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Alternative Methods of Classifying Eating Disorders: Models Incorporating Comorbid Psychopathology and Associated Features

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Abstract

There is increasing recognition of the limitations of current approaches to psychiatric classification. Nowhere is this more apparent than in the eating disorders (EDs). Several alternative methods of classifying EDs have been proposed, which can be divided into two major groups: 1) those that have classified individuals on the basis of disordered eating symptoms; and, 2) those that have classified individuals on the basis of comorbid psychopathology and associated features. Several reviews have addressed symptom-based approaches to ED classification, but we are aware of no paper that has critically examined comorbidity-based systems. Thus, in this paper, we review models of classifying EDs that incorporate information about comorbid psychopathology and associated features. Early approaches are described first, followed by more recent scholarly contributions to comorbidity-based ED classification. Importantly, several areas of overlap among the classification schemes are identified that may have implications for future research. In particular, we note similarities between early models and newer studies in the salience of impulsivity, compulsivity, distress, and inhibition versus risk taking. Finally, we close with directions for future work, with an emphasis on neurobiologically-informed research to elucidate basic behavioral and neuropsychological correlates of comorbidity-based ED classes, as well as implications for treatment.

Keywords

eating disorders; classification; nosology; subtype; comorbidity

There is growing recognition of the limitations of current categorical approaches to psychiatric classification. Although the two leading nosological systems, the Diagnostic and Statistical Manual of Mental Disorders, 4th ed. (*DSM-IV*; American Psychiatric Association, 1994) and the International Classification of Diseases, 10th ed. (ICD-10; World Health

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Organization, 2010), have many strengths, they also have several serious shortcomings including: 1) heterogeneity in symptom presentation within diagnostic categories; 2) high rates of co-occurrence between putatively distinct diagnoses; 3) lack of agreement between diagnostic categories and findings from clinical neuroscience; and 4) a failure to predict treatment response (Insel et al., 2010; Krueger, Watson, & Barlow, 2005; Widiger & Samuel, 2005).

Nowhere are the limitations of current categorical models of psychiatric classification more apparent than in the eating disorders (EDs). Current approaches to ED classification are based entirely on distinctions among individuals with respect to eating and weight-control behaviors and associated features. For example, individuals with extremely low body weight and cognitive distortions related to shape or weight are diagnosed with anorexia nervosa (AN), regardless of the presence or absence of other disordered eating symptoms (e.g., binge eating, self-induced vomiting, laxative misuse). Conversely, individuals who are normal-weight or overweight may be diagnosed with bulimia nervosa (BN), binge eating disorder (BED), or a variety of EDs not otherwise specified (EDNOS) depending on the frequency, duration, and specific constellation of ED symptoms. Although existing models of ED classification have some advantages (Keel, Brown, Holland, & Bodell, 2012), they also have significant limitations including reliance on post hoc analyses to validate categories derived from clinical consensus, lack of diagnostic stability, and in the *DSM-IV*, high rates of EDNOS diagnoses (Keel et al., 2012; Wonderlich, Joiner, Keel, Williamson, & Crosby, 2007). Moreover, current psychiatric nosologies provide no insight into potential mechanisms that may drive disordered eating, which limits their ability to inform models of etiology and maintenance, and hinders the development of interventions to target risk and maintaining factors for EDs (Insel et al., 2010).

Given the limitations of current approaches to ED classification, a number of alternative models have been described, which can be divided into two major groups: 1) those that have classified individuals on the basis of ED symptoms; and 2) those that have classified individuals on the basis of comorbid psychopathology and associated features. Alternative symptom-based approaches to ED classification have been reviewed extensively (see, e.g., Keel et al., 2012; Wonderlich, Joiner, et al., 2007; Striegel-Moore, Wonderlich, Walsh, & Mitchell, 2011), and thus will not be a focus of the current manuscript.

In contrast, we are aware of no paper that has provided a comprehensive review of studies examining comorbidity-based approaches to ED classification. Several methods of classifying EDs that incorporate comorbid psychopathology or associated features have been described, and there is a burgeoning literature documenting the validity of these models relative to existing schemes (see, e.g., Holliday, Landau, Collier, & Treasure, 2005; Steiger et al., 2009; Stice, Bohon, Marti, & Fischer, 2008; Wildes et al., 2011). Comorbidity-based approaches to ED classification offer an intriguing alternative to models that focus exclusively on ED symptoms, because heterogeneity in patterns of comorbid psychopathology among individuals with EDs might reflect different pathways to the expression or maintenance of aberrant eating (Westen & Harnden-Fischer, 2001). Furthermore, by focusing on psychopathological dimensions that may be more stable than disordered eating symptoms and have been shown to systematically differentiate ED subgroups, the EDs field could capitalize on work from other areas that has examined behavioral or biological processes that underlie the expression of these traits.

Thus, the overall aim of the current manuscript is to provide a critical review of the literature on comorbidity-based approaches to ED classification. To this end, we searched online databases (e.g., MEDLINE, PsycINFO) using the terms *eating disorder, anorexia nervosa*, *bulimia*, *binge eat**, and *purg** coupled with *comorbid**, *classification*, *classify*, *nosology*,

nosological, subgroup, *subtype*, *cluster, latent, anxiety*, *anxious*, *autis**, *avoid**, *fear*, *inhibit**, *rigid*, obsess**, *compuls**, *impulsiv**, *borderline*, *depress**, *neurocog**, *neuropsy**, *and reward*. We also scanned the reference lists from articles and chapters for additional papers. In the sections that follow, we describe the results of our review.

Early Comorbidity-Based Approaches to Classifying EDs

Interest in the potential utility of classifying individuals with EDs on the basis of comorbid psychopathology and associated features is not new. Indeed, clinicians and researchers long have noted that ED patients presenting with particular patterns of comorbid psychopathology differ from their non-comorbid peers on a variety of clinically relevant measures (e.g., trauma history, treatment response). Although some of the "early" comorbidity-based approaches to ED classification no longer are a focus of active research, these models are important because they provide clues about aspects of comorbid psychopathology that may differentiate meaningful subgroups of the ED population, and set the stage for current work incorporating comorbid psychopathology and associated features into the classification of EDs.

Multi-Impulsive Versus Uni-Impulsive EDs

One of the earliest proposals for incorporating comorbid psychopathology into the classification of EDs emphasized the distinction between "multi-impulsive" and "uni-impulsive" (p. 641) forms of bulimia (Lacey & Evans, 1986). In a series of articles, Lacey and colleagues (Lacey, 1993; Lacey & Evans, 1986; Lacey & Moureli, 1986; Lacey & Read, 1993) argued that individuals who present with multiple forms of impulsive psychopathology comprise a distinct subgroup of the psychiatric population characterized by a common underlying mechanism and a poor response to treatment. Although multi-impulsivity was not conceptualized as being specific to EDs, operational criteria for a "multi-impulsive form of bulimia" (p. 644) were described, and this construct has generated considerable interest in the EDs field.

