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Locating Eating Pathology within an Empirical Diagnostic Taxonomy: Evidence from a Community-Based Sample

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Abstract

Existing structural models of psychopathology need to be expanded to include additional diagnostic constructs beyond mood, anxiety, substance use, and antisocial behavior disorders. The goal of this study was to locate eating disorders within a hierarchical structural model of psychopathology that is anchored by broad Internalizing and Externalizing factors. Participants were female adolescent twins ($N = 1,434$) from the Minnesota Twin Family Study. We compared the fit of four models in which eating disorders (a) defined their own diagnostic class, (b) represented a subclass within Internalizing, (c) formed a subclass within Externalizing, and (d) were allowed to cross-load on both Internalizing and Externalizing. In the best-fitting model, eating disorders formed a sub-factor within Internalizing. These findings underscore the value of developing more comprehensive empirically based models of psychopathology to increase our understanding of diverse mental disorders.

Keywords

eating disorders; disordered eating; comorbidity; diagnosis; and classification

Several large-scale epidemiological studies have demonstrated that mental disorders co-occur more frequently than expected by chance (Boyd, et al., 1987; Kessler, Chiu, Demler, & Walters, 2005; Kessler, et al., 1994). This phenomenon of co-occurrence of different disorders within the same person is referred to as comorbidity (Feinstein, 1970) and poses serious conceptual problems for current diagnostic and classification systems. The *Diagnostic and Statistical Manual of Mental Disorders (4th edition) (DSM)* (APA, 2000) groups disorders on a rational, phenomenological basis that is not well supported by structural analyses of comorbidity data (Krueger & Markon, 2006). For example, although generalized anxiety disorder and social phobia are grouped under the “anxiety disorders,” the two disorders are only moderately associated. In contrast, the phenotypic (Mineka, Watson, & Clark, 1998; Watson, 2009) and genotypic (Kendler, 2004; Kendler, Neale, Kessler, Heath, & Eaves, 1992) correlations between generalized anxiety disorder and major depression both are very high.

To remedy this problem with the *DSM*, researchers have modeled the underlying dimensions of common psychiatric disorders using second-order confirmatory factor analyses, a form of structural equation modeling, and have found support for meaningful hierarchical groupings (Cox, Clara, & Enns, 2002; Krueger, 1999; Krueger, Caspi, Moffitt, & Silva, 1998; Slade & Watson, 2006; Vollebergh, et al., 2001). Conceptually, confirmatory factor analyses model the degree of covariation among observed indicators. Similar to exploratory factor analysis, confirmatory factor analysis indicates which observed indicators tend to covary or co-occur across individuals and which do not. Specifically, in a hierarchical confirmatory factor analysis structure, strongly correlated syndromes are located together within the same liability factor or spectrum, whereas weakly related disorders are distinguished by relating to different factors (although the higher order factors themselves may be correlated). Dimensional hierarchical models of mental disorders have clear advantages over categorical nosologies. These models are able to account better for the excessive diagnostic co-occurrence, poor diagnostic stability, and weak reliability often observed in psychological disorders (Watson, 2005), thus making 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” (Watson, 2005, p. 524).

A number of studies have found evidence of a highly consistent hierarchical structure (for a review and meta-analysis, see Krueger & Markon, 2006). Specifically, two correlated factors, labeled Internalizing and Externalizing, have emerged. The Internalizing factor subsumes the mood and anxiety disorders, which further divide into two sub-factors: (a) distress disorders, which include major depression, dysthymic disorder, generalized anxiety disorder, and posttraumatic stress disorder and (b) fear disorders, which encompass panic disorder, phobias, and obsessive compulsive disorder (OCD). Externalizing consists of alcohol abuse/dependence, substance abuse/dependence, conduct disorder, and adult antisocial behavior. This structure has received strong empirical support and has been replicated in both phenotypic (Slade & Watson, 2006) and genotypic investigations (Hettema, Neale, Myers, Prescott, & Kendler, 2006; Kendler, Prescott, Myers, & Neale, 2003) and across multiple nations (Krueger, et al., 1998; Vollebergh, et al., 2001). The broadband Internalizing-Externalizing structure is also invariant between sexes (Kramer, Krueger, & Hicks, 2007) and stable over time (Krueger, et al., 1998; Measelle, Stice, & Hogansen, 2006; Vollebergh, et al., 2001).

However, for a quantitative model to be broadly useful, it must be extended and refined. Specifically, this model lacks comprehensiveness because many disorders have not been included in major structural studies. Eating pathology is one of the major areas of psychopathology that has not been included in multivariate modeling of psychiatric comorbidity. Examining where eating disorders belong in the diagnostic taxonomy represents an important extension of previous statistical models of mental disorders, given that eating disorders demonstrate the highest mortality rate of any class of mental illness (Harris & Barraclough, 1998) and affect 5.9% of women in their lifetimes (Hudson, Hiripi, Pope, & Kessler, 2007).

