberal test of the *DSM* structure given that most indicators assessed key features of the disorders (e.g., chronic worry, depressive affect) rather than potentially less distinguishable associated symptoms (e.g., the somatic symptoms of GAD and the mood disorders were not modeled). This issue seems less germane to three of the five disorders analyzed (PD/A, OCD, and SOC) because their *DSM–IV* definitions are based on key features and do not include associated symptom criteria. Yet it is likely that inclusion of associated symptoms for GAD and DEP would have increased the correlation between these latent factors (and would have degraded model fit unless double-loading indicators were specified), given the definitional overlap in many of these features (e.g., sleep disturbance, fatigue, restlessness). This potential problem in differential diagnosis is handled in two ways by *DSM–IV:* (a) a hierarchical rule specifying that GAD should not be assigned if its features occur exclusively during a mood disorder, and (b) to count toward GAD or a mood disorder, the associated symptoms must accompany the key features of the disorder (e.g., in GAD, worry is associated with sleep disturbance, restlessness, etc.).

Whereas the distinguishability of generalized anxiety disorder from other anxiety and mood disorders was supported by the superiority of the five-factor measurement model, these data also indicated that of the *DSM–IV* constructs examined, GAD evidenced the highest degree of overlap with the other *DSM–IV* disorder factors (i.e., the latent factor *DSM–IV* GAD consistently had the strongest zero-order correlations with other *DSM–IV* disorder factors; see Table 2). Moreover, at the zero-order level and in the best fitting structural model (see Figure 2), *DSM–IV* GAD evidenced a strong association with the nonspecific dimension of NA (.74). Although suggesting that the construct of *DSM–IV* GAD had poorer discriminant validity relative to other disorders, these collective findings could be viewed as consistent with conceptualizations of generalized anxiety disorder as the basic emotional disorder because it is composed of features (chronic worry, negative affect) that are present to varying degrees in all emotional disorders and that reflect key vulnerability dimensions of these syndromes (which also may account for the high comorbidity rates associated with this disorder; cf. T. A. Brown et al., 1994). In light of these findings and conceptual arguments, an important direction for future inquiry is the examination of the structural longitudinal relationships among dimensions of generalized anxiety disorder and dimensions of trait vulnerability (e.g., negative affect, neuroticism). This research would evaluate whether the features of generalized anxiety disorder are best subsumed under these personality dimensions (consistent with the view that *DSM–IV* generalized anxiety disorder reflects a trait of nonspecific vulnerability rather than an Axis I disorder) or whether generalized anxiety disorder represents a distinct *DSM–IV* Axis I type construct that along with (but to a greater extent than) other anxiety and mood disorders, is influenced by these higher order dimensions (T. A. Brown, in press).

Similarly, whereas the present findings support the distinguishability of the five *DSM–IV* constructs examined, these results should not be interpreted as bearing directly on the validity of the *DSM–IV* organizational scheme (i.e., the grouping of PD/A, GAD, OCD, etc. under the broad category of *anxiety disorders;* the grouping of major depression, dysthymia, etc. under the broad heading of *mood disorders*). In future research, a more comprehensive evaluation of the *DSM–IV* nosology would use confirmatory factor analyses of data in which all constituent criteria of the anxiety and mood disorders were dimensionalized, perhaps by specifying multiple-factor loadings for indicators of shared associated symptoms and single-factor loadings for key features. Moreover, the current results could be extended with the inclusion of indicators for additional disorders (e.g., posttraumatic stress disorder, specific phobia) and the use of hierarchical factor analysis to examine if the *DSM–IV* anxiety and mood disorder constructs load onto higher order factors in a manner consistent with the *DSM–IV* organizational scheme.