The initial criteria for multi-impulsive bulimia outlined by Lacey and Evans (1986) included: 1) bulimia accompanied by at least one additional impulsive behavior; i.e., "gross alcohol abuse, 'street drug' abuse, multiple overdoses, repeated self-damage, sexual disinhibition, [or] shoplifting" (p. 644); 2) a "sense of being out of control" (p. 644) during impulsive behaviors; 3) impulsive behaviors have a fluctuating course and are interchangeable (e.g., if binge eating and purging subside, alcohol abuse or self-injury may increase); and 4) decreases in impulsive behavior are associated with increases in depression and anger. Several studies have examined the validity of this multi-impulsive subtype, with mixed results (Fahy & Eisler, 1993; Newton, Freeman, & Munro, 1993; Welch & Fairburn, 1996). For example, Fahy and Eisler (1993) reported that 51% (n = 20) of a sample of 39 patients with bulimia, and 28% (n = 7) of a sample of 25 patients with anorexia, reported at least one impulsive behavior in addition to aberrant eating. Individuals with multi-impulsive bulimia endorsed more ED symptoms at presentation and more episodes of binge eating at the end of treatment than individuals with uni-impulsive bulimia, but these differences were not significant at post-treatment follow-ups. Welch and Fairburn (1996) documented similarly high rates of multi-impulsivity in a community sample of 102 women with BN relative to 204 healthy controls and 102 women with other psychiatric disorders (30% versus 8% and 9%, respectively). However, given the variability in the number (1, 2, or 3) and types (i.e., alcohol misuse, drug misuse, or deliberate self-harm) of impulsive behaviors endorsed, the authors concluded that multi-impulsive BN is too "heterogeneous" (p. 458) to be a diagnostic subgroup. Finally, in a consecutive series of 112 treatment-seeking women with bulimia, Lacey (1993) found that 39% (n = 44) reported at least one non-eating-related impulsive behavior. No data regarding the correlates of this multi-impulsivity were

provided, but a subgroup of 15 women who engaged in three or more non-eating-related impulsive behaviors was described as being older, less likely to be married or in a stable relationship, and more likely to have a family history of alcohol abuse and a personal history of sexual abuse than the rest of the sample.

We identified five additional studies that have examined the correlates of multi-impulsive EDs using a threshold of three or more non-eating related impulsive behaviors to distinguish the multi-impulsive subtype (Fichter, Quadflieg, & Rief, 1994; Matsunaga et al., 2000; Myers et al., 2006; Nagata, Kawarada, Kiriike, & Iketani, 2000; Wiederman & Pryor, 1996). In the first of these reports, Fichter, et al. (1994) compared 32 consecutively admitted inpatients with multi-impulsive BN (defined as the presence of three or more of the following impulsive behaviors in addition to binge eating: suicide attempt, self-injury, stealing, severe alcohol abuse, promiscuity, or severe drug abuse) to 32 age-matched BN patients without additional impulsive features (i.e., uni-impulsive BN) on indices of illness severity and treatment response. Although there were few differences between the multi-impulsive and uni-impulsive BN groups at baseline and no difference in length of treatment, the two groups diverged considerably with respect to outcomes at discharge and over two-year follow-up. Specifically, multi-impulsive BN patients endorsed higher levels of anxiety, depression, anger, and hostility at discharge and over two-year follow-up; they also showed more severe eating pathology beginning in the fourth month of follow-up and a greater number of hospitalizations and medical visits during the two years after discharge.

Cross-sectional studies provide further support for the notion that a history of three or more impulsive behaviors distinguishes a clinically relevant class of EDs. Indeed, this work has shown that relative to individuals with no history of multi-impulsivity, individuals with EDs who report three or more lifetime impulsive behaviors have more severe depressive and anxiety symptoms (Matsunaga et al., 2000), higher rates of anxiety disorder comorbidity (Myers et al., 2006), an earlier age at onset of binge eating and sexual intercourse (Wiederman & Pryor, 1996), higher rates of childhood abuse (Corstorphine, Waller, Lawson, & Ganis, 2007; Myers et al., 2006; but see, Nagata et al., 2000), and a greater prevalence of borderline personality disorder (Matsunaga et al., 2000; Nagata et al., 2000). Furthermore, one report found that impulsive behaviors (viz., suicide attempts and self-injury) preceded the onset of disordered eating symptoms in individuals with multi-impulsive BN (Nagata et al., 2000). Although much of this work has been conducted in individuals with bulimic syndromes (see, e.g., Corstorphine et al., 2007; Matsunaga et al., 2000; Myers et al., 2006; Wiederman & Pryor, 1996), there is evidence that multi-impulsivity occurs in individuals with AN, including those with the restricting subtype (Favaro et al., 2005; Nagata et al., 2000). Multi-impulsive EDs also are present in non-Western cultures (Matsunaga et al., 2000; Nagata et al., 2000; Tseng & Hu, 2012).

Finally, two studies have identified multi-impulsive ED subtypes using empirical methodology (Myers et al., 2006; Tseng & Hu, 2012). Myers et al. employed latent class analysis (LCA) to characterize subtypes in 125 women with BN based on a lifetime history of impulsive behaviors (i.e., severe alcohol abuse or dependence, other drug abuse, sexual promiscuity, stealing, self-mutilation, and suicide gestures). A two-class solution was identified, consisting of multi-impulsive ($n = 61$) and non-multi-impulsive ($n = 77$) groups. Notably, 84% ($n = 51$) of the women assigned empirically to the multi-impulsive class reported three or more non-eating related impulsive behaviors, consistent with clinical descriptions of multi-impulsive BN (n.b., the remaining 10 participants in the multi-impulsive class reported two non-eating-related impulsive behaviors). Tseng and Hu (2012) reported similar findings in a study of 180 women with BN in which LCA was used to classify participants on the basis of lifetime ED symptoms, number of purging methods (i.e., self-induced vomiting, laxative misuse, diuretic/diet pill misuse), and lifetime impulsive

behaviors (viz., alcohol abuse, drug abuse, suicide attempts, self-harm, stealing, sexual promiscuity, and excessive buying). Three ED classes were identified: "impulsive multi-purgers," (n = 32), "non-impulsive multi-purgers" (n = 75), and "non-purgers/uni-purgers" (n = 75; p. 67). Of note, 85% of the impulsive multi-purger class reported a history of three or more non-eating related impulsive behaviors (Tseng & Hu, 2012).

In summary, a large body of research has focused on the identification and validation of a multi-impulsive class of EDs characterized by forms of comorbid psychopathology hypothesized to be driven by deficits in impulse control (Lacey & Evans, 1986). In general, this work has provided support for the notion that individuals with multi-impulsive EDs differ from their non-multi-impulsive counterparts on a variety of clinically relevant measures. Nevertheless, several limitations of the multi-impulsive construct warrant consideration when evaluating its utility.

First, definitions of multi-impulsivity have varied greatly in terms of the number and type of non-eating-related impulsive behaviors required for assignment to the multi-impulsive class, as well as the time frame (lifetime versus current) used to determine class membership. Consequently, data regarding the correlates of multi-impulsive EDs have been inconsistent, particularly with respect to treatment effects (see, e.g., Fahy & Eisler, 1993; Fichter, et al., 1994). Although recent empirical studies suggest that a cutoff of three or more lifetime impulsive behaviors may identify a valid multi-impulsive BN subtype (Myers et al., 2006; Tseng & Hu, 2012), this work requires replication. Second, the preponderance of research on multi-impulsivity has focused on treatment-seeking samples with BN (for notable exceptions see, Favaro et al., 2005; Nagata et al., 2000; Welch & Fairburn, 1996). Thus, it is unclear to what extent this approach has validity for classifying individuals across a broad range of EDs, particularly in non-clinical settings. Moreover, many reports had small sample sizes (see, e.g., Fahy & Eisler, 1993; Fichter et al., 1994; Matsunaga et al., 2000; Newton, et al., 1993), which may limit the stability of their findings. Third, no study to our knowledge has compared the validity of multi-impulsive and non-multi-impulsive ED classes to an existing classification scheme (e.g., the *DSM-IV* ED categories). The extent to which multi-impulsivity provides useful information over and above comorbid diagnoses (e.g., Cluster B personality disorders, bipolar disorder) also is unknown. Finally, an approach to classifying EDs based on multi-impulsivity provides no information about other forms of psychopathology (e.g., mood disturbance, anxiety symptoms, obsessive-compulsive personality traits) that might help to identify meaningful subgroups. Thus, we conclude that although impulsive behaviors or multi-impulsivity might constitute one aspect of a comorbidity-based approach to ED classification, variations in impulsivity are not sufficient for characterizing the full range of comorbid psychopathology in EDs, or for helping to guide hypotheses about potential mechanisms that may underlie systematic heterogeneity in the expression of aberrant eating.

