Evaluation of the DSM-5 Eating Disorder Severity Indices of Bulimic Syndromes

BY

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Abstract

A major change to the *DSM-5* was the integration of a new severity rating system, ranging from mild to extreme, with key parameters for each eating disorder. However, few studies have examined the clinical utility of these new severity indices. The aim of this project was to identify variables that predict psychosocial impairment in a community sample of individuals with eating disorders in order to inform future diagnostic definitions of severity. Participants were individuals with eating disorders (*N*=189; 19.9% men) recruited from two Midwestern communities. Participants completed the Eating Pathology Symptoms Inventory (EPSI), Structured Clinical Interview for *DSM-IV* Axis I Disorders (SCID-I) adjusted for *DSM-5* criteria, NEO Five-Factor Inventory-3 (NEO-FFI-3), and the World Health Organization Disability Assessment Schedule 2.0 (WHO-DAS 2.0). The Bayesian Information Criterion (BIC) was used to compare the fit of path models to the data. Individual model fit was assessed using overall model chi-square, comparative fit index (CFI), Tucker-Lewis Fit Index (TFI), and root-mean-square-error of approximation (RMSEA). Results provided some support for current *DSM-5* severity indicators for BN and BED. Findings indicated limited support for *DSM*-5's current severity indices. Two alternative methods for determining eating disorder severity, using binge eating and restricting frequency as predictors of psychosocial dysfunction, were identified. However, results also suggested that future evaluation of severity indices might benefit from integrating mood, anxiety, and eating disorder symptom based models of severity.

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Evaluation of the DSM-5 Eating Disorder Severity Indices of Bulimic Syndromes: What are the Best Indicators of Dysfunction?

Epidemiological research indicates that eating disorders are highly prevalent in the United States (Allen, Byrne, Oddy, & Crosby, 2013; Hudson, Hiripi, Pope, & Kessler, 2007; Kendler et al., 1991; Stice, Marti, & Rohde, 2013; Walters & Kendler, 1995). Indeed, it is estimated that 28.9% of male and female adolescents will be affected by an eating disorder at some time in their life (Smink, van Hoeken, Oldehinkel, & Hoek, 2014). Eating disorders are associated with significant impairments in social adjustment and quality-of-life (Doll, Petersen, & Stewart-Brown, 2005; Mond, Hay, Rodgers, Owen, & Beumont, 2005; Padierna, Quintana, Arostegui, Gonzalez, & Horcajo, 2000; Rie, Noordenbos, & Furth, 2005). Despite the seriousness of eating disorders, few studies exist to identify predictors of psychosocial impairment among individuals with eating disorders (González-Pinto et al., 2004; Hay, 2003; Latner, Vallance, & Buckett, 2008; Vallance, Latner, & Gleaves, 2011). Thus, the purpose of the current study was to identify variables that predict psychosocial impairment in a community sample of individuals with eating disorders to inform future diagnostic definitions of severity.

Definitions of DSM-5 Eating Disorders

The *Diagnostic and Statistical Manual of Mental Disorders*, 5th *edition* (DSM-5; APA, 2013) recognizes four eating disorders: anorexia nervosa (AN), bulimia nervosa (BN), binge eating disorder (BED), and other specified feeding or eating disorder (OSFED). AN is a self-starvation disorder in which individuals restrict energy intake, leading to a significantly low body weight in the context of age, sex, developmental trajectory, and physical health. BN is defined by recurrent episodes of binge eating (e.g., eating a large amount of food in a short period of time and experiencing a loss-of-control over eating) and inappropriate compensatory behaviors (ICBs)

to prevent weight gain. ICBs include self-induced vomiting; fasting/restricting; excessive exercise; and the misuse of laxatives, diuretics, suppositories, or enemas. BED is characterized by recurrent episodes of binge eating in the absence of ICBs. Finally, presentations in which symptoms of a feeding or eating disorder cause clinically significant distress or impairment, but do not meet full criteria for any of the previously mentioned eating disorders are categorized into 'other specified feeding and eating disorder' (OSFED). For example, normal weight individuals who engage in recurrent self-induced vomiting in the absence of objectively large binge-eating episodes would potentially meet criteria for an OSFED. OSFED represents one of the largest categories of eating disorders, with some studies indicating that over 50% of individuals that meet criteria for an eating disorder have an OSFED diagnosis (Keel, Brown, Holm-Denoma, & Bodell, 2011; Machado, Gonçalves, & Hoek, 2013).

Changes from *DSM-IV-TR* to *DSM-5*

The transition from the *Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revision* (*DSM-IV-TR*; APA, 2000) to *DSM-5* led to several changes within eating disorders. Changes included the recognition of BED as an official diagnostic category; revisions to the diagnostic criteria for AN and BN; and the merging of feeding disorders of childhood and eating disorders into one section (Call, Walsh, & Attia, 2013). Another major change to the *DSM-5* was the integration of new diagnostic dimensions (e.g., specifiers, subtypes, severity ratings, and cross-cutting symptom assessments) (Regier, Kuhl, & Kupfer, 2013). The inclusion of these diagnostic dimensions aimed to help clinicians capture levels of a disorder that might otherwise be ignored by the *DSM*'s categorical approach, as well as improve treatment planning (Jones, 2012; Regier et al., 2013).