Despite being the most stringent of the three structural models involving the relationships of the *DSM–IV* disorder and tripartite dimensions, the hypothesized model produced the lowest chi-square and AIC values with the greatest degree of parsimony (i.e., the smallest number of paths). It could be argued that because chi-square and AIC values did not vary substantially in the models tested (e.g., χ^2 range = 535.60 to 561.10), fit was generally equivalent across models. However, substantial fluctuations in overall fit indices are not expected in instances in which small alterations are evaluated in the context of a large model with a good fit to the data. Such was the case in the present study in which minor, yet theoretically important, structural variations (in general, the *direction* but not the *existence* of paths varied across models) were evaluated within a good-fitting measurement model. As noted earlier, this underscores the importance of considering other parameters of fit besides indices of overall fit (e.g., model parsimony, interpretability of path coefficients). Even when these parameters were considered, the hypothesized model was regarded as superior to the competing models (e.g., it produced the lowest chi-square and AIC values even though the correlations among *DSM–IV* disorder factors could be accounted for in the model only by the paths from NA and PA). Nevertheless, the lack of marked differences in fit across models may have also been due, in part, to reciprocal relationships among some factors (e.g., NA may influence the *DSM–IV* disorders, which in turn influence NA; cf. L. A. Clark, Watson, & Mineka, 1994). This possibility could not be addressed in the present cross-sectional study because specification of reciprocal paths would have led to model underidentification. Evaluation of this speculation, as well as a more definitive analysis of negative and positive affect as dispositional vulnerability dimensions of the emotional disorders, awaits longitudinal investigation.

Nevertheless, the current analyses produced several interesting findings regarding the relationships among the tripartite model dimensions and the *DSM–IV* anxiety and mood disorders. It is noteworthy that the factor correlation between NA and PA (−.36) was quite consistent with prior findings (e.g., D. A. Clark, Steer, & Beck, 1994; Watson et al., 1988), indicating that the relationship between these dimensions is relatively stable across samples, instruments, and data analytic strategies. Consistent with the tripartite model in which negative affect is viewed as a factor common to anxiety and depression, all paths from NA to the *DSM–IV* disorder factors were statistically significant. The relative magnitudes of these paths were also concordant with prediction. For instance, the largest paths from NA (and zero-order factor correlations) were to GAD and DEP, factors that correspond to *DSM–IV* disorders that are often considered to have the strongest associations with negative affect. In addition, the smaller factor correlations and path coefficients observed between NA and certain *DSM–IV* factors (e.g., SOC) align with earlier evidence that the nature and strength of the relationship of negative affect may vary across the different anxiety disorders (L. A. Clark, Watson, & Mineka, 1994).

The notion that the tripartite factors may have differential relevance or relationships to the anxiety disorders was also observed in paths and correlations involving the latent variable of AA. Findings indicating degradations in model fit and interpretability when AA was specified as a higher order factor could be taken in support of prior conclusions that of the three tripartite factors, autonomic arousal is the least related to dimensions of personality (L. A. Clark, Watson, & Mineka, 1994). Consistent with current conceptualizations that AA is of central importance to panic disorder (Barlow et al., 1996; L. A. Clark, Watson, & Mineka, 1994), the strongest paths to and factor correlations with AA were found for the latent factor PD/A. On the other hand, paths from the *DSM* disorder factors of OCD and SOC to AA were nonsignificant. In addition, these factors were weakly correlated with AA at the zero-order level (all rs = .31). These results suggest that, though generally unrelated to mood disorders (i.e., results indicated no improvement in model fit with the addition of a path from DEP to AA), autonomic arousal symptoms may be weakly related or of less discriminant value for certain anxiety disorders (e.g., discrete social phobias). Although not addressed in the present study, this would also seem to be the case for specific phobia given evidence that persons with this disorder often score within the normal range on measures of autonomic arousal or fear and general distress (T. A. Brown, Chorpita, et al., 1997).

Accordingly, the current findings highlight a possible refinement of the tripartite model with regard to autonomic arousal. Although autonomic arousal had been initially posited to be a discriminating feature for the entire range of anxiety disorders, these data suggest that the relevance of autonomic arousal may be limited primarily to panic disorder/agoraphobia. Although this interpretation is generally in accord with recent reconsiderations of the tripartite model (L. A. Clark, Watson, & Mineka, 1994), additional research is needed to examine the replicability of these results in other samples and with other indicators of AA (e.g., to rule out the possibility that the high representation of panic disorder in the sample and use of the BAI as an indicator of AA—cf. Cox, Cohen, Direnfeld, & Swinson, 1996; Steer & Beck, 1996—may have augmented the differential association between PD/A and AA in the present study).