Current Conceptualization of Eating Disorders

The *DSM* currently recognizes two types of eating disorders – anorexia nervosa and bulimia nervosa – which are classified as “severe disturbances in eating behavior” (APA, 2000), as well as binge eating disorder, which is currently listed in the main text of the *DSM-IV* as a specific example of an *eating disorder not otherwise specified*. Anorexia nervosa is a self-starvation syndrome marked by a refusal to maintain a minimally acceptable body weight. Individuals with anorexia nervosa are intensely afraid of gaining weight and experience a significant disturbance in the way they perceive their body weight/shape. Bulimia nervosa is characterized by body image disturbance and recurrent episodes of binge eating and

inappropriate compensatory behavior(s) (e.g., fasting, self-induced vomiting, excessive exercise, diuretics use, and laxative use), which occur in the absence of low weight. Finally, binge eating disorder is characterized by recurrent episodes of binge eating. However, in contrast to those with bulimia nervosa, their binge eating episodes occur in the absence of the regular use of inappropriate compensatory behaviors.

All three types of eating disorders share several behavioral and cognitive symptoms as well as many of the same patterns of comorbidity. Specifically, eating disorders display significant (lifetime) comorbidity with each other—as demonstrated in their emergence in a single class of disorders using latent class analysis (Sullivan & Kendler, 1998)—and with mood and anxiety disorders (for a recent review see Herzog & Eddy, 2007). These similarities seem to validate their placement within the same diagnostic class and appear to support suggestions that eating disorders may represent a variant of internalizing disorders (Treasure, 2006). However, it is important to note that although eating disorders are linked to mood and anxiety disorders by way of their associations with negative affectivity, this alone is not sufficient evidence to conclude that they fall within a common class of disorders, in general, or within the internalizing disorders, in particular. Rather, given the ubiquitous associations between negative affectivity and virtually all forms of psychopathology, including the externalizing disorders (see Mineka, et al., 1998; Ormel, Rosmalen, & Farmer, 2004), these data may suggest that eating disorders are indicators of a nonspecific “general psychopathology” factor. Moreover, studies distinguishing among eating disorder diagnoses and behaviors indicate several important differences in patterns of comorbidity, as discussed below.

Comorbidity Patterns in Eating Disorders

The most salient differences within the class of eating disorders have emerged between the restricting subtype of anorexia nervosa and eating disorders characterized by bulimic symptoms (e.g., binge eating and/or purging in the absence of low weight). For example, anorexia nervosa, but not bulimia nervosa, is highly comorbid with obsessive compulsive disorder (Godart, Berthoz, Perdereau, & Jeammet, 2006; Kaye, Bulik, Thornton, Barbarich, & Masters, 2004; Lilenfeld, et al., 1998). Obsessive compulsive disorder has been shown to occur in 22-37% of patients with anorexia nervosa but only 3-13% of patients with bulimia nervosa (Godart, Berthoz, Rein, et al., 2006; Speranza, et al., 2001; Thornton & Russell, 1997) and 1.1% of matched control participants (Speranza, et al., 2001). Recent studies also suggest that the prevalence of other fear-based anxiety disorders, including social phobia and agoraphobia, are significantly elevated in women with anorexia nervosa compared to women with bulimia nervosa (Godart, Berthoz, Rein, et al., 2006). These results suggest that anorexia nervosa may co-aggregate with obsessive compulsive disorder and phobias, each of which represents a fear-based internalizing disorder.

In contrast to the restricting subtype of anorexia nervosa, bulimic syndromes and behaviors (including the binge purge subtype of anorexia nervosa) are more often comorbid with substance use disorders (Bulik, et al., 2004; Kendler, et al., 1991; Lilenfeld, et al., 1997; Piran & Robinson, 2006; von Ranson, Iacono, & McGue, 2002). Blinder and colleagues (2006) found that alcohol abuse/dependence was twice as likely to occur, and poly-substance abuse/dependence three times more likely to occur, in women with bulimia nervosa compared to those with anorexia nervosa. Substance abuse/dependence rates also are elevated in individuals with binge eating disorder. Data from the National Comorbidity Survey–Replication suggest that approximately 15% of the general United States population (Kessler, Berglund, et al., 2005) and 27% of individuals with anorexia nervosa (Hudson, et al., 2007) have had at least one (lifetime) substance use disorder, whereas approximately 35% of respondents with sub- or full-threshold binge eating disorder have had at least one (lifetime) comorbid substance use disorder (Hudson, et al., 2007). The significant association between bulimic symptoms and

syndromes and substance disorders (for a review, see Gadalla & Piran, 2007) led researchers to posit a shared nonspecific risk factor for externalizing problems and bulimic pathology (Stice, 2001). This hypothesis is supported by prospective studies, which have found that aggression, alcohol misuse, and illicit substance use predict the subsequent onset of bulimic symptoms (Marmorstein, von Ranson, Iacono, & Succop, 2007; Vogeltanz-Holm, et al., 2000).

Overview of the Current Research

Based on an examination of the existing literature, it is apparent that several questions regarding the eating disorders remain unanswered, specifically: Do eating disorders represent their own class of disorders as implied by the current *DSM*? Alternatively, are they better modeled as indicators of the internalizing disorders, the externalizing disorders, or perhaps as a hybrid of the two? This study was designed to address these questions by examining where eating disorders belong within a structural model of mental illness. Given the comorbidity patterns between eating disorders and other forms of psychopathology, we hypothesize that bulimia nervosa and binge eating disorder would be significant indicators of both internalizing and externalizing psychopathology, whereas anorexia nervosa would be a significant indicator of internalizing psychopathology.

