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Incorporating Dimensions into the Classification of Eating Disorders: Three Models and Their Implications for Research and Clinical Practice

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Abstract

Objective—Given renewed interest in dimensional approaches in psychiatric nosology, we review evidence for the utility of including dimensions of eating disorder (ED) psychopathology, comorbid psychopathology, and neurobiology in the classification of EDs.

Method—We searched on-line databases and reference lists of published papers for articles on dimensional methods in psychiatric classification, with an emphasis on EDs.

Results—ED classification may be enhanced by including dimensional assessments. Specifically, some ED features appear to exist on a continuum with normality, and dimensional measures of ED severity are likely to have utility in clinical contexts. Furthermore, accumulating data support the validity of ED classification schemes that incorporate dimensions of comorbid psychopathology. Finally, neurobiological dimensions have gained increasing notice across the psychiatric literature and may be particularly salient to the classification of EDs.

Discussion—An approach to ED classification that incorporates multiple dimensional measures may have the greatest utility for advancing ED treatment.

The impending publication of the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) has led to a resurgence of interest in psychiatric nosology. Although DSM-5 will continue the descriptive, atheoretical tradition established in DSM-III (1) and shared by the International Classification of Diseases (ICD) (2), there is increasing recognition of the need for alternative approaches to classifying mental illness [see, e.g., Refs. (3, 4)]. One issue that has garnered particular attention concerns the utility of incorporating dimensional measures into the current categorical system of psychiatric classification. Based, in part, on recommendations from a meeting sponsored by the American Psychiatric Association, World Health Organization and U.S. National Institutes of Health (5), the DSM-5 Task Force has proposed that a series of “cross-cutting” dimensional assessments (e.g., depressed mood, sleep problems) be included in DSM-5, in addition to categorical diagnoses (6). Similarly, in the U.S., the National Institute of Mental Health has called upon investigators “to develop, for research purposes, new ways of classifying mental disorders based on dimensions of observable behavior and neurobiological measures” (7), a process that is being implemented through the Research Domain Criteria (RDoC) project (3).

In light of the growing interest in dimensional approaches to psychiatric classification, it is timely to reconsider the literature on dimensional models of eating disorders (EDs). There has been a long-standing interest in dimensional approaches to ED classification [for review, see Ref (8)]. However, recent advances in statistical methodology and neuroscience suggest new directions for this work that may have important implications for research and clinical practice. In the sections that follow, we present three models for incorporating dimensions into the classification of EDs that we think have particular promise for promoting new models of etiology and treatment. The first model focuses on dimensions of eating behavior and ED-related cognitions. The second model focuses on other psychopathological dimensions that often co-occur with EDs and may be involved in the expression and maintenance of ED symptoms. Finally, the third model focuses on neurobiological dimensions that may underlie both ED symptoms and comorbid psychopathology, and may provide a unifying framework for the classification of psychiatric disorders.

Model # 1: Dimensions of ED Psychopathology

EDs are heterogeneous illnesses comprised of multiple symptoms (e.g., body weight, binge eating, purging, preoccupation with eating, shape or weight) that may vary along dimensions of severity within and across diagnostic categories. Clinical observers have noted that ED patients often will change on one or more symptom dimensions without changing on the others (9), making constructs like remission and recovery difficult to define (10, 11). Furthermore, fluctuation in symptom presentation and diagnostic crossover are common across the ED spectrum (12), suggesting that an approach to ED classification that incorporates symptom dimensions may have more validity than the current categorical diagnoses.

Nevertheless, the number and specific nature of the dimensions that best describe ED psychopathology remain elusive. One issue that has received particular attention concerns the extent to which aspects of ED psychopathology are dimensional not only across diagnoses, but also in relation to normal behavior. For example, several studies have examined the underlying structure of ED symptoms using taxometric analysis, a statistical technique designed to evaluate whether constructs are dimensional (i.e., vary in degree) or categorical (i.e., vary in kind) across a population [for review, see Refs. (8, 13)]. In general, this work suggests that the cognitive correlates of disordered eating (e.g., self-reported dietary restraint, body dissatisfaction, drive for thinness) are continuous with normality (i.e., dimensional) (14). However, the underlying structure of ED behaviors is less clear, with some studies reporting that EDs characterized by binge eating are qualitatively distinct from restrictive EDs and normal behavior (i.e., they are categorical or taxonic) (8), and others finding that a dimensional structure best describes the associations among restrictive and binge-eating/purging EDs (15). Finally, it is important to note that taxonic constructs often are characterized by within-group variability that is dimensional in nature (8). Thus, even if categorical distinctions exist among ED diagnoses, dimensional assessments still may be required to fully characterize an individual's presenting symptoms.

