Categorical Versus Dimensional Models of Eating Disorders: An Examination of the Evidence

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ABSTRACT
Objective: Eating disorders have been conceptualized as discrete syndromes or categories and as dimensions that differ in degree among individuals. Until recently, researchers have not directly addressed which of these models, categorical versus dimensional, is most valid.

Methods: The primary objective of this review was to examine the evidence related to the validity of dimensional versus categorical models of eating disorders.

Results: Findings from a series of taxometric studies have suggested that a conceptual representation of eating disorders may involve a latent taxon, related to binge eating (and possibly purging), and one or more dimensions. These studies found that binge eating was identified as a factor that does not occur on a continuum with anorexia nervosa, restricting subtype. Restricting subtype anorexia is continuous with normalcy, however.

Discussion: These findings should be viewed as preliminary evidence that may have implications for the etiology, assessment, prevention, and treatment of eating disorders. © 2004 by Wiley Periodicals, Inc.

Keywords: classification; taxometric analysis; anorexia nervosa; bulimia nervosa; binge eating disorder

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Introduction

Both of the major classification systems for psychiatric disorders—the Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM-IV; American Psychiatric Association [APA], 1994) and the International Classification of Diseases, 10th revision (ICD-10; World Health Organization [WHO], 1992)—have selected categorical approaches to describe psychopathology. In contrast, many psychological theorists have often favored dimensional models of psychopathology (Adams, Luscher, & Bernat, 2001), and the controversy about the relative merits of categorical versus dimensional models has raged for many years. The field of eating disorders has not been sheltered from this controversy (Franko & Omori, 1999; Goldner, Srikanameswaran, Schroeder, Livesley, & Birmingham, 1999; Stice, Killen, Hayward, & Taylor, 1998; Stice, Ziemba, Margolis, & Flick, 1996; Widiger & Coker, 2003). The DSM-IV and ICD-10 classification systems have used a categorical approach that has changed the diagnostic criteria for eating disorders over the past 20 years. Other theorists have proposed dimensional models of eating disorders (e.g., Heatherton & Polivy, 1992; Nylander, 1971, Polivy & Norman, 1987). This dimensional approach has come to be known as the “continuum of eating disorders” (Hay & Fairburn, 1998; Stice & Agras, 1999). The current article reviews the research evidence that has purported to test these two conceptual models of eating disorders.

Conceptual Issues

Tests of categorical versus dimensional models of eating disorders have been plagued by a variety of conceptual problems. Categorical approaches have been troubled by the constant change of the criteria that are diagnostic of eating disorders (APA, 1980, 1987, 1994; corresponding ICD changes). Furthermore, the categories have also changed. For example, bulimia was added in 1980 to the 3rd ed. of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III; APA, 1980); in 1987, bulimia nervosa (BN) replaced bulimia in the 3rd Rev. ed. of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III-R; APA, 1987); and, in
1994, the subtypes for anorexia nervosa (AN) and BN were added to DSM-IV (APA, 1994). Furthermore, in DSM-III-R, it was possible for a patient to be diagnosed with AN and BN. With the new criteria of DSM-IV, such patients would be diagnosed as having the binge eating/purging subtype of AN (AN-BP).

Dimensional approaches have also been plagued by the same lack of clarity. For example, how many dimensions are required to adequately describe the eating disorders (or disturbed eating behavior)? What construct(s) define the dimension(s)? Also, if eating disorders can be conceptualized as falling upon one or more dimensions, what is the relative position of eating disorders within this dimensional scheme, for example, is AN a more extreme disturbance than BN?

One consequence of the lack of clarity concerning these conceptual issues is that there has been much discussion about the best definition of the different eating disorders, for example, binge eating disorder (BED) versus nonpurging BN (BN-NP; Hay & Fairburn, 1998) or the distinction between bulimia and BN (Williamson, Davis, & Ruggiero, 1987). Tests of the continuum of eating disorders have often made the assumption that there is only one dimension that underlies the spectrum of eating disorders (e.g., Tylka & Subich, 2003). When researchers considered multiple dimensions, they often viewed the secondary dimensions as personality variables, as opposed to eating disorder symptoms.