Method

Participants and Procedure

The sample consisted of female adolescent twins ($N = 1,434$) who participated in the Minnesota Twin Family Study (MTFS) (Iacono & McGue, 2002), a population-based, longitudinal study of the development of substance use and related disorders in reared-together male and female twins and their parents. Twins were identified from birth records obtained through the State of Minnesota and located through public databases (Lykken, McGue, Bouchard, & Tellegen, 1990). This statewide sample is broadly representative of the Minnesota population (Iacono & McGue, 2002). Almost all of the twins are Caucasian (over 95%).

The present sample consisted of two cohorts of female twins. Male twins were not included in analyses because males were not assessed for eating pathology (Iacono & McGue, 2002). The pre-adolescent cohort ($N = 760$) was recruited at approximately age 11 and given the opportunity to participate in follow-up assessments at ages 14 and 17. The late-adolescent cohort ($N = 674$) was recruited at approximately age 17. The pre-adolescent and late-adolescent protocols were very similar, although protocols were adjusted to include age-appropriate assessments. At study intake, all assessments were conducted in-person in the MTFS laboratory. Whenever possible, follow-up assessments also were conducted in-person (for the pre-adolescent cohort), with phone interviews scheduled when an in-person visit was not possible. At follow-up 1, 93.4% participated and of those, 86.5% visited in person. At follow-up 2, 91.4% participated, and 66.7% visited in person. To merge the pre- and late- adolescent cohorts' *DSM* diagnostic data for statistical analyses, age-17 lifetime diagnoses were created for participants in the pre-adolescent cohort aggregating clinical diagnostic information across the intake and two follow-up assessments. These age-17 lifetime diagnostic data from the pre-adolescent cohort were then merged with lifetime diagnostic intake data from the late-adolescent cohort.¹

¹To evaluate the impact of using aggregated lifetime diagnoses for the younger cohort, we re-ran structural analyses using only data available from the age-17 assessment for both cohorts. We found that both methods of analysis resulted in the same best-fitting final model based on a comparison of the smallest (i.e., most negative) Bayesian Information Criterion value.

Measures

Body Mass Index Percentile—Body mass index percentile was calculated using sex- and age-specific 2000 Centers for Disease Control and Prevention Growth Charts for the United States (CDC, 2009). Height and weight were measured at each visit.

Eating, Internalizing, and Externalizing Disorders—With one exception, diagnostic criteria were based on the *DSM-III-R* (APA, 1987), the diagnostic system in place when the study began; the exception was binge eating disorder, which was first included in the *DSM-IV*, so its diagnostic criteria were based on the *DSM-IV* (not *DSM-III-R*). Eating disorder diagnoses were assessed with the Eating Disorders Structured Clinical Interview (EDSCI), a structured interview based on the eating disorders module of the Structured Clinical Interview for *DSM-III-R* Axis I disorders (Spitzer, Williams, Gibbon, & First, 1990a) modified to assess both *III-R* and *IV* as well as to assess young subjects. Mothers also completed a parent version of the EDSCI at each assessment point. Bulimia nervosa and binge eating disorder, both of which involve recurrent episodes of binge eating associated with a lack of control during the binge, were significantly correlated (polychoric $r=.41$, $p<.001$). Hence, due to their low base rates in the present sample (see Table 1), they were combined in structural analyses to form a composite *bulimic syndromes* variable.

The Diagnostic Interview for Children and Adolescents-Revised (DICA-R) (Reich & Welner, 1988) was used to assess non-eating pathology when the participants were 11 and 14. At 17, the mother continued to report via the DICA, but the participants reported major depression, generalized anxiety disorder, and social and specific phobias using the Structured Clinical Interview for *DSM* diagnoses (SCID-I) (Spitzer, et al., 1990a), conduct disorder and adult antisocial behavior (i.e., the “adult” criterion “C” symptoms of antisocial personality disorder arising since the age of 15) using the Structured Clinical Interview for *DSM* Personality Disorders (SCID-II) (Spitzer, Williams, Gibbon, & First, 1990b), and drug and alcohol abuse/dependence using the Substance Abuse Module from the Composite International Diagnostic Interview (CIDI) (Robins, et al., 1988). The substances assessed included alcohol, cannabis, amphetamines, barbiturates, tranquilizers, cocaine, heroin, opiates, PCP, psychedelics, and inhalants. These assessments were combined to assess the lifetime presence of these internalizing and externalizing disorders. Individuals who fulfilled a lifetime diagnosis of generalized anxiety disorder and/or a lifetime diagnosis of overanxious disorder of childhood were coded as meeting lifetime criteria for generalized anxiety disorder. Due to the low base rates of specific illicit drug abuse/dependence categories, individual categories of illicit drug abuse and dependence were combined to create a composite *drug abuse/dependence* variable. Similarly, alcohol abuse and dependence were combined to create a composite *alcohol abuse/dependence* variable.