For example, the Eating Disorders Work Group for DSM-5 has proposed that several disorder-specific dimensions be included in the classification of EDs as a means of documenting within-group variability in severity. In particular, the proposed criteria for anorexia nervosa (AN) include a severity dimension based on current body mass index; the proposed criteria for bulimia nervosa (BN) include a severity dimension based on the frequency of inappropriate compensatory behaviors; and, the proposed criteria for binge eating disorder (BED) include a severity dimension based on the frequency of binge eating (16).

Alternatively, Keel et al. (17) have argued that a single latent severity dimension, representing the total number of symptoms endorsed, provides a more accurate description of within-group variability across ED classes than do disorder-specific dimensions. Using a series of latent class analysis, factor analysis, and factor mixture analysis techniques in a sample of 528 individuals with bulimic syndromes (i.e., AN-binge/purge type, BN, BED, purging disorder and related ED not otherwise specified diagnoses), Keel et al. documented three latent classes (i.e., categories) and one underlying severity dimension. Importantly, analyses provided no support for the notion that different features contribute to severity in different ED classes. Instead, the study results indicated that illness severity in EDs is a function of symptom count, regardless of ED diagnosis. This means that a person with four symptoms of AN would be equivalent in severity to a person with four symptoms of BN, both of whom would have a more severe illness than a person with three symptoms of AN. These findings are consistent with previous work that has documented a positive association between functional impairment and number of ED behaviors (18).

In summary, there is support for dimensional approaches to conceptualizing ED psychopathology, but evidence to guide the selection of particular dimensional assessments for ED classification is limited. Recent data suggest that the total number of ED symptoms endorsed may be a useful index of severity (17), which could help to guide clinical decision-making and provide a measure of outcome following treatment. Alternatively, for work conducted in general population settings (e.g., risk factor research, prevention efforts), dimensional assessments of drive for thinness, body dissatisfaction or other cognitive correlates of disordered eating may provide the best characterization of ED psychopathology (14). Finally, some scholars have proposed that dimensional ratings of specific ED symptoms (e.g., body weight, overvaluation of shape and weight) be incorporated into the current classification schemes (16, 19); however, empirical support for this approach relative to other models (e.g., symptom counts) is lacking.

Model # 2: Dimensions of Comorbid Psychopathology

A burgeoning literature has documented the validity of ED classification schemes that incorporate dimensions of comorbid psychopathology. The rationale for a comorbidity-based approach to ED classification derives, in part, from the observation that rates of comorbid psychopathology are high in individuals with EDs (20). There also is evidence that certain forms of comorbid psychopathology precede the onset of ED symptoms [for review, see Ref. (21)], suggesting that these conditions might play a precipitating role in the emergence of aberrant eating. Thus, an approach to classifying EDs that incorporates dimensions of comorbid psychopathology might help to elucidate within-group differences in the mechanisms that underlie the expression of disordered eating. Below we describe two comorbidity-based approaches to ED classification that have gained increasing notice in recent years. For a more complete overview of comorbidity and the classification of EDs, the interested reader is referred to Ref. (22).

Dietary and dietary-negative affect EDs

Informed by the dual-pathway model of BN (23), a large body of work has utilized dimensions of dietary restraint and negative affect, as measured by self-report questionnaires such as the Three-Factor Eating Questionnaire (24) (restraint) and the Beck Depression Inventory (25) (negative affect), to classify individuals with bulimic syndromes [e.g., Refs. (23, 26–28)]. This research is notable for several reasons. First, the dietary and dietary-negative affect (also termed dietary-depressive) subtypes have been documented empirically using cluster analytic techniques in a broad range of ED presentations including adolescents and adults with BN, BED and other ED features (23, 26–29), as well as children with loss of control eating (30). Second, data consistently have shown that the dietary-negative affect