Over the past 15 years, a few theorists have proposed multidimensional models of eating disorders. For example, Schlundt and Johnson (1990) proposed a three-dimensional model of eating disorders that included binge eating, fear of fatness, and body size. Also, Beumont, Garner, and Touyz (1994) and Walsh and Garner (1997) proposed a slightly different set of dimensions that comprised binge eating, purging, and body size. Each of these multidimensional models proposed a relative position of the eating disorder categories (i.e., DSM-III, DSM-III-R, and DSM-IV) among the three dimensions. The validity of these multidimensional models of eating disorder was never empirically tested, until very recently (Williamson et al., 2002).

Earlier Tests of Categorical Versus Dimensional Models of Eating Disorders

Many studies have purported to test the validity of categorical models or the validity of dimensional models. Few studies have directly tested whether one model is more valid (fits empirical data) than the other. An example of this earlier research is a series of cluster analytic studies (Garner, Olmsted, & Garfinkel, 1983; Hay, Fairburn, & Doll, 1996; Stice & Agras, 1999; Westen & Harnden-Fischer, 2001; Williamson, Gleaves, & Savin, 1992). These investigations asked whether there were “clusters” of people with similar symptom profiles. Thus, these cluster analytic studies attempted to test the validity of the categorical approach. All of these studies found subgroups of eating disorders with different symptom profiles. However, this evidence does not compare the validity of categorical versus dimensional models because cluster analytic techniques do not directly test for the presence or absence of a dimension (continuum) versus the presence or absence of a category (taxon). A few studies in this series used latent class analysis as the data analytic approach (e.g., Bulik, Sullivan, & Kendler, 2000; Sullivan, Bulik, & Kendler, 1998; Sullivan & Kendler, 1998). However, this approach assumes the validity of the categorical approach rather than testing the dimensional vs. categorical structure of the data.

Another series of studies tested the validity of the dimensional model or continuum of eating disorders using multivariate approaches, e.g., discriminant analysis (e.g., Franko & Omori, 1999; Goldner et al., 1999; Lowe et al., 1996; Stice et al., 1996, 1998; Tylka & Subich, 1999). These studies tested how measures of eating disorder symptoms could be reduced to dimensions and how different patient groups and/or controls differed along this dimension. Invariably, these studies found evidence in support of a dimensional approach for classifying eating disorders. They did not, however, directly contrast the validity of categorical versus dimensional models of eating disorders.

By the end of the 20th century, there was considerable evidence in support of both conceptual models of eating disorders. Therefore, both sides could conclude victory and the argument about which approach was most valid could devolve into a matter of opinion. Yet, there were some valuable insights gleaned from this era. In particular, the issue of the possibility of multiple dimensions of eating disorders was addressed in a series of factor analytic studies.

Factor Analytic Studies of Eating Disorders

Factor analysis can be used to identify the latent structure of a dataset, specifically the number of factors that underlie the observed variables (e.g.,

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These factors (often referred to as dimensions) can be evaluated as being correlated or uncorrelated, depending on the factor solution that is hypothesized by theory. Also, the measures of eating disorder symptoms that are used in a study determine the range of factors that can be identified. Furthermore, the researcher must use some judgment about the determination of the best factor solution, so it is best to view findings from factor analysis in the context of a series of studies. Because of this subjectivity, it is best to conduct exploratory and confirmatory factor analyses to validate a factor solution. Some studies tested the validity of specific factor solutions using confirmatory factor analysis and other did not. Table 1 summarizes the results of eight factor analytic studies of eating disorder symptoms that were conducted over the past 15 years. These studies used different measures with very diverse samples and found a variety of factor solutions. However, given the extreme variation in methods, it is encouraging to find such agreement across the studies.

The strongest evidence indicates the presence of two factors, general psychopathology and binge eating (and purging), in seven of eight studies. A binge eating factor was confirmed in all four studies that employed confirmatory factor analysis, which is a more stringent test of factor structure. Also, restrictive eating and fear of fatness/body image concerns were the factors found in five of the eight studies. A factor related to drive for thinness was found in two of the eight studies. These factor analytic studies suggest that a multidimensional model of eating disorders is required to fit the data from a very diverse set of studies. These findings do not, however, answer the question of the nature of these latent factors, that is, whether they are dimensional versus discontinuous. That is, although factor analysis is often used to measure dimensional constructs, obtained factors may also be discrete (e.g., Gangestad & Snyder, 1984).