ED Classes Defined By the Presence or Absence of Borderline Personality Disorder Traits

Another early comorbidity-based approach to classifying EDs emphasized differences between individuals with and without borderline personality disorder (BPD) traits. Because BPD is characterized, in part, by impulsivity (Friedel, 2004), this classification scheme overlaps with distinctions between multi-impulsive and uni-impulsive EDs. However, unlike the multi-impulsive construct which focuses entirely on impulsive behaviors, the borderline syndrome includes impairments in emotion regulation, cognitive-perceptual domains, and interpersonal relationships (Friedel, 2004). Thus, a classification scheme based on BPD traits incorporates a broader range of comorbid psychopathology than the multi-impulsive ED scheme.

Two initial reports documented significantly greater psychiatric and emotional distress, more impulsive behaviors (viz., suicide attempts, self-injury, and substance abuse), poorer social functioning, greater psychosocial stress, more negative family environments, and higher rates of sexual abuse in ED patients with BPD symptoms relative those without BPD features (Johnson, Tobin, & Enright, 1989; Wonderlich & Swift, 1990). Moreover, one study found that borderline ED patients were twice as likely as their nonborderline peers to have a history of psychiatric hospitalization (Johnson, et al., 1989), despite no differences between the borderline and nonborderline groups in severity, duration, or age of onset of ED symptoms.

However, some scholars have criticized work comparing borderline and nonborderline EDs on the grounds that BPD symptoms might be a sequlea of eating pathology or a proxy for general mood disturbance, rather than an indicator of a distinct ED subtype (see, e.g., Garner et al., 1990; Vitousek & Manke, 1994). For example, Steiger, Thibaudeau, Ghadirian, and Houle (1992) compared 31 BN patients with BPD to 60 BN patients with other PDs ($n = 39$) or no PD ($n = 21$) on indices of psychiatric morbidity and personality functioning. Although univariate analyses documented differences among the groups on several measures, these effects largely disappeared after controlling for severity of depressive symptoms. In contrast, another study found differences between borderline and nonborderline BN groups on severity of depressive symptoms and psychopathological personality traits before and after intervention, controlling for the effects of ED symptoms (Steiger, Leung, Thibaudeau, Houle, & Ghadirian, 1993). These findings suggest that effects of BPD traits on the expression of psychiatric symptoms cannot be explained fully by ED pathology; however, distinctions between borderline and nonborderline BN groups may be driven by general mood disturbance rather than specific BPD traits.

Another source of debate in the literature on BPD and EDs concerns the evidence, or lack thereof, that “borderline” individuals comprise a distinct subgroup of the ED population. For example, Steiger et al. (1992) noted that most of the differences between the borderline and nonborderline groups in their initial study reflected variations in the severity of psychopathology rather than a distinct profile associated with the borderline BN subtype. Yet, other research has found differences between borderline and nonborderline ED patients on family history variables, which may suggest distinct pathways to the onset or maintenance of disordered eating. For example, two studies reported positive associations between BPD and childhood abuse in individuals with EDs (Steiger, Jabalpurwala, & Champagne, 1996; Waller, 1993). Furthermore, Waller (1994) found that women with AN or BN plus BPD ($n = 26$) reported significantly poorer family problem-solving, affective responding, and general functioning than ED patients without BPD ($n = 75$). Notably, follow-up analyses indicated that perceived family functioning was related to specific BPD symptoms (viz., unstable relationships, impulsivity, unstable affect, and identity disturbance), and that other aspects of borderline personality psychopathology (e.g., poor anger control, self-harm/suicidal behavior) had little salience for the prediction of perceived family functioning in individuals with EDs.

Finally, several studies have evaluated the impact of BPD symptoms on the treatment of EDs, with mixed results. Although one report found that borderline BN patients showed less improvement in ED symptoms after one year of treatment than nonborderline patients (Johnson, Tobin, & Dennis, 1990), other investigations have been unable to replicate this finding (e.g., Steiger & Stotland, 1996; Steiger, Thibaudeau, Leung, Houle, & Ghadirian, 1994). Still, there is some evidence that BPD comorbidity is associated with higher levels of general psychiatric disturbance following ED treatment (Steiger & Stotland, 1996; Steiger, Thibaudeau, et al., 1994), and certain aspects of borderline personality psychopathology may predict a more protracted course of disordered eating (Steiger, Leung, Thibaudeau, &

Houle, 1993; Steiger, Stotland, & Houle, 1994). For example, Steiger, Leung, Thibaudeau, and Houle (1993) found that having BPD, compared to another PD or no PD, predicted less improvement in ED symptoms after three months of outpatient treatment in 61 individuals with bulimic syndromes. Of note, follow-up analyses suggested that this effect might have been mediated by depressive symptoms and disturbances in "object-relations" (i.e., "the ability to achieve stable or satisfying relationships," p. 190), leading Steiger et al. (1993) to conclude that these "specific subcomponents of borderline personality pathology" (p. 187) might predict a poorer response to ED treatment.

In summary, the evidence in support of classifying individuals with EDs on the basis of BPD symptoms is equivocal. Although some studies have found differences between borderline and nonborderline ED groups on indices of clinical severity, family history, and treatment response, others have not, and there is no validator on which these groups have been shown to differ consistently. The literature comparing borderline and nonborderline ED presentations also suffers from many of the same limitations as the literature on multi-impulsive EDs, namely, small sample sizes and a reliance on data from specialized ED programs, a focus on bulimic symptoms, and an absence of information about the validity of a BPD-based system of classifying EDs relative to other classification schemes and comorbid diagnoses. Furthermore, several of the studies focusing on the relation between BPD and EDs have significant methodological limitations (e.g., a failure to account for missing data, a focus only on treatment completers) that make it difficult to draw conclusions from their findings. Perhaps for these reasons, the borderline-nonborderline distinction no longer is a focus of research in the EDs field. Still, this work does offer some insight into aspects of comorbid psychopathology that might inform future work on ED classification. Of particular relevance are data that suggest that dimensional constructs associated with BPD, such as impulsivity, mood disturbance and interpersonal difficulties, may have more salience for predicting clinically-relevant outcomes than categorical PD diagnoses (Steiger, Leung, Thibaudeau, & Houle, 1993; Waller, 1994).

ED Classes Defined by the Presence or Absence of Obsessive-Compulsive Disorder

A small number of studies have examined the utility of grouping individuals with EDs on the basis of obsessive-compulsive disorder (OCD) comorbidity. Unlike the multi-impulsive and borderline classification schemes, which focus on aspects of psychopathology that have been linked most strongly to bulimic symptoms (Cassin & von Ranson, 2005), OCD comorbidity is associated with the full range of ED diagnoses (Altman & Shankman, 2009). Thus, this approach could be useful in classifying a broader spectrum of ED presentations.