Nowhere was the potential differential relationship of the anxiety disorders with autonomic arousal more evident than in the path from the GAD factor to AA. Indeed, the suppressor effect observed in the hypothesized structural model (path = −.22, despite a zero-order correlation of .48) may help to account for the conflicting findings regarding the association between generalized anxiety disorder and symptoms of autonomic arousal. As discussed earlier, although patients with generalized anxiety disorder endorse these symptoms frequently (T. A. Brown, Marten, & Barlow, 1995), recent data indicate that these patients may respond to psychological stress with autonomic inflexibility (Borkovec et al., 1993; Hoehn-Saric et al., 1989). Our findings suggest that autonomic symptoms in this disorder may be due to high levels of negative affect (i.e., in addition to GAD being associated with the highest levels of NA—cf. T. A. Brown et al., 1996, T. A. Brown, Chorpita, et al., 1997—both GAD and AA are strongly correlated with NA). However, after accounting for variance in AA due to NA, the true direct influence of GAD on AA may have been illuminated in these analyses; namely, the disorder-specific features of GAD (i.e., worry; most of the GAD indicators were measures of worry) act to decrease (suppress) autonomic arousal.[7] If interpreted in this manner, this attests to the robustness of the association between worry and autonomic suppression given the disparate methodologies that have produced this result (i.e., structural equation modeling vs. laboratory challenges).

As was true for the higher order latent factor NA, results involving PA were generally consistent with our predictions. Comparisons of the various models indicated that model fit and interpretability of the resulting path estimates were optimal when PA was specified as a higher order factor to the *DSM–IV* disorder factors. Moreover, support for the tripartite model was obtained by findings of a significant negative path from PA to

[7] Similarly, the high zero-order factor correlation between DEP and AA (.60) could have been due to the strong relationships between DEP and NA (.77) and AA and NA (.77). This conclusion was supported by small modification indices from the structural model indicating that adding a path from DEP to AA would not improve model fit (i.e., the path would be nonsignificant) because DEP could not account for additional variance in AA.

DEP and low modification indices, indicating that model fit would not improve (i.e., the paths would be nonsignificant) if paths were added from PA to GAD, PD/A, or OCD. Such findings support the contention that low positive affect is more specifically linked to depression. However, counter to our expectations, inspection of fit diagnostics of the initial hypothesized model revealed that fit would improve with the specification of a path from PA to SOC. In addition, SOC had a significantly stronger zero-order association with PA than did the other anxiety disorder factors (see Table 3). In fact, unbeknownst to us when the initial hypothesized model was constructed, previous research has found an association between social phobia and lower levels of PA (Amies et al., 1983; Watson, Clark, & Carey, 1988). The unique relationship of social phobia and PA relative to the other anxiety disorders has been interpreted as being based on the interpersonal character of low PA (e.g., low confidence, unassertiveness; L. A. Clark, Watson, & Mineka, 1994). The addition of this path in the revised model was statistically significant and in the direction (i.e., negative) consistent with prior findings. However, somewhat counter to the tripartite model, which asserts that PA is more strongly linked to depression than to the anxiety disorders, this path (−.28) was virtually identical to the path from PA to DEP (−.29), suggesting no differential influence of PA on DEP and SOC after controlling for variance in NA (although DEP was more strongly correlated with PA than SOC at the zero-order level; see Table 3). These results underscore the importance of examining the influence of these dimensions at the multivariate level given the complexity of their posited interrelationships (e.g., hierarchical structure, differential strength of influence) and potential overlap in their underlying domains (e.g., zero-order relationships of the tripartite dimensions to emotional disorder symptoms and syndromes are affected to varying degrees by shared variance in NA, PA, and AA).

Results of multiple regression analyses reported in Watson, Clark, and Carey (1988), in which PA and NA were entered as predictors of major depression, dysthymia, and social phobia, did reveal greater differential magnitudes of the paths (standardized regression coefficients) of PA and depression (−.26) and PA and social phobia (−.19). However, inconsistent with predictions of the tripartite model and the results of the present study, Watson et al. found that NA did not add to the prediction of social phobia after controlling for variance in PA. It is also interesting to note that, although of similar relative magnitude, the zero-order factor correlations and path coefficients among the tripartite and *DSM–IV* disorder factors in the present study were generally stronger than the zero-order correlations and regression coefficients obtained by Watson et al. Possible reasons for these differences include (a) the use of a categorical versus a dimensional approach to the assessment of *DSM–IV* disorders (diagnoses were scored dichotomously as absent or present in Watson et al.), and (b) control versus no control of measurement error (because standard correlational and regression analyses were used in Watson et al., the resulting correlations and path coefficients were not adjusted for measurement error). Thus, it could be contended that the structural-modeling approach of the present study provided a better analysis for the multivariate relationships of the tripartite and *DSM–IV* dimensions for such reasons as (a) the dimensional nature of psychopathology (i.e., key features of *DSM–IV* disorders) was retained, and (b) the interrelationships among the *DSM–IV* and tripartite dimensions were examined after accounting for the influence of measurement error in the quantification of these domains (cf. Bagozzi, 1993; Green, Goldman, & Salovey, 1993). Also, it is possible that other methodological differences, such as sample composition and the measurement of PA and NA (these traits were assessed using the Multidimensional Personality Questionnaire in Watson et al.), were partly responsible for these differential results.