All clinical interviews were administered by trained research assistants and advanced graduate students who held either a bachelor's or master's degree in psychology. All structured interviews were reviewed and final symptom assignments were made by a consensus of two or more advanced clinical psychology graduate students with specific training in differential diagnosis and descriptive psychopathology. Both members of the consensus team had to agree as to the presence or absence of a diagnostic symptom. Consensus teams reviewed mother and child interviews independently, assigning symptoms without knowledge of how both the twin and the mother answered the same question. A symptom was counted toward the diagnosis if either the mother or the twin endorsed it, consistent with best-estimate diagnostic procedures (Bird, Gould, & Staghezza, 1992). In other words, information was combined across informants after the consensus review was completed to provide diagnoses that would make the best use of all available information. Best estimate diagnoses represent a more valid and comprehensive approach compared to diagnoses based on either report alone, because each

reporter contributes valid information not accounted for by the other reporter (Achenbach, McConaughy, & Howell, 1987; Burt, Krueger, McGue, & Iacono, 2001). Evidence suggesting the presence of a symptom was given more weight than refusal to endorse a symptom, particularly if the symptom endorsed was corroborated by indications of functional impairment or by multiple sources. Lifetime diagnoses for internalizing and externalizing disorders were coded as 0 (absent), 1 (possible, missing two diagnostic criteria), 2 (probable, missing one diagnostic requirement) and 3 (definite, satisfying all diagnostic requirements), in order to capture a more dimensional (vs. categorical) conceptualization of psychopathology, while at the same time recognizing that our method of assessment still is based on *DSM* diagnostic categories. For conduct disorder, because following these rules would lead to a “possible” diagnosis being assigned to a person with only a single symptom, we did not use the possible category for this disorder. For the composite alcohol and drug abuse/dependence variables, these coded values reflected the number of symptoms of abuse or dependence that were present (e.g., a score of three indicated three symptoms were present). (Note that the three dependence symptoms corresponded to a definite diagnosis of substance dependence.)

Diagnostic reliability was examined by pairs of clinicians who reviewed all clinically-relevant information (including audio tapes). Clinicians determined the presence or absence of each symptom without knowledge of the original diagnostic assessment. Although this method for establishing reliability is commonly used, it can be expected to yield higher estimates of reliability than would be expected if different clinicians interviewed the same person twice. The kappa values for this sample indicate good to excellent interrater reliability for 0-3 codings of psychopathology (see also Iacono, Carlson, Taylor, Elkins, & McGue, 1999 for other interrater reliability data in this sample): .84 for major depression, .80 for social phobia, .75 for simple phobia, .59 for generalized anxiety disorder, .82 for adult antisocial behaviors, .80 for conduct disorder, .98-.99 for alcohol misuse disorders, .92 or above for the drug misuse disorders assessed, .68 for anorexia nervosa, .69 for bulimia nervosa, and .71 for binge eating disorder.

Statistical Analyses

Data were analyzed using SPSS 16.0 for Windows (SPSS, 2007) and Mplus version 5 (Muthén & Muthén, 2007). Missing data were handled using the default option in Mplus version 5, which estimates models under missing data theory using all available data (see Muthén & Muthén, 2007). Raw data were used as input for Mplus analyses. All models were estimated using robust weighted least squares (WLSMV) procedures, which is the appropriate choice for analyzing categorical, ordinal indicators (such as psychiatric diagnoses) because it accounts for a non-normal distribution of measured variables (Bollen, 1989; Krueger, et al., 1998). In order for models to be identified, the variance of each latent factor was fixed to 1.0. To account for non-independent sampling of twins within families (i.e., to fully account for the nested nature of the data), all structural analyses were conducted with individual twin-level data clustered within twin pairs using the *cluster* command in Mplus.

Overall model fit was evaluated using the values for acceptable fit levels recommended by Hu and Bentler (1999): (a) the *comparative fit index (CFI)* and *Tucker-Lewis fit index (TLI)*, close to .95 or greater and (b) the *root mean square error of approximation (RMSEA)*, close to .06 or less. Hu and Bentler's (1999) use of the phrase “close to” is deliberate because these recommended cutoff values fluctuate as a function of model conditions and whether or not a particular fit index is used alone or in combination with other indices (see Brown, 2006 for further discussion of goodness-of-fit indices). These indices were selected based on their favorable performance in Monte Carlo research (Hu & Bentler, 1998, 1999; Marsh, Balla, & McDonald, 1988) and because they provide a range of information about model fit (i.e.,

absolute fit, fit adjusting for model parsimony, and comparative/incremental fit) (Brown, 2006).

The *Bayesian Information Criterion (BIC)* was used to compare nested and non-nested models using the formula provided by Raftery (1995) $\chi^2 - df * \ln(N)$, where χ^2 is the chi-square fit statistic for the model, df is the corresponding degrees of freedom, and N is the sample size. The objective of Bayesian approaches to model selection is to choose the model that has the highest posterior probability (i.e., the highest *Bayes* factor or odds). Thus, given the observed data, the difference in *BIC* values provides an odds ratio of the probability that the second model is correct divided by the probability that the first model is correct. In comparing two models, a difference between *BIC*s of 2-6 represents positive evidence (3:1 to 20:1 odds), differences of 6-10 represent strong evidence (20:1 to 150:1 odds), and differences larger than 10 represent very strong evidence (greater than 150:1 odds) in favor of the model with the *smaller* (i.e., more negative) *BIC* value, respectively (Kass & Raftery, 1995; see also Nagin, 1999). An important feature of the *BIC* is that it balances two important aspects of model fit: the discrepancy between obtained and model implied moments and the number of parameters estimated in the fitted model. Thus, the *BIC* favors parsimonious models that also accurately reproduce the obtained data (for further discussion of the *BIC*, see Krueger, et al., 1998).