Yet, data supporting the validity of an OCD-based classification scheme are sparse. Although cross-sectional studies generally have shown that individuals with AN or BN and OCD score higher than ED patients without OCD on self-report measures of ED psychopathology (Albert, Venturello, Maina, Ravizza, & Bogetto, 2001; Matsunaga et al., 1999; Thiel, Broocks, Ohlmeier, Jacoby, & Schussler, 1995), research examining other correlates of OCD comorbidity has produced mixed findings. Some studies have reported positive associations of OCD comorbidity with age and duration of illness (Matsunaga et al., 1999; Milos, Spindler, Ruggiero, Klaghofer, & Schnyder, 2002), while others have failed to find these relationships (Albert et al., 2001). Similarly, work comparing ED patients with and without OCD on treatment-related variables has produced diverse results, with some studies finding no effect of OCD comorbidity on prognosis after treatment discharge (e.g., Cumella, Kally, & Wall, 2007; Thiel, Zuger, Jacoby, & Schussler, 1998), and another reporting that OCD symptoms were positively related to length of stay in residential treatment (Weltzin et al., 2007).

With notable exceptions (Cumella et al., 2007; Weltzin et al., 2007), current interest in an OCD-based model of ED classification is limited. Still, research in this area is worth mentioning because a growing body of literature has documented the association of childhood OCD symptoms and obsessive-compulsive personality traits with AN (Anderluh, Tchanturia, Rabe-Hesketh, & Treasure, 2003; Micali et al., 2011), and there is evidence that these features are associated with a poorer prognosis following treatment (Crane, Roberts, & Treasure, 2007). Furthermore, as detailed below, OCD symptoms and obsessive-compulsive personality traits feature prominently in some newer models of comorbidity-based ED classification, particularly those that have grouped individuals on the basis of multiple forms of comorbid psychopathology.

More Recent Models

The following sections describe models of ED classification that have generated at least five empirical papers since 2007, or have been proposed in the last five years. Collectively, these recent studies document ongoing scholarly interest in utilizing comorbid psychopathology or associated features to classify EDs.

Dietary versus Dietary-Negative Affect EDs

One recent model that has received particular attention focuses on the delineation of dietary and dietary-negative affect (also termed dietary-depressive) ED subtypes. This approach derives from the "dual-pathway model" of BN (Stice, 2001, p. 124), which postulates that dietary restraint and negative affect may play independent roles in the expression of bulimic symptoms, or may combine to produce aberrant eating. Accordingly, there may be subtypes of ED characterized primarily by dietary restraint, negative affect, or both. Stice and Agras (1999) tested this hypothesis in 265 females with BN. Using cluster analysis, they identified two subtypes: 1) a dietary subtype (n = 160; 62%) characterized by high levels of dietary restraint and minimal negative affect, and 2) a dietary-depressive subtype (n = 101; 38%) characterized by high levels of dietary restraint and depressive symptoms and low self-esteem. Although the two groups demonstrated few differences in the severity of bulimic behaviors, individuals with dietary-depressive BN endorsed significantly more eating, weight, and shape concerns, higher levels of eating and weight preoccupation and rituals, greater social maladjustment, and higher rates of Axis I and II comorbidity than individuals with dietary BN. Moreover, analyses conducted in a subgroup of participants (n = 189) found that individuals with dietary-depressive BN had lower rates of abstinence from binge eating and purging at the end of treatment compared to individuals with dietary BN, despite no difference in rate of treatment dropout.

We identified four other studies that have used cluster analysis to document dietary and dietary-negative affect classes in individuals with BN or subthreshold bulimic syndromes (Chen & Le Grange, 2007; Grilo, Masheb, & Berman, 2001; Stice et al., 2008; Stice & Fairburn, 2003). In general, these reports have replicated the findings of Stice and Agras (1999), namely, that the dietary-negative affect subtype comprises approximately one-third of individuals with BN, and is associated with more severe ED symptoms, greater emotional distress, higher rates of comorbid psychopathology, and greater psychosocial impairment than the dietary BN subtype. This work also has extended the literature on dietary and dietary-negative affect subtypes by documenting that these classes have more concurrent and predictive validity than the purging and nonpurging BN subtypes included in the *DSM-IV*, and are stable over short-term follow-up. For example, using three different samples, Stice and colleagues (Stice et al., 2008; Stice & Fairburn, 2003) found that the dietary and dietary-negative affect classes differed on significantly more indices of clinical severity than did purging and nonpurging BN classes. The dietary and dietary-negative affect subtyping scheme also was superior to the purging and nonpurging subtypes in predicting persistence

of binge eating and comorbid psychopathology over five-year follow-up in one sample (Stice & Fairburn, 2003), and recovery from bulimic symptoms at three-year follow-up in another (Stice et al., 2008; Study 2). In both groups, the dietary-negative affect class had a poorer outcome than the dietary class, but the purging and nonpurging subtypes did not differ. It is important to note, however, that results from a third sample showed no difference between the dietary/dietary-negative affect and purging/nonpurging subtyping schemes in the prediction of recovery from bulimic symptoms (Stice et al., 2008; Study 1). Finally, Stice et al. (2008) found that the dietary and dietary-negative affect classes were more stable over a four-week period than the purging and nonpurging BN subtypes.

In addition to research focusing on individuals with bulimic syndromes, several studies have identified dietary and dietary-negative affect classes in individuals with BED (Carrard, Crepin, Ceschi, Golay, & Van der Linden, 2012; Grilo, Masheb, & Wilson, 2001; Masheb & Grilo, 2008; Stice et al., 2001). The rationale for extending the dietary/dietary-negative affect subtyping scheme to individuals with BED derives from work documenting the roles of dietary restraint and negative affect in the onset and maintenance of binge eating (Grilo, Masheb, & Wilson, 2001; Stice et al., 2001). Collectively, studies that have examined dietary and dietary-negative affect classes in BED have produced findings that are similar to results reported in individuals with bulimic syndromes. In particular, available data suggest that the dietary-negative affect subtype comprises approximately 28%-37% of individuals with BED, and is associated with more severe ED symptoms and other psychopathology, higher rates of psychiatric comorbidity, and a poorer response to treatment than the dietary subtype (Carrard et al., 2012; Grilo, Masheb, & Wilson, 2001; Masheb & Grilo, 2008; Stice et al., 2001).

BED research also has shown that the dietary and dietary-negative affect subtypes are superior to other subtyping schemes and comorbid diagnoses in predicting clinically relevant outcomes. For example, Grilo, Masheb, and Wilson (2001) evaluated the validity of the dietary and dietary-negative affect classes relative to subtypes based on current and lifetime major depressive disorder (MDD) diagnoses and rates of binge eating in 101 women with BED. The dietary and dietary-negative affect classes differed on eight indices of clinical severity compared to three differences between the MDD and no MDD classes, and one difference between the high and low frequency binge eating classes. Similarly, in a sample of 75 individuals with BED, Masheb and Grilo (2008) found that the dietary/dietary-negative affect classification scheme was superior to subtypes based on overvaluation of shape or weight in predicting severity of binge eating following guided self-help treatments for BED. However, overvaluation-based subtypes were better than dietary and dietary-negative affect classes at predicting severity of cognitive ED symptoms at the end of treatment (Masheb & Grilo, 2008). Finally, consistent with research in individuals with BN (Stice et al., 2008), there is evidence that dietary and dietary-negative affect BED classes are stable over short-term follow-up (Grilo, Masheb, & Wilson, 2001).