class is associated with more severe ED symptoms and comorbid psychopathology, greater psychosocial impairment and a poorer response to treatment than the dietary class (23, 26–28). Moreover, the dietary and dietary-negative affect subtypes have demonstrated superiority relative to other classification schemes (e.g., purging/non-purging BN subtypes, classification based on categorical indices of comorbid psychopathology) in predicting these outcomes (26–28). Third, membership in the dietary and dietary-negative affect classes appears to be stable over short-term follow-up (26, 28). Finally, emerging data suggest that the dietary and dietary-negative affect classes are present in individuals at-risk for disordered eating (31), suggesting that an approach to ED classification that incorporates dimensions of dietary restraint and negative affect could have utility for risk factor research and prevention efforts.

Nevertheless, several issues warrant consideration before dimensions of dietary restraint and negative affect could be included in the classification of EDs. Of particular concern, because the theoretical basis for the dietary and dietary-negative affect groups derives from a model of the etiology and maintenance of bulimic syndromes (23), the relevance of this classification scheme to EDs not characterized by binge eating or loss of control is unclear. Furthermore, most studies have characterized dietary and dietary-negative affect subtypes solely on the basis of self-reported dietary restraint, depressive symptoms and low self-esteem [for a notable exception, see Ref. (28)], without regard to other dimensions of psychopathology that might have salience for the classification of EDs (e.g., anxiety, impulsivity). Finally, the inclusion of a dietary restraint dimension in this classification scheme appears to have limited utility for distinguishing subgroups of the EDs population, given that scores on this dimension are elevated in both subtypes.

Undercontrolled, overcontrolled and low psychopathology EDs

Another approach to incorporating dimensions in the classification of EDs emphasizes systematic heterogeneity in multiple domains of comorbid psychopathology that have relevance to aberrant eating (e.g., mood disturbance, anxiety, impulsivity). As reviewed in detail elsewhere (22), although much of this work has focused on dimensions of temperament and personality that have been hypothesized to drive the expression of ED symptoms and general psychopathology, patterned heterogeneity in Axis I diagnoses or a combination of personality traits and psychiatric symptoms also have been examined. Despite variability in the constructs that have been studied and the measures used to assess them (viz., interviews, self-report questionnaires and clinician reports), studies using both quantitative (e.g., latent structure analyses) and qualitative techniques to characterize ED subgroups on the basis of multiple dimensions of comorbid psychopathology generally have documented 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 psychopathology” class characterized by the relative absence of comorbid psychopathology (22). Moreover, even in studies that have identified greater or fewer than three subtypes, groups resembling the undercontrolled, overcontrolled and low psychopathology classes have emerged [e.g., (32)]. Undercontrolled, overcontrolled and low psychopathology subtypes have demonstrated superiority relative to categorical ED diagnoses in predicting clinical outcomes (33). Furthermore, preliminary data suggest that undercontrolled, overcontrolled and low psychopathology classes are associated more strongly with hypothesized risk factors for EDs (e.g., genetic liability, childhood trauma) than are current ED diagnoses (34, 35). This is particularly important because an approach to classifying EDs that distinguishes individuals with respect to putative mechanisms is a crucial first step in the development of novel interventions to target subtype-specific risk and maintaining factors for aberrant eating.

The inclusion of comorbid psychopathology dimensions reflecting the undercontrolled, overcontrolled and low psychopathology classes is a promising direction for future work in ED classification. However, several areas of uncertainty need to be addressed. Critically, because few studies have used identical measures to characterize the undercontrolled, overcontrolled and low psychopathology ED classes (22), it is unclear which dimensions of comorbid psychopathology are most important for distinguishing these groups. In addition, the stability of the classes, as well as their associations with other comorbidity-based subgroups (e.g., dietary-negative affect), are unknown. Finally, although the low comorbid psychopathology group has been documented in numerous reports, the theoretical mechanisms that underlie the expression of ED symptoms in this class are difficult to discern. Future research is needed to determine whether low psychopathology individuals are characterized by dimensions that have not been assessed in previous work, or if an alternative approach to classification would fit this group better.