### An Alternative: Taxometric Analysis

Taxometric analysis specifically tests for discontinuity or the presence of a latent taxon in a dataset. Thus, taxometric analysis is uniquely designed to test whether a set of symptoms are best described as indicating a dimensional versus categorical construct (Meehl, 1992, 1995, 1999; Meehl & Golden, 1982; Waller & Meehl, 1998). There are several taxometric methods, including mean above minus below a sliding cut score (MAMBAC; Meehl & Yonce, 1994), maximum covariance analysis (MAXCOV; Meehl & Yonce, 1996), and L-mode factor analysis (Waller & Meehl, 1998). Taxometric studies of eating disorders have used these three methods to test for the presence of taxa (syndromes that are discontinuous from normalcy).

Four published studies have reported taxometric analyses of eating disorders (Gleaves, Lowe, Green, Cororve, & Williams, 2000; Gleaves, Lowe, Snow, Green, & Murphy-Eberenz, 2000; Tylka & Subich, 2003; Williamson et al., 2002). The studies differed in the patient populations that were studied and the measures of eating disorder symptoms, but were similar in the data analytic approaches that were used. Furthermore, these investigations included independent samples from different locations in the United States.

Table 2 summarizes the study samples of the recent taxometric investigations. Gleaves, Lowe, Snow, et al. (2000) studied a sample of women diagnosed with BN and a group of college women

### Table 1. Factor analytic studies of eating disorder symptoms

<table>
<thead>
<tr>
<th>Article (Authors/Date)</th>
<th>General Psychopathology</th>
<th>Binge Eating/Bulimic Behaviors</th>
<th>Restrictive Eating</th>
<th>Fear of Fatness/Body Image</th>
<th>DFT/Thin Body</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vanderheyden et al. (1988)</td>
<td>X</td>
<td>X</td>
<td></td>
<td>X</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>Tobin, Johnson, Steihberg, Staats, and Dennis (1991)</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gleaves et al. (1993)</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
<td></td>
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<tr>
<td>Gleaves and Eberenz (1993)</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
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<tr>
<td>Gleaves and Eberenz (1995)</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Varnado Williamson, and Netemeyer (1995)</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Price Foundation Collaborative Group (2001)</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Williamson et al. (2002)</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: DFT = Drive for Thinness.

* Used only exploratory factor analysis.
* Used only confirmatory factor analysis.
* Used exploratory factor analysis, followed by confirmatory factor analysis to test the validity of the factor solution based on exploratory factor analysis.
using items from the Bulimia Test-Revised (Thelen, Farmer, Wonderlich, & Smith, 1991) and the 26-item Eating Attitude Test (EAT-26; Garner & Garfinkel, 1979) as measures of BN symptoms. Gleaves, Lowe, Green, et al. (2000) analyzed the data from a sample of women diagnosed with a variety of eating disorders using items from the Eating Disorder Inventory (EDI; Garner, 1991) as indicators for eating disorder symptoms. Williamson et al. (2002) studied a broad range of eating disorders. Eating disorder symptoms were measured using an interview procedure called the Interview for Diagnosis of Eating Disorders, Version IV (IDED-IV; Kutlesic, Williamson, Gleaves, Barbin, & Murphy-Eberenz, 1998). Factor analysis (including exploratory and confirmatory factor analysis) of the DSM-IV eating disorder symptoms yielded three factors: binge eating, fear of fatness/compensatory behaviors, and drive for extreme thinness.

Table 3 summarizes the findings from these taxometric studies. The data analytic approach used in these investigations directly tested for the presence of one or more latent taxa versus a dimension when two or more diagnostic or control groups were included in the taxometric analysis. Table 3 is structured to illustrate the results of each of these analyses. If a taxometric analysis indicated the presence of a taxon, this finding can be interpreted as evidence in support of a categorical distinction within the dataset. If a dimensional finding was indicated, support for a dimension was concluded.