Because the theoretical basis for the dietary and dietary-negative affect subtypes originates from research on the etiology and maintenance of BN, one might question whether this classification scheme can be applied across the ED spectrum, particularly in individuals with no history of binge eating. Three studies have addressed this issue in children and adults with various forms of eating pathology, with mixed results. For example, Grilo (2004) used cluster analysis to classify 137 adolescent inpatients with ED “features” (defined as a score > 40 on a self-report questionnaire, p. 67) based on measures of dietary restraint, depressive symptoms, and self-esteem. Results documented the presence of two subtypes, dietary (n = 78; 57%) and dietary-negative affect (n = 59; 43%), that differed on cognitive ED symptoms, pathological personality traits, suicidal tendencies, and childhood abuse history (all of which were more pronounced in the dietary-negative affect class). Furthermore, the

dietary and dietary-negative affect classes demonstrated better concurrent validity than a subtyping scheme defined by the presence or absence of self-induced vomiting (Grilo, 2004). Similarly, Goldschmidt et al. (2008) used cluster analysis to identify dietary and dietary-negative affect subtypes in 159 children and adolescents with loss of control over eating (LOC), a hypothesized marker of binge eating characterized by the feeling that one cannot control what, when, or how much one is eating. Consistent with previous work, children with LOC were classified empirically into dietary (n = 114; 72%) and dietary-negative affect (n = 45; 28%) subtypes. Moreover, children assigned to the dietary-negative affect subtype endorsed more shape and weight concerns and higher rates of objective binge eating, and had greater levels of parent-reported psychopathology, relative to children in the dietary subtype. Finally, E. Penas-Lledo et al. (2009) used empirical methodology to document dietary and dietary-negative affect classes in a sample of 1,005 individuals with AN, BN, BED or EDNOS; however, their results also revealed two new subtypes, a “mild dietary restraint” subtype characterized by low scores on depressive symptoms and drive for thinness and a “depressive-moderate dietary restraint” subtype characterized by elevated depressive symptom severity, but milder levels of drive for thinness than the dietary-negative affect group (p. 515). Notably, individuals with AN (n = 194) were overrepresented in the mild dietary restraint and depressive-moderate dietary restraint groups, which the authors speculate might reflect satisfaction with low body weight in acutely ill AN patients (E. Penas-Lledo et al., 2009). In contrast, the majority of individuals with the purging subtype of BN (n = 450) were classified in the traditional dietary and dietary-negative affect classes (n.b., individuals with BED and EDNOS were evenly distributed across the four subtypes.

Finally, two studies have attempted to document dietary and dietary-negative affect subtypes in non-referred samples in an effort to identify individuals at-risk for disordered eating. Using cluster analysis in a sample of 623 undergraduate females, E. M. Penas-Lledo, Loeb, Puerto, Hildebrandt, and Llerena (2008) found three groups: 1) a dietary subtype characterized by high levels of restraint and low negative affect (36%), 2) a “mixed” (p. 728) dietary-negative affect subtype characterized by high levels of restraint and depressive symptoms (16%), and 3) a healthy subtype characterized by low restraint and depressive symptoms (47%). Not surprisingly, rates of self-reported binge eating, fasting, and purging were higher in the dietary and dietary-negative affect classes relative to the healthy group. Moreover, individuals in the dietary-negative affect subtype endorsed more severe disinhibition and purging than individuals in the dietary subtype, while individuals in the dietary subtype reported greater use of exercise to control weight than individuals in the dietary-negative affect group (E. M. Penas-Lledo et al., 2008). Chen, McCloskey, and Keenan (2009) found a similar pattern of results in a sample of 543 girls enrolled in the longitudinal Pittsburgh Girls Study (Hipwell, et al., 2002). Using assessments conducted when the participants were 10 years old, Chen et al. (2009) identified two subtypes: 1) an “at-risk” (p. 278) dietary-depressive subtype characterized by high levels of depressive symptomatology and dietary restraint, and 2) a low risk subtype characterized by minimal depressive symptoms and little restraint. Moreover, membership in the dietary-depressive subtype at age 10 predicted binge eating at ages 12 and 14. Chen et al. (2009) did not identify a pure dietary subtype in their study, but this may have been a confound of the analytic approach used, as they specified two clusters in the cluster analysis, and it is unlikely that only dietary and dietary-depressive subtypes would emerge in a non-referred sample (a more probable outcome would be three clusters, similar to the results of E. M. Penas-Lledo et al., 2008).

In summary, there is ample support for the presence of dietary and dietary-negative affect subtypes in individuals with EDs, particularly disorders characterized by binge eating or loss of control. Moreover, emerging data suggest that dietary and dietary-negative affect classes

also may be present in individuals at-risk for disordered eating. As detailed above, the literature on dietary and dietary-negative affect ED subtypes overcomes many of the limitations of earlier comorbidity-based classification schemes by: 1) using empirical methodology (i.e., cluster analysis) to define the groups, 2) focusing primarily on outpatient and community samples rather than individuals receiving treatment at tertiary care facilities, 3) evaluating the validity of the subtypes relative to existing classification schemes and comorbid diagnoses, and 4) providing preliminary evidence for the short-term stability of the dietary and dietary-negative affect groups. Nevertheless, several issues warrant consideration when evaluating the utility of the dietary/dietary-negative affect subtyping scheme for classifying individuals with EDs.

First, it is unclear to what extent dietary and dietary-negative affect classes are salient to individuals with restricting AN and other forms of eating disturbance not characterized by binge eating or loss of control. As noted above, the theoretical basis for these subtypes derives from models of the etiology and maintenance of binge eating and bulimic syndromes, and the one study that examined dietary and dietary negative affect classes in a full range of EDs found that AN was overrepresented in other subtypes (E. Penas-Lledo et al., 2009). Second, most studies have documented dietary and dietary-negative affect subtypes based on a very limited range of comorbid psychopathology (i.e., depressive symptoms) and associated features (i.e., self-esteem) (see Stice et al., 2008 for a notable exception). Thus, it is unclear to what extent heterogeneity may exist within the dietary and dietary-negative affect classes with respect to other forms of psychiatric disturbance that might help to identify meaningful subgroups of the ED population (e.g., anxiety, impulsivity, obsessive-compulsive traits). The degree to which the dietary-negative affect class overlaps with other proposed comorbidity subtypes (e.g., borderline BN) also is unknown. Third, given that dietary restraint is elevated in both the dietary and dietary-negative affect subtypes, some scholars have questioned whether it is necessary to include measures of dietary restraint in this classification scheme, or if it would be better to focus on other conceptually-relevant dimensions (Masheb & Grilo, 2008). Finally, some studies have documented higher rates of binge eating or purging in the dietary-negative affect subtype relative to the dietary subtype (Carrard et al., 2012; Stice et al., 2001; Stice et al., 2008; Stice & Fairburn, 2003), which might suggest that this scheme is a proxy for severity of illness rather than an index of distinct subgroups within the ED population. However, other reports have found no differences between the dietary and dietary-negative affect classes with respect to severity of ED behaviors (Chen & Le Grange, 2007; Grilo, Masheb, & Berman, 2001; Grilo, Masheb, & Wilson, 2001; Masheb & Grilo, 2008), and there is evidence that these groups have more validity than classification schemes based on ED severity (Chen & Le Grange, 2007) or comorbid diagnoses (Grilo, Masheb, & Wilson, 2001).

Undercontrolled, Overcontrolled and Low Psychopathology EDs

Another model of comorbidity-based classification that has gained increasing attention in the EDs field emphasizes systematic heterogeneity in patterns of comorbid psychopathology across multiple domains that have salience to disordered eating (e.g., mood disturbance, anxiety, impulsivity). Table 1 provides a chronological overview this research. Due to space limitations, descriptions of the studies are abbreviated; a more comprehensive overview of this literature is provided as an on-line supplement (see Table A.1 in Appendix A).