Although the present investigation evaluated one of the most appropriate samples to be studied to date (e.g., prior studies have often used samples in which anxiety and mood disorders were not well represented), certain disorders occurred infrequently in our data set (e.g., obsessive–compulsive disorder; although mood disorders occurred frequently, most patients with a depressive disorder had an anxiety disorder principal diagnosis). In this light, it would be of interest to examine the structural and parameter equivalence of our revised model (Figure 2) in other samples (e.g., inpatient populations). Moreover, as noted earlier, structural research of longitudinal data would be of considerable value in the verification of NA and PA (or related constructs; cf. Carver & White, 1994) as higher order dimensions exerting strong influence on the pathogenesis, course, and treatment response of the emotional disorders.

References

Akaike, H. (1987). Factor analysis and AIC. *Psychometrika, 52,* 317–332.

American Psychiatric Association. (1968). *Diagnostic and statistical manual of mental disorders* (2nd ed.). Washington, DC: Author.

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., rev.). Washington, DC: Author.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Amies, P. L., Gelder, M. G., & Shaw, P. M. (1983). Social phobia: A comparative clinical study. *British Journal of Psychiatry, 142,* 174–179.

Andrews, G. (1996). Comorbidity in neurotic disorders: The similarities are more important than the differences. In R. M. Rapee (Ed.), *Current controversies in the anxiety disorders* (pp. 3–20). New York: Guilford Press.

Andrews, G., Stewart, G., Morris-Yates, A., Holt, P., & Henderson, S. (1990). Evidence for a general neurotic syndrome. *British Journal of Psychiatry, 157,* 6–12.

Bagozzi, R. P. (1993). An examination of the psychometric properties of measures of negative affect in the PANAS-X scales. *Journal of Personality and Social Psychology, 65,* 836–851.

Barlow, D. H., Chorpita, B. F., & Turovsky, J. (1996). Fear, panic, anxiety, and disorders of emotion. In D. A. Hope (Ed.), *Perspectives on anxiety, panic, and fear* (Vol. 43, pp. 251–328). Lincoln, NE: University of Nebraska Press.

Beck, A. T., & Steer, R. A. (1987). *Manual for the revised Beck Depression Inventory.* San Antonio, TX: Psychological Corporation.

Beck, A. T., & Steer, R. A. (1990). *Manual for the Beck Anxiety Inventory.* San Antonio, TX: Psychological Corporation.

Bentler, P. M. (1990). Comparative fit indices in structural models. *Psychological Bulletin, 107,* 238–246.

Bentler, P. M., & Bonett, D. G. (1980). Significance tests and goodness of fit in the analysis of covariance structures. *Psychological Bulletin, 88,* 588–606.

Blashfield, R. K. (1990). Comorbidity and classification. In J. D. Maser & C. R. Cloninger (Eds.), *Comorbidity of mood and anxiety disorders* (pp. 61–82). Washington, DC: American Psychiatric Press.

Bollen, K. A. (1989). *Structural equations with latent variables.* New York: Wiley.

Borkovec, T. D., Abel, J. L., & Newman, H. (1995). Effects of psychotherapy on comorbid conditions in generalized anxiety disorder. *Journal of Consulting and Clinical Psychology, 63,* 479–483.

Borkovec, T. D., Lyonfields, J. D., Wiser, S. L., & Diehl, L. (1993). The role of worrisome thinking in the suppression of cardiovascular response to phobic imagery. *Behaviour Research and Therapy, 31,* 321–324.

Brown, E. J., Turovsky, J., Heimberg, R. G., Juster, H. R., Brown, T. A., & Barlow, D. H. (1997). Validation of the Social Interaction Anxiety Scale and the Social Phobia Scale across the anxiety disorders. *Psychological Assessment, 9,* 21–27.