Model # 3: Neurobiological Dimensions and the Classification of Eating Disorders

A major criticism of descriptive approaches to psychiatric nosology concerns the lack of agreement between current diagnostic categories and findings from clinical neuroscience (3). Although there is little question that individual differences in biological mechanisms are central to the expression of psychiatric symptoms, including EDs, attempts to map diagnostic categories onto neural circuits, and the genetic and epigenetic factors that shape the development of these circuits, have proven problematic (36). This difficulty almost certainly is due, in large part, to a mismatch between the phenotypes studied in neuroscience research and current conceptualizations of psychiatric syndromes. Neuroscience studies generally focus on the elucidation of neural pathways associated with specific cognitive or behavioral processes (36). In contrast, psychiatric syndromes by definition are multifactorial, and individuals assigned to the same diagnostic category (e.g., AN) may vary considerably with respect to presenting symptoms and associated features (e.g., restricting versus binge-eating/purging AN, undercontrolled versus overcontrolled personality traits). Furthermore, certain symptoms (e.g., binge eating, purging, preoccupation with eating, shape or weight) occur in multiple diagnostic groups, which may suggest the presence of unifying biological mechanisms across these syndromes (e.g., all individuals with binge eating may share a particular mechanism regardless of diagnosis) or the possibility of multiple biological pathways to the expression of the same symptom. Thus, efforts to incorporate neurobiologically-salient dimensions into the classification of psychiatric disorders have received increasing emphasis in recent years. In the U.S., this approach is exemplified by the RDoC initiative (3). However, interest in the role of neurobiological dimensions in psychiatric classification is by no means limited to the U.S. [e.g., see Ref. (37)].

Several models of ED psychopathology suggest neurobiological dimensions that may have relevance for classification. For example, Strober's fear conditioning model of AN postulates that dysfunctions in neural systems responsible for regulating emotions, particularly processes involved in fear-based learning, likely contribute to the expression of disordered symptoms in some individuals (38). Similarly, several theorists have speculated that variability in brain serotonin function, ranging from underactivity to overactivity, may help to explain heterogeneity across the ED spectrum with respect to eating behaviors (e.g., binge eating versus extreme dietary restraint) and comorbid psychopathology (e.g., impulsivity versus anxiety and obsessions) [see, e.g., Refs. (39, 40)].

One neurobiological 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 (37). According to a recent comprehensive review (41), impulsivity is “a predisposition toward rapid, unplanned reactions to internal or external stimuli with diminished regard to the negative consequences of these actions to the impulsive individual or to others” (p. 517). And, compulsivity is “a tendency to perform unpleasantly repetitive acts in a habitual or stereotyped manner to prevent perceived negative consequences, leading to functional impairment” (p. 517). Although often viewed as opposites, work in cognitive neuroscience indicates that the relation between impulsivity and compulsivity is complex, and likely involves interactions among several distinct and overlapping neurocognitive mechanisms (41). Examples of these mechanisms include: 1) motor disinhibition (i.e., failure to inhibit prepotent motor responses); 2) inefficient attentional set-shifting (i.e., difficulty moving back and forth among multiple tasks, operations, or mental sets); 3) impaired reversal learning (i.e., inability to adapt behavior in response to changing contingencies); and 4) reward discounting (i.e., overvaluing immediate relative to future rewards) (37, 41, 42).

Several sensitive and domain-specific experimental tasks have been developed to isolate neurocognitive dimensions associated with impulsivity and compulsivity, and an emerging literature has begun to document the neurobiological correlates of performance on these measures. Notably, although there is evidence for overlap in the neurobiological mechanisms associated with performance on particular tasks, several distinctions have been observed. For example, animal research has found a “double dissociation” between reversal learning and attentional set-shifting such that impairments in reversal learning are linked to lesions of the orbitofrontal cortex (OFC), but not the lateral prefrontal cortex (PFC), while impairments in attentional set-shifting result from lesions to the lateral PFC, but not the OFC (41). Double dissociations for neural circuits related to motor inhibition and cognitive set-shifting and delay discounting and reversal learning also have been reported, as well as evidence that distinct neurotransmitters modulate motor inhibition and reversal learning (41).