As shown in Table 1, we identified 23 published studies, representing 21 unique ED samples (N = 5,030) that have characterized subgroups on the basis of a broad range of comorbid symptoms, diagnoses, or psychopathological personality traits. On first glance, this body of work appears quite heterogeneous. Samples have varied in size from N = 46 (Karwautz, Troop, Rabe-Hesketh, Collier, & Treasure, 2003) to N = 1,312 (Krug et al., 2011) and have comprised adolescents (see, e.g., Strober, 1983; Thompson-Brenner, Eddy, Satir, Boisseau,

& Westen, 2008) and adults (see, e.g., Holliday et al., 2005; Wonderlich et al., 2005), and individuals with AN (e.g., Holliday et al., 2005; Wildes et al., 2011), BN (e.g., Wonderlich, Crosby et al., 2007; Wonderlich et al., 2005), or a combination of AN, BN, and EDNOS diagnoses (e.g., Claes et al., 2006; Steiger et al., 2009). Studies have been conducted in inpatient (Strober, 1983; Wildes et al., 2011), outpatient (Espelage et al., 2002; Goldner, Srikameswaran, Schroeder, Livesley, & Birmingham, 1999), and non-treatment settings (Holliday et al., 2005; Wonderlich, Crosby et al., 2007), and assessments have included self-report questionnaires (e.g., Claes et al., 2006; Holliday et al., 2005; Steiger et al., 2009; Wonderlich, Crosby et al., 2007), diagnostic interviews (Duncan et al., 2005), and clinician reports (e.g., Thompson-Brenner, Eddy, Satir et al., 2008; Westen & Harnden-Fischer, 2001). The majority of studies have focused on comorbid personality psychopathology, but some research groups have examined patterned heterogeneity in Axis I diagnoses (Duncan et al., 2005) or a combination of personality psychopathology and other psychiatric symptoms (e.g., depression, substance use; Steiger et al., 2009; Wonderlich et al., 2005). Finally, methods of classifying individuals have varied widely from empirical techniques such as cluster analysis (e.g., Espelage et al., 2002) and latent structure analysis (e.g., Duncan et al., 2005; Wildes et al., 2011; Wonderlich et al., 2005) to dimensional approaches (e.g., Q-sort; Thompson-Brenner, Eddy, Satir et al., 2008; Westen & Harnden-Fischer, 2001) and clinician assignment (Thompson-Brenner & Westen, 2005).

Yet, despite marked variability in sample composition, type of assessment, comorbid psychopathology, and statistical analysis, findings generally have converged around three classes: 1) an “undercontrolled” class characterized by impulsivity, risky or dissocial behaviors (e.g., suicide attempts, substance misuse), and emotional reactivity; 2) an “overcontrolled” class characterized by rigidity, compulsivity, inhibition, and avoidance; and 3) a “low (comorbid) psychopathology” class characterized by the relative absence of comorbid psychopathology. Moreover, even in studies that have identified greater or fewer than three comorbidity subtypes (e.g., Duncan et al., 2005; Krug et al., 2011; E. Penas-Lledo et al., 2010; Thompson-Brenner, Eddy, Franko, Dorer, Vashchenko, Kass et al., 2008), groups resembling the undercontrolled, overcontrolled, and low psychopathology classes have emerged. For example, Thompson-Brenner et al. documented five personality “prototypes” in a sample of 213 females with AN or BN (Thompson-Brenner, Eddy, Franko, Dorer, Vashchenko, & Herzog, 2008; Thompson-Brenner, Eddy, Franko, Dorer, Vashchenko, Kass et al., 2008). However, as shown in Table A.1, one prototype (“high functioning”) resembles the low psychopathology class, and the other four appear to reflect aspects of the undercontrolled (“behaviorally dysregulated,” “emotionally dysregulated”) and overcontrolled (“avoidant-insecure,” “obsessional-sensitive”) groups.

Given the documented instability of ED diagnoses (e.g., Fichter & Quadflieg, 2007), the applicability of the undercontrolled, overcontrolled, and low psychopathology subtypes to a broad range of ED presentations is a major advantage of this approach. Moreover, some scholars have speculated that systematic heterogeneity in comorbid psychopathology might be associated with distinct pathways to the expression or maintenance of ED symptoms (see, e.g., Westen & Harnden-Fischer, 2001; Wonderlich et al., 2005), which could inform a novel system of classification. In support of this idea, one study found that AN patients resembling the low psychopathology group were twice as likely to have a family history of ED than AN patients resembling the undercontrolled and overcontrolled groups (Holliday et al., 2005), suggesting that low psychopathology patients may have a stronger genetic diathesis for disordered eating. Alternatively, individuals with different ED symptoms, but a similar pattern of comorbid psychopathology might share risk or maintaining factors that distinguish them from other individuals with the same eating behaviors (e.g., an overcontrolled AN patient might have more in common with an overcontrolled BN patient than an undercontrolled AN patient). As evidence for this hypothesis, Steiger et al. (2009)

found that variations in the expression of the 5HTTLPR genetic polymorphism, which have been linked to EDs (Di Bella, Catalano, Cavallini, Riboldi, & Bellodi, 2000), were more strongly related to membership in "inhibited/compulsive" (i.e., overcontrolled) and "dissocial/impulsive" (i.e., undercontrolled) classes than to ED diagnosis. Childhood abuse history also has been shown to be associated more strongly with undercontrolled class membership than with ED diagnosis (Steiger et al., 2010; Westen & Harnden-Fischer, 2001).

In addition to documenting differences among the undercontrolled, overcontrolled, and low psychopathology classes with respect to putative mechanisms that may underlie the expression of aberrant eating, several studies have examined the clinical utility of this classification scheme. Collectively, this work provides preliminary support for the idea that undercontrolled, overcontrolled, and low psychopathology classes are superior to categorical ED diagnoses in predicting patterns of treatment utilization and response, psychosocial functioning, and course of illness (Thompson-Brenner, Eddy, Franko, Dorer, Vashchenko, Kass et al., 2008; Thompson-Brenner & Westen, 2005; Westen & Harnden-Fischer, 2001; Wildes et al., 2011). Furthermore, several reports have indicated that membership in the undercontrolled class is associated with a particularly pernicious course of disordered eating (for a notable exception, see (Thompson-Brenner, Eddy, Franko, Dorer, Vashchenko, Kass et al., 2008). For example, in a sample of 154 adolescents and adults with AN, Wildes et al. (2011) found that membership in an undercontrolled class, relative to overcontrolled and low psychopathology groups, predicted a poorer response to intensive treatment and an elevated risk of readmission within three months of discharge, even after controlling for other predictors of illness course and treatment response. Undercontrolled, overcontrolled, and low psychopathology classes also were superior to restricting and binge-eating/purging AN subtypes in predicting clinical outcomes.

Finally, groups resembling the undercontrolled and overcontrolled classes have been documented in individuals recovered from EDs, suggesting that this approach might offer a stable method of classifying eating-related psychopathology. Specifically, Wagner et al. (2006) identified two personality-based subgroups in a sample of 60 females recovered from AN or BN; one was characterized by impulsivity and novelty seeking (i.e., undercontrolled) and the other by harm avoidance (i.e., overcontrolled). Individuals assigned empirically to the overcontrolled class had higher levels of state anxiety than individuals in the undercontrolled class, but there were no differences between the groups in lifetime Axis I and II diagnoses. It is important to note that severity ratings on the measures used to identify the undercontrolled and overcontrolled classes were lower in the recovered sample (Wagner et al., 2006) than in studies of individuals actively ill with EDs (Krug et al., 2011). Thus, stability might be conceptualized best as an enduring systematic heterogeneity with respect to comorbid symptoms or traits that, when exacerbated, may promote the expression or maintenance of disordered eating.