Brown, T. A. (1996). Validity of the *DSM–III–R* and *DSM–IV* classification systems for anxiety disorders. In R. M. Rapee (Ed.), *Current controversies in the anxiety disorders* (pp. 21–45). New York: Guilford Press.

Brown, T. A. (in press). Generalized anxiety disorder and obsessive–compulsive disorder. In T. Millon, P. H. Blaney, & R. Davis (Eds.), *Oxford textbook of psychopathology.* New York: Oxford University Press.

Brown, T. A., Anson, A. M., & DiBartolo, P. M. (1996). *The distinctiveness of DSM–IV generalized anxiety disorder from major depression and dysthymia.* Manuscript in preparation.

Brown, T. A., Antony, M. M., & Barlow, D. H. (1992). Psychometric properties of the Penn State Worry Questionnaire in a clinical anxiety disorders sample. *Behaviour Research and Therapy, 30,* 33–37.

Brown, T. A., Antony, M. M., & Barlow, D. H. (1995). Diagnostic comorbidity in panic disorder: Effect on treatment outcome and course of comorbid diagnoses following treatment. *Journal of Consulting and Clinical Psychology, 63,* 408–418.

Brown, T. A., & Barlow, D. H. (1992). Comorbidity among anxiety disorders: Implications for treatment and *DSM–IV. Journal of Consulting and Clinical Psychology, 60,* 835–844.

Brown, T. A., Barlow, D. H., & Liebowitz, M. R. (1994). The empirical basis of generalized anxiety disorder. *American Journal of Psychiatry, 151,* 1272–1280.

Brown, T. A., & Chorpita, B. F. (1996). On the validity and comorbidity of the *DSM–III–R* and *DSM–IV* anxiety disorders. In R. M. Rapee (Ed.), *Current controversies in the anxiety disorders* (pp. 48–52). New York: Guilford Press.

Brown, T. A., Chorpita, B. F., Korotitsch, W., & Barlow, D. H. (1997). Psychometric properties of the Depression Anxiety Stress Scales (DASS) in clinical samples. *Behaviour Research and Therapy, 35,* 79–89.

Brown, T. A., Marten, P. A., & Barlow, D. H. (1995). Discriminant validity of the symptoms constituting the *DSM–III–R* and *DSM–IV* associated symptom criterion of generalized anxiety disorder. *Journal of Anxiety Disorders, 9,* 317–328.

Brown, T. A., Moras, K., Zinbarg, R. E., & Barlow, D. H. (1993). Diagnostic and symptom distinguishability of generalized anxiety disorder and obsessive–compulsive disorder. *Behavior Therapy, 24,* 227–240.

Carver, C. S., & White, T. L. (1994). Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: The BIS/BAS scales. *Journal of Personality and Social Psychology, 67, 319–333.*

Clark, D. A., Steer, R. A., & Beck, A. T. (1994). Common and specific dimensions of self-reported anxiety and depression: Implications for the cognitive and tripartite models. *Journal of Abnormal Psychology, 103,* 645–654.

Clark, L. A., & Watson, D. (1991). Tripartite model of anxiety and depression: Psychometric evidence and taxonomic implications. *Journal of Abnormal Psychology, 100,* 316–336.

Clark, L. A., Watson, D., & Mineka, S. (1994). Temperament, personality, and the mood and anxiety disorders. *Journal of Abnormal Psychology, 103,* 103–116.

Cohen, J., & Cohen, P. (1983). *Applied multiple regression/correlation analysis for the behavioral sciences* (2nd ed.). Hillsdale, NJ: Erlbaum.

Costa, P. T., & McCrae, R. R. (1988). Personality in adulthood: A six-year longitudinal study of self-reports and spouse ratings on the NEO Personality Inventory. *Journal of Personality and Social Psychology, 54,* 853–863.

Cox, B. J., Cohen, E., Direnfeld, D. M., & Swinson, R. P. (1996). Does the Beck Anxiety Inventory measure anything beyond panic attack symptoms? *Behaviour Research and Therapy, 34,* 949–954.

Di Nardo, P. A., Brown, T. A., & Barlow, D. H. (1994). *Anxiety Disorders Interview Schedule for DSM–IV: Lifetime Version (ADIS-IV-L).* San Antonio, TX: Psychological Corporation.