An approach to classifying EDs that incorporates neurocognitive dimensions of impulsivity and compulsivity is intriguing for several reasons. First, 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. Second, studies using diagnostic interviews and self-report questionnaires consistently have documented the association of impulsive and compulsive disorders and traits to EDs [e.g., Refs. (43–45)]. Third, alterations in several neural circuits (e.g., anterior cingulate cortex, OFC, ventral striatum) and neurotransmitter systems (e.g., serotonin, dopamine) related to impulsivity and compulsivity have been found in individuals with EDs (40, 46). Fourth, neuropsychological research provides support for the notion that neurocognitive dimensions related to impulsivity and compulsivity are salient to EDs. In particular, impairments in motor inhibition, attentional set-shifting and decision-making [a multidimensional construct comprised, in part, of steeper reward discounting and impaired reversal learning (47)] have been reported in ED patients [e.g., Refs. (42, 48, 49)]. It is important to note, however, that this work has relied primarily on clinical neuropsychological tests, which often tap multiple neurocognitive domains. For example, research has shown that perseverative responding on the Wisconsin Card Sorting Test (50), one of the most widely-used measures of set-shifting in the EDs field (42), is driven by two distinct neurocognitive processes: 1) attentional set-shifting and 2) reversal learning (51). Thus, future work utilizing the types of experimental tasks that have been employed in neuroscience research is needed to clarify which neurocognitive dimensions are most relevant to the classification of EDs. Finally, several interventions have shown efficacy in helping individuals to enhance or compensate for neurocognitive impairments associated with impulsivity and compulsivity, leading to improvements in psychiatric symptoms and social functioning [e.g., Refs. (52–54)]. Thus, an

approach to ED classification that incorporates neurocognitive dimensions related to impulsivity and compulsivity could have important implications for the development of novel treatments.

Directions for Future Research

Ultimately, more research will be needed to determine whether a categorical system of ED classification, a dimensional approach or a combination of these models offers the clearest framework for understanding the etiopathology of EDs. To this end, longitudinal studies comparing the temporal stability and predictive validity of various ED classification schemes would be especially valuable. For example, would a comorbidity-based approach or a neurobiological model be more useful than a symptom-based nosology for predicting ED course or treatment response?

Research also is needed to examine the relative utility of incorporating specific dimensional assessments (e.g., symptom severity versus comorbid psychopathology) into the current categorical system of ED classification. The selection of validators will be especially important in this regard, as different dimensional assessments may have utility in different contexts (e.g., etiologic research versus treatment development).

Finally, explicating the associations among dimensions of ED symptomatology, comorbid psychopathology and neurobiology is a critical step in the development of a more valid system of classification. One plausible model would be to consider the ways in which specific neurobiological dimensions might underlie the expression of particular ED symptom dimensions and dimensions of comorbid psychopathology. For example, deficits in motor inhibition have been linked to bulimic symptoms and impulsive and compulsive behaviors (41, 49). Alternatively, documenting within-group heterogeneity across comorbid psychopathology dimensions in individuals carrying the same ED diagnosis or even in those with the same ED symptom dimension could provide clues about variability across the ED spectrum in the neurobiological mechanisms that drive aberrant eating. Indeed, it may be the case someday that individuals with psychiatric disorders, including EDs, are grouped not on the basis of similarities in their presenting symptomatology, but rather by the neurobiological mechanisms that underlie this symptomatology whether the symptoms are eating-related or characterized by some other form of psychiatric disturbance (e.g., depressed mood, heightened anxiety, poor impulse control).

Clinical Utility of Incorporating Dimensions into the Classification of EDs

In addition to suggesting directions for future research, the dimensional models described herein may provide significant information for the clinical management of ED patients across ED diagnoses. Importantly, the models are not mutually exclusive, and indeed, the inclusion of multiple dimensional measures representing several levels of analysis (e.g., ED symptoms, comorbid psychopathology, neurobiology) may have the greatest utility for advancing ED treatment. For example, one could imagine an approach in which dimensional assessment of ED symptoms is used to determine clinical severity in order to guide decisions about treatment setting and intensity, while dimensional assessment of comorbid psychopathology might be used to generate hypotheses about specific factors involved in the expression and maintenance of ED symptoms. Finally, dimensional measures of neurobiological or neurocognitive functioning could help to facilitate the selection of psychological (e.g., cognitive remediation) or pharmacological interventions designed to target biological processes involved in the persistence of ED symptoms.