It should be noted that for most taxometric analyses, only one test has been reported. Therefore, the findings shown in Table 3 should be viewed with caution until they have been replicated. One finding was reported in all three studies that used eating disorder symptoms in the taxometric analysis—BN was best viewed as taxonic when analyzed with normal weight controls. Also, Williamson et al. (2002) and Gleaves, Lowe, Green, et al. (2000) both reported that AN was on a continuum with normalcy but was taxonic when analyzed with BN. Finally, as shown in Table 3, there are a number of taxometric analyses that have not been conducted. Within this context, the findings of these three studies suggest that disorders involving binge eating (BN, BED, AN-BP) are qualitatively different from AN, restricting subtype (AN-R), normal weight controls, and obese controls. In contrast, AN-R occurred on a continuum with both normalcy and obesity. Therefore, these taxometric findings have suggested that eating disorders can be conceptualized in terms of dimensions and at least one latent taxon involving binge eating (and possibly purging). For a more complete discussion of these findings, readers are directed to the original articles or a recent review by Gleaves, Brown, and Warren (in press).

As with any statistical technique, taxometrics have their limitations. First, as Meehl (1999) wrote, “No statistic is self-interpreting [original italics], and taxometrics is not different from any other psychometric

**TABLE 2. Subject demographics from taxometric studies**

<table>
<thead>
<tr>
<th>Article (Author/Date)</th>
<th>N</th>
<th>AN/R</th>
<th>AN/BP</th>
<th>BN/P</th>
<th>BN/NP</th>
<th>EDNOS</th>
<th>BED</th>
<th>Obese</th>
<th>Non-ED</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gleaves, Lowe, Snow, Green, and Murphy-Eberenz (2000)</td>
<td>613</td>
<td>201</td>
<td></td>
<td></td>
<td></td>
<td>412</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gleaves, Lowe, Green, Cororve, and Williams (2000)</td>
<td>959</td>
<td>70</td>
<td>95</td>
<td>514</td>
<td>48</td>
<td>18</td>
<td>214</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Williamson et al. (2002)</td>
<td>341</td>
<td>19</td>
<td>16</td>
<td>38</td>
<td>7</td>
<td>78</td>
<td>43</td>
<td>37</td>
<td>116</td>
</tr>
<tr>
<td>Tylka and Subich (2003)</td>
<td>652</td>
<td>2</td>
<td>23</td>
<td>40</td>
<td>47</td>
<td>647</td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

Note: AN/R = restricting subtype of anorexia nervosa; AN/BP = binge/purge subtype of anorexia nervosa; BN/P = purging subtype of anorexia nervosa; BN/NP = nonpurging bulimia nervosa; EDNOS = eating disorder not otherwise specified; BED = binge eating disorder; ED = eating disorder.

*In this study, participants with bulimia nervosa were not separated into subtypes.*

*In this study, the entire sample consisted of college students. Eating disorders were defined using the Questionnaire for Eating Disorders (Mintz, O’Halloran, Mulholland, & Schneider, 1997). Participants with anorexia and bulimia nervosa were not separated into subtypes.*

**TABLE 3. Summary of taxometric studies of eating disorders**

<table>
<thead>
<tr>
<th>Diagnostic Group</th>
<th>AN-BP</th>
<th>BN-P</th>
<th>BN-NP</th>
<th>BED</th>
<th>Normal</th>
<th>Obese</th>
</tr>
</thead>
<tbody>
<tr>
<td>AN-R</td>
<td>T</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AN-BP</td>
<td>D</td>
<td>D</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BN-P</td>
<td>D</td>
<td></td>
<td></td>
<td></td>
<td>T</td>
<td></td>
</tr>
<tr>
<td>BN-NP</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>T</td>
<td></td>
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<tr>
<td>BED</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>T</td>
<td></td>
</tr>
</tbody>
</table>

Note: T = taxonic; D = dimensional; ? = not tested; AN-R = restricting subtype of anorexia nervosa; AN-BP = binge/purge subtype of anorexia nervosa; BN-P = purging subtype of bulimia nervosa; BN-NP = nonpurging bulimia nervosa; BED = binge eating disorder.
MODELS OF EATING DISORDERS

Which Model is Most Valid?