Di Nardo, P. A., Brown, T. A., Lawton, J. K., & Barlow, D. H. (1995, November). *The Anxiety Disorders Interview Schedule for DSM–IV Lifetime Version: Description and initial evidence for diagnostic reliability.* Paper presented at the 29th annual meeting of the Association for Advancement of Behavior Therapy, Washington, DC.

Emmelkamp, P. M. G. (1988). Maudsley Obsessional–Compulsive Inventory. In M. Hersen & A. S. Bellack (Eds.), *Dictionary of behavioral assessment techniques* (pp. 294–295). New York: Pergamon Press.

Fenigstein, A., Scheier, M. F., & Buss, A. H. (1975). Public and private self-consciousness: Assessment and theory. *Journal of Consulting and Clinical Psychology, 43,* 522–527.

Flett, G. L., Vredenburg, K., & Krames, L. (1997). The continuity of depression in clinical and nonclinical samples. *Psychological Bulletin, 121,* 395–416.

Frances, A., Widiger, T., & Fyer, M. R. (1990). The influence of classification methods on comorbidity. In J. D. Maser & C. R. Cloninger (Eds.), *Comorbidity of mood and anxiety disorders* (pp. 41–59). Washington, DC: American Psychiatric Press.

Green, D. P., Goldman, S. L., & Salovey, P. (1993). Measurement error masks bipolarity in affect ratings. *Journal of Personality and Social Psychology, 64,* 1029–1041.

Hodgson, R. J., & Rachman, S. J. (1977). Obsessional–compulsive complaints. *Behaviour Research and Therapy, 15,* 389–395.

Hoehn-Saric, R., McLeod, D. R., & Zimmerli, W. D. (1989). Somatic manifestations in women with generalized anxiety disorder: Psychophysiological responses to psychological stress. *Archives of General Psychiatry, 46,* 1113–1119.

Hope, D. A., & Heimberg, R. G. (1988). Public and private self-consciousness and social phobia. *Journal of Personality Assessment, 52,* 626–639.

Hudson, J. I., & Pope, H. G. (1990). Affective spectrum disorder: Does antidepressant response identify a family of disorders with a common pathophysiology? *American Journal of Psychiatry, 147,* 552–564.

Jaccard, J. C., & Wan, C. K. (1996). *LISREL approaches to interaction effects in multiple regression.* Thousand Oaks, CA: Sage.

Joiner, T. E. (1996). A confirmatory factor-analytic investigation of the tripartite model of depression and anxiety in college students. *Cognitive Therapy and Research, 20,* 521–539.

Joiner, T. E., Catanzaro, S. J., & Laurent, J. (1996). Tripartite structure of positive and negative affect, depression, and anxiety in child and adolescent psychiatric inpatients. *Journal of Abnormal Psychology, 105,* 401–409.

Jöreskog, K., & Sörbom, D. (1993). LISREL 8.12a [Computer software]. Chicago: Scientific Software.

Kendall, P. C., & Watson, D. (Eds.). (1989). *Anxiety and depression: Distinctive and overlapping features.* San Diego, CA: Academic Press.

Kendler, K. S., Neale, M. C., Kessler, R. C., Heath, A. C., & Eaves, L. J. (1992). Major depression and generalized anxiety disorder: Same genes, (partly) different environments? *Archives of General Psychiatry, 49,* 716–722.

Livesley, W. J., Schroeder, M. L., Jackson, D. N., & Jang, K. L. (1994). Categorical distinctions in the study of personality disorder: Implications for classification. *Journal of Abnormal Psychology, 103,* 6–17.

Lovibond, P. F., & Lovibond, S. H. (1995). The structure of negative emotional states: Comparison of the Depression Anxiety Stress Scales (DASS) with the Beck Depression and Anxiety Inventories. *Behaviour Research and Therapy, 33,* 335–342.

Lovibond, S. H., & Lovibond, P. F. (1995). *Manual for the Depression Anxiety Stress Scales* (2nd ed.). Syndey, Australia: Psychological Foundation of Australia.

Marten, P. A., Brown, T. A., Barlow, D. H., Borkovec, T. D., Shear, M. K., & Lydiard, R. B. (1993). Evaluation of the ratings comprising the associated symptom criterion of *DSM–III–R* generalized anxiety disorder. *Journal of Nervous and Mental Disease, 181,* 676–682.

Mattick, R. P., Peters, L., & Clarke, J. C. (1989). Exposure and cognitive restructuring for social phobia: A controlled study. *Behavior Therapy, 20,* 3–23.