Results

For the pre-adolescent cohort, mean (*SD*) body mass index percentile was 61.05 (28.07) at intake, 66.88 (24.30) at follow-up 1, and 63.99 (25.24) at follow-up 2. Mean (*SD*) body mass index percentile for the late-adolescent cohort was 60.45 (24.20) at intake. We present the *N*, *SD*, and prevalence for each diagnostic indicator in Table 1. Although confirmatory factor analysis utilizes variances and covariances (rather than correlations), polychoric correlations between *DSM* diagnoses are reported in Table 2 for ease of interpretation.

Replication of the Structure of Common Mental Disorders

Our first goal was to replicate the structure of common mental disorders documented in the literature (Krueger & Markon, 2006; Watson, 2005). Specifically, we carried out a confirmatory factor analysis with two correlated latent exogenous factors labeled Internalizing and Externalizing. The latent Internalizing factor consisted of two latent endogenous sub-factors called Distress and Fear. Major depression and generalized anxiety disorder were indicators of the Distress factor and social phobia and specific phobia were indicators of the Fear factor. Alcohol abuse/dependence, drug abuse/dependence, adult antisocial behaviors, and conduct disorder were indicators of the Externalizing factor. These disorders were chosen because they have been included in previous structural models of mental illness and because their prevalence in the current sample was sufficient to carry out analyses with adequate statistical precision.

Although the structure of common mental disorders is very robust (for a meta-analysis, see Krueger & Markon, 2006), we believe it is nevertheless important to examine this baseline structure in the MTFS (1) to ensure that there are not any peculiarities within the current sample and (2) to increase our confidence that the eating pathology results will replicate/generalize to other samples. Goodness-of-fit indices indicated that this model fit the data adequately ($\chi^2(13) = 74.83$, $p < .001$, $CFI = .946$, $TLI = .937$, $RMSEA = .058$), replicating previous structural work on common mental disorders. Subsequent structural equation models reported herein build on this structure.

Examining Eating Pathology within the Diagnostic Taxonomy

To determine where eating disorders belong in a quantitative structural model of the diagnostic taxonomy, we initially evaluated the fit of four models, representing the broad range of hierarchical models incorporating eating pathology: (1) In Model 1, Eating Pathology represented its own latent exogenous factor, with anorexia nervosa and bulimic syndromes serving as observed indicators of a latent eating pathology construct (see Figure 1). In this model, the latent Eating Pathology factor was allowed to covary with both the Internalizing and Externalizing factors; (2) Model 2 was a second-order confirmatory factor analysis in which a latent endogenous Eating Pathology factor was regressed on Internalizing (see Figure 1); (3) In Model 3, eating disorders were regressed on Externalizing (see Figure 1); and (4) finally, in Model 4, Eating Pathology was estimated as an indicator of both the Internalizing and Externalizing factors (see Figure 1), consistent with recent research that replicated and extended a structural model of mental disorders by allowing certain disorders to cross-load on the latent Internalizing and Externalizing factors (James & Taylor, 2008; Miller, Fogler, Wolf, Kaloupek, & Keane, 2008).

Comparison of Models 1-4

Fit indices indicated that Model 3, in which the eating disorders were regressed on the Externalizing factor, resulted in a somewhat poor overall fit (Table 3), whereas Models 1, 2, and 4 demonstrated better fits to the data (Table 3). A comparison of BIC values (which are preferable to other fit indices for comparing nested and non-nested models) indicated that Model 2, in which Eating Pathology was estimated as an indicator of the higher order Internalizing factor—rather than as a separate Eating Pathology factor or as an indicator of both the Internalizing and Externalizing factors—is the best-fitting remaining model (i.e., when comparing Models 1, 2, and 4; see Table 3). This indicates that overall eating pathology is not a good indicator of the externalizing dimension.

Cross-Loadings of Individual Eating Disorder Indicators on Externalizing

Because we hypothesized that bulimia nervosa and binge eating disorder should be significant indicators of externalizing psychopathology, whereas anorexia nervosa would not be a significant indicator of the externalizing spectrum, we were interested in determining whether the individual eating disorder diagnostic indicators had significant cross-loadings on Externalizing. We carried out two *separate* confirmatory factor analyses in which each individual eating disorder indicator (i.e., anorexia nervosa and bulimic syndromes) was allowed to load simultaneously on: (1) the Eating Pathology factor within Internalizing and (2) the Externalizing factor.

Fit indices indicated that allowing bulimic syndromes to cross-load on Externalizing (labeled Model 5) did not result in a better overall model fit compared to a model in which anorexia nervosa was allowed to cross-load on Externalizing (labeled Model 6) (see Table 3). Moreover, allowing the individual eating disorder diagnostic indicators to cross-load on Externalizing did not improve model fit compared to Model 2 (in which Eating Pathology was regressed on Internalizing).

Discussion

Summary of Findings

The goal of the present investigation was to determine where eating pathology belongs within a structural model of the diagnostic taxonomy. In the best-fitting structural model, eating disorders formed a sub-factor of the latent Internalizing factor, rather than a sub-factor of the latent Externalizing factor, or their own latent class of disorder as suggested by the *DSM-IV*.