Before attempting to answer this question, we would like to clarify a common area of confusion regarding the taxon/dimension question. To say that a construct is taxonic does not mean that it is purely dichotomous or that there is no variability within the taxon or nontaxon groups. Furthermore, saying that something is taxonic does not mean that there cannot also be one or more dimensions involved. As Waller and Meehl (1998) wrote:

Inferring that a latent structure is taxonic does not imply that there is no latent dimension involved. In most situations, the existence of taxonicty is an “additional” feature, something along with latent dimensions that in turn “underlie” the manifest dimensions that produce them. Thus, the convenient dichotomy taxonic-vs.-dimensional should, strictly speaking read “taxonic-dimensional vs. dimensional only.” (p. 9)

Indeed, regarding eating disorders research, the findings from the three taxometric studies suggest that both categorical and dimensional models of eating disorders are too simplistic to adequately represent eating disorder symptoms. Instead, these preliminary findings indicate that eating disorders that involve binge eating (and possibly purging) are discontinuous with normalcy, including obesity, whereas eating disorders that do not involve binge eating and normal controls may differ only in the degree of severity on one or more continua. The questions of the number and types of dimensions are very much unsettled, however. If the findings of Williamson et al. (2002) are used as a guide, then the eating disorders can be conceptualized in terms of a three-dimensional model. Using this model, binge eating is viewed as qualitatively different from normalcy and two true dimensions, fear of fatness/compensatory behaviors and extreme drive for thinness, are viewed as continua with normalcy. Figure 1 illustrates this model of eating disorders. The “floor” of this three-dimensional model is the two continua and the third dimension is binge eating that is discontinuous. It should be noted that in the study reported by Williamson et al (2002), the two continua, drive for thinness and fear of fatness/concern for body size and shape, were positively correlated. Therefore, it is possible that there is one latent dimension that accounts for these two continua. As can be seen in Figure 1, AN-R, obese, and normal groups are conceptualized as positioned along the two continua, whereas disorders involving binge eating, BN, purging subtype (BN-P), BN-NP, AN-BP, and BED are viewed as discontinuous with AN-R, obese, and normal groups.

Implications for Classification of Eating Disorders

The DSM and ICD classification systems are primarily designed to describe various psychiatric problems so that research and clinical practice can be served by a single set of descriptions of different psychopathologies. However, if these classification systems reflect current conceptual representations of diagnostic entities, then the findings of these taxometric studies have relevance for the next revisions of these classification systems. One approach that would be consistent with these findings would be to create three eating disorder categories: AN-R, BN, and binge eating syndrome (BES). In this scheme, a diagnosis of BN would be specific to binge eating combined with regular use of compensatory behaviors such as self-induced vomiting or misuse of laxatives, diuretics, or enemas. BN would have two types based on body weight: normal body weight, and low body weight. BES would refer to binge eating without regular use of compensatory behaviors other than fasting or excessive exercise. BES would have two subtypes based on the degree of concerns about body size/shape: (1) BES with extreme body concerns (similar to BN-NP) and (2) BES with low to moderate body concerns...
concerns (similar to BED). We note that this diagnostic scheme utilizes a framework that combines categorical and dimensional conceptualizations of eating disorders. In this scheme, traditional linear modeling techniques can be used to assess symptoms within a category.

**Implications for Eating Disorders Research**

Another reason why the issue of taxonicity versus dimensionality is important concerns how the disorders have been and continue to be studied. If a disorder is, indeed, determined to be taxonic, it will be important to consider the fact that persons with the disorder differ in kind (or, perhaps, in degree and kind) from persons with more normative eating and dieting concerns. What this means is that one cannot study the symptomatology of the disorder without actually studying persons that possess the taxon. Studies of subclinical eating disorders or normative forms of dieting, eating problems, or body/weight concerns may be investigating phenomena that are qualitatively different from (and, hence, cannot be generalized to) the phenomena of interest.

Furthermore, if one or more eating disorders are determined to be taxonic, it will also be important to consider that sample composition (i.e., purely nonclinical vs. purely clinical vs. mixed) will radically affect the results of research. Several examples of this effect can be seen in recent research. For example, there is a body of research linking bulimic behavior with dieting behavior. That is, bulimic behavior has been correlated with dieting and body concerns. However, the majority of these findings have been based on primarily nonclinical samples (which may contain participants with and without eating disorders). In two factor analytic studies conducted purely within samples of persons diagnosed as having BN (Gleaves & Eberenz, 1993, 1995), bulimic behavior was not found to be highly correlated with either dieting behaviors or body concerns. Although these findings were initially puzzling to some researchers, they are what one would expect (the indicators being uncorrelated within the taxon group) if bulimia were actually taxonic. This effect is especially clear when one also considers that Varnado, Williamson, and Netemeyer (1995) used a very similar factor analytic methodology and found that bulimic behavior was highly correlated with both restrictive eating ($r = .56$) and body image ($r = .24$) among a sample of college women. The authors noted that, of their 200 participants, 13 met the diagnostic criteria for an eating disorder. Thus, this study used a mixed sample, and the results were what would be expected if bulimia were taxonic. With a purely nonclinical (nontaxon) sample, one would not expect restrictive eating and body image to be correlated with binge eating. Indeed Lowe et al. (1996) found no relationship between dieting intensity and binge eating severity in a purely nonclinical sample. Thus, one point is that we may frequently find inflated correlations between various features of the disorder by examining mixed samples. The second point is that clarifying the taxonic versus dimensional latent structure of eating disorders will help make sense of these seemingly inconsistent data.