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References

1. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 3rd ed.. Washington, DC: Author; 1980.
2. World Health Organization. The ICD-10 classification of mental and behavioural disorders: clinical descriptions and diagnostic guidelines. Geneva: World Health Organization; 1992.
3. Insel T, Cuthbert B, Garvey M, Heinssen R, Pine DS, Quinn K, et al. Research domain criteria (RDoC): toward a new classification framework for research on mental disorders. *Am J Psychiatry*. 2010; 167(7):748–751. [PubMed: 20595427]
4. Widiger TA, Samuel DB. Diagnostic categories or dimensions? A question for the Diagnostic and Statistical Manual of Mental Disorders--Fifth Edition. *J Abnorm Psychol*. 2005; 114(4):494–504. Epub 2005/12/15. [PubMed: 16351373]
5. Helzer, JE.; Kraemer, HC.; Krueger, RF.; Wittchen, H-U.; Sirobatka, PJ.; Regier, DA., editors. Dimensional approaches in diagnostic classification: refining the research agenda for DSM-V. Arlington, VA: American Psychiatric Association; 2008.
6. Cross-cutting dimensional assessment in DSM-5. American Psychiatric Association; 2012. DSM-5 Task Force. Available from: <http://www.dsm5.org/proposedrevisions/pages/cross-cuttingdimensionalassessmentindsm-5.aspx>. [cited October 8, 2012]
7. National Institute of Mental Health. National Institute of Mental Health Strategic Plan. Washington, D.C.: U.S. Department of Health and Human Services; 2008.
8. Williamson DA, Gleaves DH, Stewart TM. Categorical versus dimensional models of eating disorders: an examination of the evidence. *Int J Eat Disord*. 2005; 37(1):1–10. [PubMed: 15690459]
9. Wonderlich S, Mitchell JE, Crosby RD, Myers TC, Kadlec K, Lahaise K, et al. Minimizing and treating chronicity in the eating disorders: a clinical overview. *Int J Eat Disord*. 2012; 45(4):467–475. [PubMed: 22271525]
10. Couturier J, Lock J. What is recovery in adolescent anorexia nervosa? *Int J Eat Disord*. 2006; 39(7):550–555. [PubMed: 16791851]
11. Olmsted MP, Kaplan AS, Rockert W. Defining remission and relapse in bulimia nervosa. *Int J Eat Disord*. 2005; 38(1):1–6. [PubMed: 15971233]
12. Fichter MM, Quadflieg N. Long-term stability of eating disorder diagnoses. *Int J Eat Disord*. 2007; 40(Suppl):S61–S66. [PubMed: 17702021]
13. Gordon K, Holm-Denoma J, Smith A, Fink E, Joiner T Jr. Taxometric analysis: introduction and overview. *Int J Eat Disord*. 2007; 40(Suppl):S35–S39. [PubMed: 17607694]
14. Holm-Denoma JM, Richey JA, Joiner TE. The latent structure of dietary restraint, body dissatisfaction, and drive for thinness: a series of taxometric analyses. *Psychol Assess*. 2010; 22(4):788–797. [PubMed: 20822258]
15. Olatunji BO, Broman-Fulks JJ, Ciesielski BG, Zawilinski LL, Shewmaker S, Wall D. A taxometric investigation of the latent structure of eating disorders. *Psychiatry Res*. 2012; 197(1–2):97–102. [PubMed: 22406392]
16. Eating Disorders Work Group. Eating disorders. American Psychiatric Association; 2012. <http://www.dsm5.org/proposedrevisions/pages/eatingdisorders.aspx>. [cited October 8, 2012]
17. Keel PK, Crosby RD, Hildebrandt TB, Haedt-Matt AA, Gravener JA. Evaluating new severity dimensions in the DSM-5 for bulimic syndromes using mixture modeling. *Int J Eat Disord*. 2012 on-line publication ahead of print.
18. Wade TD, Bergin JL, Martin NG, Gillespie NA, Fairburn CG. A transdiagnostic approach to understanding eating disorders. *J Nerv Ment Dis*. 2006; 194(7):510–517. [PubMed: 16840847]
19. Grilo CM, Crosby RD, Masheb RM, White MA, Peterson CB, Wonderlich SA, et al. Overvaluation of shape and weight in binge eating disorder, bulimia nervosa, and sub-threshold bulimia nervosa. *Behav Res Ther*. 2009; 47(8):692–696. [PubMed: 19552897]