Thus, the results of the study indicate that eating pathology is primarily a variant of internalizing disorders. This finding is consistent with a great deal of research documenting high rates of comorbidity between eating disorders and internalizing disorders, particularly depression and anxiety (Blinder, et al., 2006; Godart, Berthoz, Perdereau, et al., 2006; Hudson, et al., 2007).

Study Limitations

The present study has certain limitations that may affect the interpretation of our results. First, because the present study did not assess male participants for eating pathology, it is unclear whether our results would generalize to men. This is important because recent studies indicate that sex differences in the prevalence of common mental disorders stem from sex differences in latent Internalizing and Externalizing propensities (Kramer, et al., 2007).

Second, participants in the study were all approximately 17 years old, and had not yet passed through the peak period of risk for eating disorder onset (as well as some other comorbid disorders). Because our rates of bulimic syndromes were low (Lewinsohn, Striegel-Moore, & Seeley, 2000; Stice, Marti, Shaw, & Jaconis, 2009), we may have limited our ability to examine associations between bulimic syndromes and externalizing psychopathology. The lifetime prevalence of bulimia nervosa and binge eating disorder will increase as participants pass through young adulthood. Thus, it will be important to test whether the structure we obtained changes over time. Nonetheless, previous structural analyses of common psychiatric illnesses indicate that the structure of mental disorders is strongly stable over time (Krueger, et al., 1998; Vollebergh, et al., 2001), even in early and late adolescent samples in which developmental changes are occurring much more quickly than in adulthood (Huizink, van den Berg, van der Ende, & Verhulst, 2007; Measelle, et al., 2006). In addition to our rates of bulimic syndromes being low, our rates of anorexia nervosa tended to be high. Although we cannot explain with certainty what accounts for this elevated rate, it is important to note that our lifetime prevalence estimates for definite diagnoses of anorexia nervosa are within the range of past epidemiological studies (0.6% to 2.6%) (Favaro, Ferrara, & Santonastaso, 2003; Hudson, et al., 2007; Isomaa, Isomaa, Marttunen, Kaltiala-Heino, & Björkqvist, 2009; Stice, et al., 2009; Wade, Bergin, Tiggemann, Bulik, & Fairburn, 2006).

Third, the factor loading of anorexia nervosa on the latent Eating Pathology factor was relatively low compared to the loading of bulimic syndromes. These results suggest that anorexia nervosa is not a particularly strong marker of Eating Pathology. However, the lowered loading of anorexia nervosa on Eating Pathology might be due to the fact that we combined bulimia nervosa and binge eating disorder into one diagnostic class, which may have over-modeled bulimic disorders and under-modeled anorexia nervosa. Moreover, because eating disorders not otherwise specified represents the most common eating disorder diagnosis (Paulo, Barbara, Sónia, & Hans, 2007), there likely remains a substantial amount of eating pathology variance that was not modeled in our structural analyses. It may be that studies including a broader range of clinically significant eating disorders would find that anorexia nervosa combines with restrictive forms of eating disorders not otherwise specified and loads more strongly on the latent Eating Pathology factor. It also is possible that anorexia nervosa would load more strongly on an independent latent factor ².

²To examine the hypothesis that anorexia nervosa would load more strongly on an independent latent factor, we allowed anorexia nervosa to load on a latent exogenous factor, which covaried with both Internalizing and Externalizing, and bulimic syndromes to load on Distress within Internalizing. Because we had only one indicator of anorexia nervosa, and at least two indicator variables are required to model a latent factor, we constrained the factor loading for anorexia nervosa to the reliability of the diagnosis (i.e., kappa). Results indicated that this model fit the data better than our final Model 2 ($\chi^2(23) = 94.37, p < .001, CFI (= .942), TLI (= .937), RMSEA (= .047), BIC (= -72.80)$), suggesting that anorexia nervosa is largely independent of the Internalizing and Externalizing latent factors. Future studies are needed to replicate these results by including multiple indicators of anorexia-related pathology.

The lowered loading of anorexia nervosa on Eating Pathology also may be due to the young age of our participants, which may have lowered the comorbidity between anorexia nervosa and bulimic syndromes, given that diagnostic cross-over between eating disorder diagnoses and subtypes typically occurs several years after onset (Eddy, et al., 2002; Tozzi, et al., 2005).

Finally, although the correlation between bulimia nervosa and binge eating disorder was significant, it was modest. Thus, our decision to collapse across bulimia nervosa and binge eating disorder could have affected our results in unforeseen ways. Future studies are needed in order to examine whether the structure we found replicates when modeling bulimia nervosa and binge eating disorder as separate indicators of eating pathology.

Implications and Future Directions

Despite its limitations, this study has several strengths. First, although previous research has examined the internal structure of eating pathology and its association with other disorders using latent class analysis (Sullivan & Kendler, 1998), this study is the first to examine where eating disorders belong within a hierarchical model of mental illness. In addition, this study was based on a large community-dwelling sample ($N = 1,434$). Finally, these data are of significant potential value for understanding the development of psychopathology, as discussed below.