In summary, studies that have classified individuals with EDs on the basis of patterned heterogeneity in multiple domains of comorbid psychopathology consistently have documented three groups – undercontrolled, overcontrolled, and low psychopathology. Notably, these groups have been robust to wide variations in sample composition, assessment procedure, reporter characteristics (i.e., self-versus clinician-report), and statistical methodology. Moreover, preliminary data suggest that membership in the undercontrolled, overcontrolled, and low psychopathology subgroups may be associated with distinct pathways to the expression or maintenance of disordered eating symptoms (Holliday et al., 2005; Steiger, et al., 2009), and may have implications for illness course and treatment response (Thompson-Brenner, Eddy, Franko, Dorer, Vashchenko, Kass, et al., 2008; Wildes et al., 2011).

Nevertheless, like other comorbidity-based nosologies, the literature on undercontrolled, overcontrolled, and low psychopathology ED classes has several limitations. First, although groups resembling the three major classes have been documented in numerous reports, few studies have used identical sets of class indicators (for notable exceptions, see Holliday et al., 2005 and Wonderlich, Crosby et al., 2007, as well as Claes et al., 2012; Karwautz et al., 2003; and Krug et al., 2011). Consequently, the characteristics of the undercontrolled, overcontrolled, and low psychopathology classes have varied across studies, and it is unclear what forms of comorbid psychopathology constitute the cardinal features of each group. Second, the extent to which the undercontrolled and overcontrolled classes overlap with alternative comorbidity-based classification schemes is unknown. Descriptive similarities among the undercontrolled, multi-impulsive, and borderline ED classes are especially noteworthy. It also is possible that the undercontrolled and overcontrolled groups might overlap with the dietary-negative affect ED class, and the low psychopathology group might intersect with the dietary subtype. Third, no study has examined the stability of the undercontrolled, overcontrolled, and low psychopathology classes; thus, it is unclear whether diagnostic crossover exists with this scheme. Finally, although the low psychopathology group has been documented in numerous reports, the theoretical mechanisms that underlie the expression and maintenance of disordered eating symptoms in this class are difficult to discern. One possibility is that like the dietary subtype of BN and BED, low psychopathology EDs are more strongly associated with behavioral (i.e., dietary restraint) or cultural (e.g., pressures for thinness) factors that have been linked to disordered eating than to comorbid psychopathology. The low psychopathology class also may have a stronger genetic diathesis for disordered eating than the undercontrolled and overcontrolled groups (Holliday et al., 2005). However, an alternative hypothesis might be that the low psychopathology group includes individuals who have underreported comorbid psychopathology in previous research, or those for whom existing assessments have not captured aspects of psychiatric comorbidity that are most salient to disordered eating. It also is possible that low psychopathology EDs might be characterized by a more narrow range of comorbid psychopathology than the undercontrolled and overcontrolled classes.

Emerging Methods of Classifying EDs

Finally, we identified three recent proposals for grouping individuals with EDs on the basis of comorbid psychopathology or associated features that may hold promise for future research. For example, Hopwood and colleagues have published two reports examining the utility of including information about interpersonal problems in the assessment of individuals with “bulimic features” (Ambwani & Hopwood, 2009; Hopwood, Clarke, & Perez, 2007). Preliminary data suggest that women with bulimic features are characterized by four distinct interpersonal profiles that differ with respect to the influence of dietary restraint and negative affect on the expression of ED symptoms (Ambwani & Hopwood, 2009). Similarly, Turner et al. have published two papers evaluating the presence and validity of four ED subgroups defined, in part, by attachment and coping style (Turner, Bryant-Waugh, & Peveler, 2009, 2010). Initial findings suggest that these groups are superior to *DSM-IV* diagnoses in predicting functional impairment, social functioning, and general mental health (Turner et al., 2010). Finally, one recent study found that individuals with AN and a history of autism spectrum disorder (ASD) had poorer clinical outcomes at 18-year follow-up compared to individuals with AN and no ASD comorbidity (Anckarsater et al., 2012).

General Summary

In closing, there has been a long-standing interest in the implications of comorbid psychopathology for the classification of EDs. Although the models reviewed herein differ in theoretical rationale and methodology, each approach has provided support for the notion

that there are subgroups of the ED population that vary systematically with respect to comorbid psychopathology and associated features (e.g., high-risk behaviors, low self-esteem). Moreover, research has indicated that systems of classifying EDs that incorporate information about comorbid psychopathology likely have more concurrent and predictive validity than approaches that rely exclusively on descriptions of disordered eating symptoms (e.g., Stice et al., 2008; Stice & Fairburn, 2003; Westen & Harnden-Fischer, 2001; Wildes et al., 2011). Finally, the extant literature provides insight into aspects of comorbid psychopathology that are most salient to the classification of EDs. For example, impulsive behaviors and personality traits have been shown to distinguish a subgroup of EDs in several models (viz., multi-impulsive EDs, borderline BN, undercontrolled EDs). Similarly, emotional reactivity and distress have characterized a number of comorbidity-based ED classes (e.g., borderline BN, dietary-negative affect classes, undercontrolled and overcontrolled classes). Rigidity and compulsivity have emerged as distinguishing features of the overcontrolled ED class in several reports, and less severe manifestations of these traits also may characterize low psychopathology individuals (Holliday et al., 2005; Westen & Harnden-Fischer, 2001). Finally, variations in inhibition or avoidance versus approach and risk-taking appear to distinguish overcontrolled ED presentations from undercontrolled, borderline, and multi-impulsive classes.

Directions for Future Research

Findings to date suggest several directions for future research on alternative methods of classifying EDs. First, many of the comorbidity-related constructs that have been used to classify individuals with EDs, such as impulsivity, BPD traits, and negative affect, are associated most strongly with bulimic symptoms (Cassin & von Ranson, 2005; Stice 2001). Moreover, research on the multi-impulsive, borderline, and dietary/dietary-negative affect classification schemes has focused almost exclusively on individuals with bulimic syndromes. Although some of the more recent alternative ED nosologies have incorporated aspects of comorbid psychopathology that are salient to restrictive syndromes (e.g., ASD symptoms, inhibited and compulsive personality traits) and have been tested in individuals with AN and related EDNOS diagnoses (see, e.g., Table 1), additional research is needed to evaluate whether these approaches have validity across the full range of ED presentations.

Second, longitudinal studies focusing on the temporal stability and predictive validity of a comorbidity-based approach to ED classification are needed. High rates of diagnostic crossover constitute one of the most serious limitations of current ED nosologies, because models of etiology and maintenance developed for one disorder (e.g., AN) may not apply to individuals who migrate to another diagnostic category (e.g., transition from AN to BN). Preliminary data suggest that approaches to ED classification that incorporate comorbid psychopathology are more stable than the *DSM-IV* ED diagnoses (Grilo, Masheb, & Wilson, 2001; Stice et al., 2008). However, no research, to our knowledge has examined the stability of such alternative approaches to ED classification over a period longer than four weeks. Similarly, few studies have evaluated the predictive validity of comorbidity-based systems of classifying EDs using prospective longitudinal designs (see Stice et al., 2008; Thompson-Brenner, Eddy, Franko, Dorer, Vashchenko, Kass et al., 2008; Wildes et al., 2011 for notable exceptions). Finally, it will be important for future studies to evaluate the relative utility of ED classification schemes based solely on comorbid psychopathology (e.g., undercontrolled, overcontrolled and low psychopathology classes) versus those that incorporate information about disordered eating (e.g., dietary and dietary-negative affect subtypes).