20. Hudson JI, Hiripi E, Pope HG Jr, Kessler RC. The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. *Biol Psychiatry*. 2007; 61(3):348–358. [PubMed: 16815322]
21. Jacobi C, Hayward C, de Zwaan M, Kraemer HC, Agras WS. Coming to terms with risk factors for eating disorders: application of risk terminology and suggestions for a general taxonomy. *Psychol Bull*. 2004; 130(1):19–65. [PubMed: 14717649]
22. Wildes JE, Marcus MD. Alternative methods of classifying eating disorders: models incorporating comorbid psychopathology and associated traits. Under review.
23. Stice E, Agras WS. Subtyping bulimic women along dietary restraint and negative affect dimensions. *J Consult Clin Psychol*. 1999; 67(4):460–469. [PubMed: 10450616]
24. Stunkard AJ, Messick S. The three-factor eating questionnaire to measure dietary restraint, disinhibition and hunger. *J Psychosom Res*. 1985; 29(1):71–83. [PubMed: 3981480]
25. Beck AT, Steer RA, Garbin MG. Psychometric properties of the Beck Depression Inventory: twenty-five years of evaluation. *Clin Psychol Rev*. 1988; 8:77–100.
26. Grilo CM, Masheb RM, Wilson GT. Subtyping binge eating disorder. *J Consult Clin Psychol*. 2001; 69(6):1066–1072. [PubMed: 11777111]
27. Stice E, Agras WS, Telch CF, Halmi KA, Mitchell JE, Wilson T. Subtyping binge eating-disordered women along dieting and negative affect dimensions. *Int J Eat Disord*. 2001; 30(1):11–27. [PubMed: 11439405]
28. Stice E, Bohon C, Marti CN, Fischer K. Subtyping women with bulimia nervosa along dietary and negative affect dimensions: further evidence of reliability and validity. *J Consult Clin Psychol*. 2008; 76(6):1022–1033. [PubMed: 19045970]
29. Grilo CM. Subtyping female adolescent psychiatric inpatients with features of eating disorders along dietary restraint and negative affect dimensions. *Behav Res Ther*. 2004; 42(1):67–78. [PubMed: 14744524]
30. Goldschmidt AB, Tanofsky-Kraff M, Goossens L, Eddy KT, Ringham R, Yanovski SZ, et al. Subtyping children and adolescents with loss of control eating by negative affect and dietary restraint. *Behav Res Ther*. 2008; 46(7):777–787. [PubMed: 18460404]
31. Penas-Lledo EM, Loeb KL, Puerto R, Hildebrandt TB, Llerena A. Subtyping undergraduate women along dietary restraint and negative affect. *Appetite*. 2008; 51(3):727–730. [PubMed: 18501473]
32. Thompson-Brenner H, Eddy KT, Franko DL, Dorer DJ, Vashchenko M, Kass AE, et al. A personality classification system for eating disorders: a longitudinal study. *Compr Psychiatry*. 2008; 49(6):551–560. [PubMed: 18970903]
33. Wildes JE, Marcus MD, Crosby RD, Ringham RM, Dapelo MM, Gaskill JA, et al. The clinical utility of personality subtypes in patients with anorexia nervosa. *J Consult Clin Psychol*. 2011; 79(5):665–674. [PubMed: 21767000]
34. Steiger H, Richardson J, Schmitz N, Joobar R, Israel M, Bruce KR, et al. Association of trait-defined, eating-disorder sub-phenotypes with (biallelic and triallelic) 5HTTLPR variations. *J Psychiatr Res*. 2009; 43(13):1086–1094. [PubMed: 19383563]
35. Westen D, Harnden-Fischer J. Personality profiles in eating disorders: rethinking the distinction between axis I and axis II. *Am J Psychiatry*. 2001; 158(4):547–562. [PubMed: 11282688]
36. Sanislow CA, Pine DS, Quinn KJ, Kozak MJ, Garvey MA, Heinssen RK, et al. Developing constructs for psychopathology research: Research Domain Criteria. *J Abnorm Psychol*. 2010; 119(4):631–639. [PubMed: 20939653]
37. Robbins TW, Gillan CM, Smith DG, de Wit S, Ersche KD. Neurocognitive endophenotypes of impulsivity and compulsivity: towards dimensional psychiatry. *Trends Cogn Sci*. 2012; 16(1):81–91. [PubMed: 22155014]
38. Strober M. Pathologic fear conditioning and anorexia nervosa: on the search for novel paradigms. *Int J Eat Disord*. 2004; 35(4):504–508. [PubMed: 15101066]
39. Steiger H, Bruce KR. Phenotypes, endophenotypes, and genotypes in bulimia spectrum eating disorders. *Can J Psychiatry*. 2007; 52(4):220–227. [PubMed: 17500302]
40. Kaye W. Neurobiology of anorexia and bulimia nervosa. *Physiol Behav*. 2008; 94(1):121–135. [PubMed: 18164737]