Diagnostic Implications—Due to problems with the current *DSM* (e.g., excessive diagnostic comorbidity, poor diagnostic stability, and weak reliability), many researchers have suggested that future editions of the *DSM* should move toward a more empirically based model of psychopathology (Clark, 2007; Helzer, Kraemer, & Krueger, 2006; Watson & Clark, 2006). These proposals aim to supplement or supplant the current *DSM-IV* system—which groups disorders into putatively discrete diagnostic classes (e.g., eating disorders, mood disorders, anxiety disorders) based on phenomenological similarities—with empirically derived dimensional groupings that reflects the covariation among mental disorders. In the current edition of the *DSM*, eating disorders are phenomenologically grouped together within the same diagnostic class because they all involve abnormal eating patterns. However, our analyses, which directly modeled the statistical covariation among disorders, provide preliminary quantitative evidence that eating disorders may be better characterized if they were nomologically³ (Cronbach & Meehl, 1955) grouped together within the internalizing spectrum.

A major advantage of an empirically derived diagnostic and classification system is that it can be used to guide the design of future etiologic psychopathology research. For example, although the present analyses were cross-sectional and, therefore, cannot establish common genetic vulnerabilities that may give rise to the phenotypic structure of mental disorders, results both from cross-sectional and longitudinal biometrical studies have found that the phenotypic structure of mental disorders is highly heritable (Huizink, et al., 2007; Kendler, et al., 2003; Kendler, Walters, Neale, Kessler, & et al., 1995; Krueger, et al., 2002). These findings demonstrate that disorders with high levels of phenotypic similarity are also linked at an etiologic level. Thus, a hierarchical model of mental disorders has the potential to facilitate the examination of general (i.e., cross-disorder) genetic influences that may confer risk for various

³The term *nomologically* is derived from the word *nomological*, which means “lawful” and is based on Cronbach and Meehl’s (1955) paper on construct validity in which they developed the concept of nomological networks (or “an interlocking system of laws that constitute a theory” p. 290). Their proposed laws include describing: (a) statistical associations between variables, (b) associations between theories and variables, and (c) associations between various theories. Thus, the term “nomologically” is being used to highlight the fact that rational theories (or rationally derived nosological systems) constitute necessary but not sufficient evidence of construct validity.

forms of eating pathology, given that the phenotypic relations among the externalizing and internalizing syndromes have been shown to arise from common genetic vulnerabilities.

Conceptualizing eating pathology as a sub-factor within the internalizing spectrum also provides a framework for future biometrical studies to explore specific etiologic mechanisms that give rise to the development of various eating disorders. In fact, studying eating disorders in isolation from other related disorders is not likely to be as profitable as research examining the full spectrum of mental disorders, given that it is difficult to determine whether an etiologic factor is specific to eating disorders when other disorders that share common underlying features are not studied in tandem.

Of course the statistical covariation between disorders is not the only important consideration when formulating a nosological system. In order to “carve nature at its joints” it will be necessary to consider relevant data from a variety of methodological and theoretical viewpoints. For example, a primary purpose of diagnosis is to provide information regarding whether different disorders or symptom constellations show differential clinical course, outcome, and treatment response. Although the current study was not designed to address these questions, it is important to note that available evidence from neuroscience, genetics, epidemiology and therapeutics generally provide strong support for the validity and clinical utility of the phenotypic structure of common mental disorders (for a review, see Andrews, et al., in press).

Conclusion

The results of the current study provide preliminary evidence that eating pathology may be best conceptualized as a distinct type of internalizing disorder distinguished by undue influence of body weight and shape on self-evaluation. Given the significant heterogeneity within eating disorder diagnoses, as well as the increased prevalence of eating disorders not otherwise specified compared to anorexia nervosa and bulimia nervosa, future research may benefit by examining where the individual symptoms dimensions that comprise *DSM*-defined eating disorders fall within this quantitative scheme.⁴

Finally, as Cronbach and Meehl (1955) stated in their classic paper on construct validity, “Learning more about a theoretical construct is a matter of elaborating the nomological network in which it occurs, or of increasing the definiteness of its components” (p.290); thus, as the number of disorders included in statistical analyses of the diagnostic taxonomy increases, the more we will learn about the overall nature of psychopathology.

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⁴To address this issue, we examined where disordered eating behaviors and attitudes fall within a structural model of mental illness using the subscales of the Minnesota Eating Behaviors Survey (MEBS) as indicators of a latent eating pathology factor (instead of *DSM* eating disorder diagnoses). The results of this analysis indicated that disordered eating attitudes and behaviors formed a sub-factor of a latent Internalizing factor, with Binge Eating and Compensatory Behaviors cross-loading on an Externalizing factor. We chose not to report results using the MEBS in our main analyses due to problems related to method variance between self-report (for eating pathology) and diagnostic interview data (for all other forms of psychopathology), as well as concerns with the measure's ability to capture clinically significant eating pathology.

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Model 1. Eating Pathology as a Latent Exogenous Factor **Model 2. Eating Pathology as an Indicator of Internalizing** **Model 3. Eating Pathology as an Indicator of Externalizing** **Model 4. Eating Pathology as an Indicator of Internalizing and Externalizing** **Figure 1.**

Structural Equation Models of Eating Pathology within the Diagnostic Taxonomy

Note. MD = Major Depression, GAD = Generalized Anxiety Disorder, SOP = Social Phobia, SPP = Simple Phobia, AN=Anorexia Nervosa, BS=Bulimic Syndromes, ASP = Antisocial Behavior, CD = Conduct Disorder, DRG= Drug Abuse/Dependence, ALC = Alcohol Abuse/Dependence.