Third, hypothesis-driven studies are needed to elucidate common psychopathological dimensions that may underlie the expression of different comorbidity-based classification

schemes. There are noteworthy similarities in the subgroups derived from the multi-impulsive, borderline, dietary/dietary-negative affect, and undercontrolled, overcontrolled and low psychopathology models, and integration of these approaches could help to maximize the validity of a comorbidity-based approach to ED classification. To this end, studies using neurobiologically-informed constructs such as those proposed by the Research Domain Criteria project (RDoC; Insel et al., 2010; Sanislow et al., 2010) may be particular helpful. Indeed, several of the RDoC domains appear to have salience for identifying comorbidity-based ED subgroups, and the RDoC matrices suggest using multiple levels of analysis to measure these phenomena. For example, at a basic behavioral level, experimental neurocognitive tasks (e.g., delay discounting) might be used to examine the degree to which specific RDoC constructs (e.g., reward valuation) are shared by and differentiate various comorbidity-based ED classes.

One model that we think holds particular promise for characterizing EDs focuses on incorporating neurocognitive dimensions of impulsivity and compulsivity into the classification of psychiatric disorders (Robbins, Gillan, Smith, de Wit, & Ersche, 2012). Impulsivity and compulsivity are salient to several comorbidity-based approaches to ED classification (e.g., multi-impulsive, borderline, OCD, undercontrolled, overcontrolled and low psychopathology). Moreover, the descriptions of many ED symptoms appear to include impulsive (e.g., loss of control in binge eating) or compulsive (e.g., recurrent inappropriate compensatory behaviors to prevent weight gain) components. Alterations in several neural circuits (e.g., anterior cingulate cortex, orbitofrontal cortex, ventral striatum) and neurotransmitter systems (e.g., serotonin, dopamine) related to impulsivity and compulsivity have been found in individuals with EDs (Frank & Kaye, 2012; Kaye, 2008). Finally, neuropsychological research provides support for the notion that neurocognitive dimensions related to impulsivity and compulsivity are relevant to EDs (Friederich & Herzog, 2011; Van den Eynde, et al., 2011). Thus, research using experimental neurocognitive tasks to examine specific aspects of impulsivity and compulsivity in individuals with EDs could shed light on mechanisms associated with the expression of both ED symptoms and comorbid psychopathology and help to validate comorbidity-based approaches to ED classification. Furthermore, the notion that patterned heterogeneity in comorbid psychopathology might distinguish subgroups of individuals with the same categorical diagnosis is not limited to EDs (see, e.g., Wolf, Miller, Harrington, & Reardon, 2012). Thus, research focusing on the behavioral and biological correlates of comorbidity-based ED classes could have implications outside the EDs field.

Clinical Implications of Including Comorbid Psychopathology in the Classification of EDs

An approach to classifying EDs that incorporates comorbid psychopathology and associated features also has important implications for clinical practice. Indeed, if systematic heterogeneity in patterns of comorbid psychopathology among individuals with EDs reflects distinct pathways to the expression or maintenance of disordered eating symptoms, as hypothesized by several scholars (Stice & Agras, 1999; Westen & Harnden-Fischer, 2001; Wonderlich et al., 2005), then interventions likely will need to be tailored to address factors that are most salient to particular subgroups. For example, if additional research supports the validity of an impulsive/undercontrolled ED subtype, interventions for these individuals might focus on strategies to improve emotion regulation and impulse control, such as those proposed in dialectical behavior therapy and recent neuropsychological treatments (Bickel, Yi, Landes, Hill, & Baxter, 2011; Linehan et al., 2006). Alternatively, interventions for a well-characterized and validated overcontrolled ED subtype might emphasize cognitive flexibility and attention retraining to decrease rigidity and anxiety related to eating (Najmi & Amir, 2010; Tchanturia, Davies, & Campbell, 2007). Ultimately, the promise of a comorbidity-based system of ED classification lies in the notion that including aspects of

psychopathology that are not directly related to eating, shape or weight will lead to the delineation of precise behavioral phenotypes that will promote the identification of neurobiological mechanisms that underlie the expression of ED symptoms and facilitate the selection of targeted treatments for individual patients.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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Table 1

Chronological Overview of Studies that Have Characterized Eating Disorder Subgroups on the Basis of Systematic Heterogeneity in Multiple Domains of Comorbid Psychopathology

Study	Diagnosis	N	Indicators	Number of Subgroups
Strober (1983)	AN	130	Minnesota Multiphasic Personality Inventory	3
Goldner, et al. (1999)	AN, BN & EDNOS	136	Dimensional Assessment of Personality Psychopathology – Basic Questionnaire (DAPP-BQ)	3
Westen and Harnden-Fischer (2001)	AN, BN & EDNOS	103	Shelder-Westen Assessment Procedure-200 (SWAP-200)	3
Espelage, et al. (2002)	AN, BN & EDNOS	183	Millon Clinical Multiaxial Inventory-II	3
Karwautz, et al. (2003)	AN-R	46	Temperament and Character Inventory (TCI)	3
Duncan, et al. (2005)	BN	122	*DSM-III-R* mood, anxiety, substance use, and antisocial PD diagnoses	2
Holliday, et al. (2005)	AN	153	DAPP-BQ	3
Thompson-Brenner and Westen (2005)	AN-BP, BN, & EDNOS	145	Clinician descriptions	3
Wonderlich, et al. (2005)	BN	178	Impulsive Behavior Scale; Frost Multi- Dimensional Perfectionism Scale; Inventory of Depressive Symptoms- Self-report; State-Trait Anxiety Inventory; Michigan	3
Wonderlich, et al. (2005) - *continued*			Assessment Screening Test/Alcohol-Drug; 5-HTTLPR s allele	
Claes, et al. (2006)	AN, BN & EDNOS	335	Neuroticism, Extraversion, Openness to New Experiences-Five Factor Inventory	3
Wagner, et al. (2006)	AN & BN	60	TCI; Barratt Impulsiveness Scale (BIS)	2
Wonderlich, Crosby, et al. (2007)	BN	131	DAPP-BQ	3
Thompson-Brenner, Eddy, Franko, Dorer, Vashchenko, Kass, et al. (2008)	AN & BN	213	Structured Interview for *DSM-III* PDs	5
Thompson-Brenner, Eddy, Franko, Dorer, Vashchenko, and Herzog (2008)	Same as Thompson-Brenner, Eddy, Franko, Dorer, Vashchenko, Kass, et al. (2008)			
Thompson-Brenner, Eddy, Satir, et al. (2008)	AN, BN & EDNOS	120	SWAP-200 adolescent version	3
Steiger, et al. (2009)	AN, BN & EDNOS	185	DAPP-BQ; BIS; Center for Epidemiologic Studies Depression (CES-D)	3
Steiger, et al. (2010)	Same as Steiger, et al. (2009)			
Hopwood, Ansell, Fehon, and Grilo (2010)	ED features	153	Millon Adolescent Clinical Inventory	3
E. Penas-Lledo, et al. (2010)	AN, BN & EDNOS	825	Social Avoidance Distress Scale; TCI-revised	5

Study	Diagnosis	N	Indicators	Number of Subgroups
Krug, et al. (2011)	AN, BN, BED & EDNOS	1,312	TCI-revised	6
Wildes, et al. (2011)	AN	154	Schedule for Nonadaptive and Adaptive Personality – 2nd edition	3
Claes, et al. (2012)	AN, BN, & EDNOS	132	TCI-revised	3
Waller, Ormonde, and Kuteyi (2012)	AN, BN, BED & EDNOS	214	Personality Belief Questionnaire-Short Form	3

Note. AN = anorexia nervosa; BN = bulimia nervosa; EDNOS = eating disorder not otherwise specified; AN-R = anorexia nervosa, restricting type; AN-B/P = anorexia nervosa, binge eating/purging type; ED = eating disorder; BED = binge eating disorder; PD = personality disorder; *DSM-III* = Diagnostic and Statistical Manual of Mental Disorders, 3rd ed.