41. Fineberg NA, Potenza MN, Chamberlain SR, Berlin HA, Menzies L, Bechara A, et al. Probing compulsive and impulsive behaviors, from animal models to endophenotypes: a narrative review. *Neuropsychopharmacology*. 2010; 35(3):591–604. [PubMed: 19940844]
42. Roberts ME, Tchanturia K, Stahl D, Southgate L, Treasure J. A systematic review and meta-analysis of set-shifting ability in eating disorders. *Psychol Med*. 2007; 37(8):1075–1084. [PubMed: 17261218]
43. Altman SE, Shankman SA. What is the association between obsessive-compulsive disorder and eating disorders? *Clin Psychol Rev*. 2009; 29(7):638–646. [PubMed: 19744759]
44. Cassin SE, von Ranson KM. Personality and eating disorders: a decade in review. *Clin Psychol Rev*. 2005; 25(7):895–916. [PubMed: 16099563]
45. Root TL, Pisetsky EM, Thornton L, Lichtenstein P, Pedersen NL, Bulik CM. Patterns of comorbidity of eating disorders and substance use in Swedish females. *Psychol Med*. 2010; 40(1):105–115. [PubMed: 19379530]
46. Frank GK, Kaye WH. Current status of functional imaging in eating disorders. *Int J Eat Disord*. 2012; 45(6):723–736. [PubMed: 22532388]
47. Clark L, Cools R, Robbins TW. The neuropsychology of ventral prefrontal cortex: decision-making and reversal learning. *Brain Cogn*. 2004; 55(1):41–53. [PubMed: 15134842]
48. Brogan A, Hevey D, Pignatti R. Anorexia, bulimia, and obesity: Shared decision making deficits on the Iowa Gambling Task (IGT). *J Int Neuropsych Soc*. 2010; 16(4):711–715.
49. Marsh R, Steinglass JE, Gerber AJ, Graziano O'Leary K, Wang Z, Murphy D, et al. Deficient activity in the neural systems that mediate self-regulatory control in bulimia nervosa. *Arch Gen Psychiatry*. 2009; 66(1):51–63. [PubMed: 19124688]
50. Heaton, RK.; Chelune, GJ.; Talley, JL.; Kay, GG.; Curtiss, G. Wisconsin Card Sorting Test manual: revised and expanded. Lutz, FL: Psychological Assessment Resources, Inc.; 1993.
51. Rogers RD, Andrews TC, Grasby PM, Brooks DJ, Robbins TW. Contrasting cortical and subcortical activations produced by attentional-set shifting and reversal learning in humans. *J Cogn Neurosci*. 2000; 12(1):142–162. [PubMed: 10769312]
52. Wykes T, Huddy V, Cellard C, McGurk SR, Czobor P. A meta-analysis of cognitive remediation for schizophrenia: methodology and effect sizes. *Am J Psychiatry*. 2011; 168(5):472–485. [PubMed: 21406461]
53. Grant JE, Chamberlain SR, Odlaug BL, Potenza MN, Kim SW. Memantine shows promise in reducing gambling severity and cognitive inflexibility in pathological gambling: a pilot study. *Psychopharmacology*. 2010; 212(4):603–612. [PubMed: 20721537]
54. Potenza MN, Sofuoglu M, Carroll KM, Rounsaville BJ. Neuroscience of behavioral and pharmacological treatments for addictions. *Neuron*. 2011; 69(4):695–712. [PubMed: 21338880]