**Implications for Etiologic Understanding of Eating Disorders**

Etiologic models of the eating disorders (as well as any form of psychopathology) may also be potentially guided by the results of taxometric research. Identification of a taxonic structure does not necessarily suggest a genetic basis. However, the identification of a taxonic structure may at least give a genetic etiology a higher probability than if the disorder was determined to be nothing more than
an extreme point on one or more continua (Meehl, 1992). Thus, continuing the search for a genetic basis of binge eating (or bulimic behavior) may be more rationally supported than if it was found to be clearly dimensional. However, there may also be ways in which a taxonic structure is related to etiology other than through genetics. The concept of environmental mold taxa needs further investigation as does the possibility that the taxon may represent effects of different types of specific environmental events or behavioral threshold effects (e.g., Blundell & Hill, 1993).

Implications for Assessment

Measurement/assessment is also affected by the nature of the construct being measured. If a construct is continuous, the psychometric goal is to choose indicators that assess all aspects of the construct and discriminate all regions of the dimension (Meehl, 1992; Ruscio & Ruscio, 2002). When attempting to measure a categorical construct, the goal is to sort items at a best cut so as to minimize misclassifications (Meehl, 1992). Ruscio and Ruscio (2002) recently described a general approach by which an assessment strategy should be dependent on the latent structure of the construct being measured. Following this approach, current and future taxometric research may be used to guide the development of new eating disorder assessment strategies and the refinement of current approaches. For example, the findings of current taxometric studies suggest that it would be useful to measure concerns about body size/shape, drive for thinness, and fear of fatness using continuous variables that could operationally define these characteristics for the purposes of diagnosis and treatment planning. Variables such as weight status are routinely measured using continuous variables and this approach is recommended given current evidence. Variables such as the presence of significant binge eating and regular use of compensatory behaviors to control body weight may be measured on a continuous scale, but given current evidence (on discontinuity) by taxometric research, these variables might be defined discretely, that is, within normal limits versus pathologic limits.

Implications for Treatment

In the context of these taxometric findings, it is noteworthy that treatments for BN and BED have been reported to be considerably less intense and more efficacious than treatments for AN (Hay & Bacaltchuk, 2002; Treasure & Schmidt, 2002). Furthermore, efficacious pharmacologic therapy for BN and BED has been developed; whereas similar therapy for AN has not been developed despite considerable research (Treasure & Schmidt, 2002). If the eating disorders that involve binge eating are more easily treated, we must ask the question, “Why is this so”? Given current research evidence, the field probably does not have a definitive answer for this question. However, the results of taxometric research may have implications for understanding treatment effects.

For example, in his discussion of Type A behavior, Strube (1989) observed:

Once developed, changing the behaviors arising from a class variable should occur in a relatively all-or-nothing fashion. That is, partial or incomplete change is less likely than with a continuous variable. Furthermore, change may be difficult to initiate with a class variable, but once initiated should be more complete and dramatic. The reason for these differences, of course, is that a latent type of class variable really has only two “values”, whereas a latent continuum has multiple and intermediate levels. (p. 972).

To elaborate, from Strube’s perspective, one might argue that disorders that have a discrete “cause” (that may be psychologically or biologically determined) may be more easily treated in comparison to disorders that are extreme variants of normalcy. Perhaps, there is a genetic or biologic basis for binge eating (Blundell & Stubbs, 1998; Koopmans, 1998). Or, perhaps, binge eating is caused by a disruption of normal appetitive control mechanisms that may have both psychological and biologic determinants (Blundell & Hill, 1993). These speculations address why binge eating may be more easily treated, but it does not address why AN-R is so resistant to treatment. This question is especially perplexing when one considers that AN-R may be continuous with normalcy. Why would a syndrome that is an extreme level of normalcy be especially difficult to treat? Could it be that disorders that are acquired from a culture that overvalues thinness in certain subgroups (Strong, Williamson, Netemeyer, & Geer, 2000) are more difficult to treat than disorders that stem from disturbances of biologic or psychological regulatory mechanisms? The challenge of successfully treating AN-R may be analogous to the treatment of personality disorders. Personality disorders have been conceptualized as varying from normalcy in terms of extremes along various personality dimensions (Wideger & Shea, 1991). Treatment of personality
disorders is often more challenging than treatment of certain Axis I disorders such as major depression or panic disorder (Target, 1998).