Figure 2.

Completely Standardized Path Diagram of the Best-fitting Structural Equation Model of Eating Pathology within the Diagnostic Taxonomy

Note. MD = Major Depression, GAD = Generalized Anxiety Disorder, SOP = Social Phobia, SPP = Simple Phobia, AN=Anorexia Nervosa, BS=Bulimic Syndromes, ASP = Antisocial Behavior, CD = Conduct Disorder, DRG = Drug Abuse/Dependence, ALC = Alcohol Abuse/Dependence. In order for this model to be identified, the factor loadings of observed indicators of Distress (i.e., MD and GAD) were fixed to be equal to each other, and the observed indicators of Fear (i.e., SOP and SPP) were also fixed to be equal to each other. All parameter estimates were statistically significant (p 's were all $<.001$).

Table 1
Sample Sizes, Standard Deviation, and Rates of Lifetime Diagnoses

Indicator	Possible Diagnosis		Probable Diagnosis		Full Threshold/Definite Diagnosis		SD
	N	Prevalence	N	Prevalence	N	Prevalence	
Major Depression	66	4.6%	49	3.4%	193	13.5%	1.06
Generalized Anxiety Disorder	158	11%	79	5.5%	108	7.5%	.90
Social Phobia	76	5.8%	79	6.0%	73	5.5%	.82
Simple Phobia	67	5.1%	75	5.7%	86	6.5%	.86
Anorexia Nervosa	130	9.3%	45	3.2%	30	2.1%	.60
Bulimia Nervosa	12	0.8%	5	0.3%	8	0.6%	.27
Binge Eating Disorder	6	0.4%	5	0.3%	4	0.3%	.21
Antisocial Behavior	85	6.4%	35	2.6%	27	2.0%	.57
Conduct Disorder	N/A	N/A	78	5.4%	62	4.3%	.74
Drug Abuse/Dependence	1	.1%	10	0.7%	108	7.7%	.81
Alcohol Abuse/Dependence	2	.1%	32	2.3%	146	10.4%	.95

Note. Structural analyses were based on a categorical 0-3 coding of diagnostic data, representing whether the disorder was absent (0), possible (1), probable (2), and definite (3). Due to their low prevalence, bulimia nervosa and binge eating disorder were combined. Because it would have been based on only a single symptom, a “possible” diagnosis of conduct disorder was not allowed.

Table 2
Polychoric Correlations among Indicators of Psychopathology

Diagnosis	MD	GAD	SOP	SPP	AN	BS	ASP	CD	Drug
GAD	.33***								
SOP	.11*	.28***							
SPP	.13*	.40***	.42***						
AN	.17**	.14**	-.07	-.05					
BS	.37***	.33***	.17	.20*	.21***				
ASP	.39***	.14*	.14*	.16**	.09	.02			
CD	.24***	.13*	.18**	.17**	.05	.06	.61***		
DRG	.38***	.05	-.01	.03	.14*	.07	.78***	.51***	
ALC	.28***	.03	.04	.02	.17**	.07	.74***	.43***	.74***

Note. $N = 1,434$. $*p < .05$. $**p < .01$. $***p < .001$. Correlations above |.30| are in bold. Polychoric correlations were based on a categorical 0-3 coding of diagnostic data. MD = Major Depression, GAD = Generalized Anxiety Disorder, SOP = Social Phobia, AN = Anorexia Nervosa, BS = Bulimic Syndromes, ASP = Antisocial Behavior arising after age 15 pertinent to the diagnosis of antisocial personality disorder, CD = Conduct Disorder, DRG = Drug Abuse/Dependence, ALC = Alcohol Abuse/Dependence.

Table 3

Model Fit and Comparisons

Model	χ^2	df	p	CFI	TLI	RMSEA	BIC
1	96.87	22	<.001	0.939	0.931	0.049	-63.03
2	92.79	22	<.001	0.942	0.934	0.047	-67.11
3	128.36	23	<.001	0.914	0.907	0.057	-38.81
4	113.29	23	<.001	0.926	0.920	0.052	-53.88
5	106.34	23	<.001	0.932	0.926	0.050	-60.83
6	107.26	23	<.001	0.931	0.925	0.051	-59.91

Note. $N = 1,434$. See Figure 1 for Path Diagram representations of Models 1–4. Model 5: Eating pathology regressed on Internalizing, with bulimic syndromes loading simultaneously on Externalizing. Model 6: Eating pathology regressed on Internalizing, with anorexia nervosa loading simultaneously on Externalizing. CFI = comparative fit index, TLI = Tucker-Lewis fit index, RMSEA = root mean square error of approximation, BIC = Bayesian Information Criterion. For Models 1, 2, and 3 to be identified, the factor loadings of observed indicators of Distress (i.e., MD and GAD) were fixed to be equal to each other, and the observed indicators of Fear (i.e., SOP and SPP) were fixed to be equal to each other. Models 4, 5, and 6 were identified by fixing observed indicators of Distress, Fear, and Eating Pathology to equality. Degrees of freedom do not directly correspond to the number of estimated parameters. See the Mplus Technical Appendices at www.statmodel.com or the index of the Mplus User's Guide (Muthén & Muthén, 2007) for the formula used to calculate degrees of freedom when using robust weighted least squares procedures such as those employed here.