Meehl, P. E. (1995). Bootstraps taxometrics: Solving the classification problem in psychopathology. *American Psychologist, 50,* 266–275.

Meng, X.-L., Rosenthal, R., & Rubin, D. B. (1992). Comparing correlated correlation coefficients. *Psychological Bulletin, 111,* 172–175.

Meyer, T. J., Miller, M. L., Metzger, R. L., & Borkovec, T. D. (1990). Development and validation of the Penn State Worry Questionnaire. *Behaviour Research and Therapy, 28,* 487–495.

Moldin, S. O., Rice, J. P., Erlenmeyer-Kimling, L., & Squires-Wheeler, E. (1994). Latent structure of *DSM–III–R* Axis II psychopathology in a normal sample. *Journal of Abnormal Psychology, 103,* 259–266.

Peterson, R. A., & Reiss, S. (1992). *Anxiety Sensitivity Index manual* (2nd ed.). Worthington, OH: IDS Publishing.

Rapee, R. M., Craske, M. G., & Barlow, D. H. (1995). Assessment instrument for panic disorder that includes fear of sensation-producing activities: The Albany Panic and Phobia Questionnaire. *Anxiety, 1,* 114–122.

Steer, R. A., & Beck, A. T. (1996). Generalized anxiety and panic disorders: Response to Cox, Cohen, Direnfeld, and Swinson (1996). *Behaviour Research and Therapy, 34,* 955–957.

Steiger, J. H. (1990). Structural model evaluation and modification: An interval estimation approach. *Multivariate Behavioral Research, 25,* 173–180.

Tallis, F., Davey, G. C. L., & Bond, A. (1994). The Worry Domains Questionnaire. In G. Davey & F. Tallis (Eds.), *Worrying: Perspectives on theory, assessment, and treatment* (pp. 285–297). New York: Wiley.

Tallis, F., Eysenck, M. W., & Mathews, A. (1992). A questionnaire for the measurement of nonpathological worry. *Personality and Individual Differences, 13,* 161–168.

Tellegen, A., Lykken, D. T., Bouchard, T. J., Wilcox, K. J., Segal, N. L., & Rich, S. (1988). Personality similarity in twins reared apart and together. *Journal of Personality and Social Psychology, 54,* 1031–1039.

Turner, S. M., Beidel, D. C., & Stanley, M. A. (1992). Are obsessional thoughts and worry different cognitive phenomena? *Clinical Psychology Review, 12,* 257–270.

Tyrer, P., Seivewright, N., Murphy, S., Ferguson, B., Kingdon, D., Barczak, B., Brothwell, J., Darling, C., Gregory, S., & Johnson, A. L. (1988). The Nottingham study of neurotic disorder: Comparison of drug and psychological treatments. *Lancet, 2,* 235–240.

Watson, D., & Clark, L. A. (1984). Negative affectivity: The disposition to experience aversive emotional states. *Psychological Bulletin, 96,* 465–490.

Watson, D., Clark, L. A., & Carey, G. (1988). Positive and negative affectivity and their relation to the anxiety and depressive disorders. *Journal of Abnormal Psychology, 97,* 346–353.

Watson, D., Clark, L. A., & Harkness, A. R. (1994). Structures of personality and their relevance to psychopathology. *Journal of Abnormal Psychology, 103,* 18–31.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54,* 1063–1070.

Watson, D., Clark, L. A., Weber, K., Assenheimer, J. S., Strauss, M. E., & McCormick, R. A. (1995). Testing a tripartite model: II. Exploring the symptom structure of anxiety and depression in student, adult, and patient samples. *Journal of Abnormal Psychology, 104,* 15–25.

Watson, D., Weber, K., Assenheimer, J. S., Clark, L. A., Strauss, M. E., & McCormick, R. A. (1995). Testing a tripartite model: I. Evaluating the convergent and discriminant validity of anxiety and depression symptom scales. *Journal of Abnormal Psychology, 104,* 3–14.

World Health Organization. (1993). *The ICD-10 classification of mental and behavioural disorders: Diagnostic criteria for research.* Geneva, Switzerland: Author.

Zinbarg, R. E., & Barlow, D. H. (1996). Structure of anxiety and anxiety disorders: A hierarchical model. *Journal of Abnormal Psychology, 105,* 181–193.

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