Implications for Prevention

Much of the research on prevention of eating disorders (e.g., Baranowski & Hetherington, 2001; Varnado-Sullivan et al., 2001) is based on a continuum model of eating disorders (Nylander, 1971; Ruderman & Besbeas, 1992). The findings of these taxometric studies partially support the continuum model, but only with respect to AN-R. For eating disorders involving binge eating (which are actually much more common than anorexia), these findings suggest that there may be determinants of binge eating that are not psychological, environmental, or cultural variables (e.g., Koopmans, 1998). For many years, dieting (or the intent to diet) has been viewed as a highly probable psychological determinant of binge eating (Polivy & Herman, 1987). This perspective has gradually shifted to emphasize the possibility of more complex determinants of binge eating (Polivy & Herman, 1993). Recently, there have been reports that binge eating does not necessarily follow a period of intense dieting (e.g., Spurrell, Willfley, Tanofsky, & Brownell, 1997). Other research has indicated that some cases of binge eating are associated with escape from negative affect (e.g., Heatherton & Polivy, 1992). If the findings related to binge eating are replicated and the etiology of binge eating is better understood, prevention studies may need to develop different strategies for preventing eating disorders stemming from excessive concern with body size/shape with less emphasis on the hazards of dieting (e.g., Varnado-Sullivan et al., 2001). The findings of Tylka and Subich (2003) are consistent with this perspective. Unlike the other taxometric studies of eating disorder symptoms, they did not include measures of binge eating or purging. They found no evidence of a latent taxon using measures of body shape concerns, neuroticism, interoceptive awareness, internalization of a thin-ideal stereotype, and perceived social pressure for thinness. Prevention programs based on this conceptualization of the etiology of eating disorders might place less emphasis on the hazards of dieting for a population that is at considerable risk for the development of obesity (Seidell & Rissanen, 1998).

Most prevention efforts are based on the assumption that all people are affected equally by the risk factors for eating disorders. Most specifically, excessive concern with body size/shape and dieting is seen as the greater risk factor. When viewed simplistically, this view hypothesizes that anyone who diets intensely may be vulnerable to the development of an eating disorder. However, if the phenomenon detected by taxometric research involves a latent vulnerability to binge eating, there may be a subset of persons who are vulnerable to the negative effects of dieting. This finding is consistent with other studies that have not found dieting to be harmful in obese adults, overweight binge eaters, or healthy women (Goodrick, Poston, Kimball, Reeves, & Foreyt, 1998; Klem, Wing, Simkin-Silverman, & Kuller, 1997; Presnell & Stice, 2003; Reeves et al., 2001). Prevention efforts may eventually be able to identify a subgroup of persons that could be deemed to be at high risk for the development of binge eating. One implication of this conclusion is that prevention efforts for AN and BN (and BES) may not be identical in content or structure. For example, prevention of AN may emphasize myths about the pursuit of extreme thinness and prevention of binge eating may emphasize the importance of eating three healthy meals, healthy physical activity, and not skipping meals.

Conclusions

Research on the classification of eating disorders is only recently approaching maturity. Much has been written on the topic and many opinions have been expressed. Recent taxometric research suggests that earlier conceptual models of eating disorders may have been too simplistic. Neither categorical nor dimensional models of eating disorders adequately represent the full spectrum of disturbed eating. Results from these studies must be viewed as preliminary and should be replicated before strong conclusions are made. These preliminary findings suggest that binge eating may be qualitatively different from simple overeating and that AN-R may occur on one or more continua with normalcy. If these conclusions are valid, there are clear implications for understanding the etiology of eating disorders. Hopefully, these advances in basic knowledge will facilitate more effective prevention and treatment strategies for disturbed patterns of eating.

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