With regard to the eating disorders, the *DSM-5* introduced a new severity rating system, which ranged from mild to extreme, with key severity parameters for each eating disorder. The severity index for AN was defined based an individual's weight using body mass index (BMI), which takes into account weight relative to height (kg/m^2): mild (BMI $\geq$ 17 kg/m^2), moderate (BMI between 16 and 16.99 kg/m^2), severe (BMI between 15 and 15.99 kg/m^2), and extreme (BMI < 15 kg/m^2). BN was defined by the frequency of ICBs per week: mild (1-3 ICBs/week), moderate (4-7 ICBs/week), severe (8-13 ICBs/week), and extreme (14 or more ICBs/week). The severity specifier for BED was based on the frequency of binge-eating episodes per week: mild (1-3 episodes/week), moderate (4-7 episodes/week), severe (8-13 episodes/week), and extreme (14 or more episodes/week). To date, there has been no statement from the *DSM-5* Eating Disorders Task Force supporting the inclusion of the previously described severity ratings for eating disorders. However, as I describe below, some researchers have examined the utility of the new eating disorder severity ratings in the *DSM-5*.

Empirical Evidence for *DSM-5* Eating Disorder Severity Index

Grilo, Ivezaj, and White examined the utility of the *DSM-5* severity criteria for BED (2015a, 2015b) and BN (Grilo et al., 2015c) in community and clinical samples using a general linear model analysis of variance. In community samples, partial eta-squared (η^2) effect sizes were calculated to determine the proportion of variance in the criteria measures [The Questionnaire for Eating and Weight Patterns – Revised (QEWP-R; Yanovski, 1993); The Eating Disorder Examination Questionnaire (EDE-Q; Fairburn & Beglin, 2008); and The Beck Depression Inventory (BDI; Beck & Steer, 1987)] that were accounted for by group membership in the severity categories (i.e., mild, moderate, severe, extreme). Participants included community men and women who met *DSM-5* criteria for BED or BN. Grilo et al. (2015b, 2015c)

compared self-reported eating-disorder and depressive symptoms between groups. Individuals who were classified into the "moderate severity" group for BED (based on their self-reported binge-eating frequency) reported significantly greater eating-disorder psychopathology than individuals who met criteria for "mild" severity (Grilo et al., 2015b). Individuals who were grouped within the "extreme" severity group for BN reported significantly higher levels of eating concerns than those in the "severe," "moderate," and "mild" groups (Grilo et al., 2015c). However, the effect sizes (partial η^2) for these studies were small, ranging from 0.014 to 0.108 for individuals meeting criteria for BED (Grilo et al., 2015b) and 0.031 to 0.081 for individuals meeting criteria for BN (Grilo et al., 2015c). In a clinical sample of adults meeting *DSM-5* criteria for BED, eating disorder and depressive symptoms collected from semi-structured clinical interviews [The Structured Clinical Interview for *DSM-IV* Axis I Disorders (SCID; First, Spitzer, Gibbon, & Williams, 2001); The Eating Disorder Examination Interview (EDDI; Cooper & Fairburn, 1987); and the Beck Depression Inventory (BDI; Beck & Steer, 1987)] were compared by group membership in the severity categories (i.e., mild, moderate, severe, extreme) (Grilo et al., 2015a). Similarly, individuals who were grouped within the "extreme" severity group for BED reported significantly more eating disorder psychopathology that those within the "moderate" and "mild" severity groups and effect sizes (partial η^2) ranged from .001 to .751. These three studies provided small-sized effects that supported the *DSM-5*'s severity criteria for BED and BN. Specifically, Grilo et al.'s results indicated that participants classified into the more severe categories had greater eating-disorder psychopathology than individuals who were grouped into milder categories. However, Grilo et al.'s findings are not surprising given that, by definition, those with more eating-disorder symptoms were classified as belonging to a more pathological severity group than those with fewer symptoms.

Unlike bulimic syndromes, few studies have examined the utility of the *DSM-5* severity ratings in individuals with AN. Research has shown that lower BMI (the proposed severity index for AN) has a strong negative association with mortality among individuals with AN (Franko et al., 2013; Nielsen et al., 1998; Rosling, Sparén, Norring, & von Knorring, 2011). Despite these findings, to my knowledge, no studies have found negative associations between BMI and eating-disorder psychopathology, and other research indicates that BMI and eating-disorder psychopathology are positively correlated in individuals with AN (Berner, Shaw, Witt, & Lowe, 2013). Thus, additional research is needed to clarify the clinical utility of the *DSM-5* severity index for AN.

Alternative Methods for Assessing Eating Disorder Severity

Recently, researchers have suggested alternative severity indices for eating disorders, including overvaluation of shape and weight (Grilo et al., 2015a, 2015b) (i.e., basing one's self-esteem and sense of self-worth on body weight and shape), dietary restriction (De Young et al., 2013), number of purging methods (Edler, Haedt, & Keel, 2007) (e.g., self-induced vomiting and laxative use), and total eating-disorder symptom count (Keel, Crosby, Hildebrandt, Haedt-Matt, & Gravener, 2013). For example, Keel et al. (2013) used factor-mixture modeling to determine whether bulimic syndromes (anorexia nervosa binge-purge subtype, bulimia nervosa, and binge eating disorder) were best conceptualized as categorical or continuous. Analyses tested three conceptualizations of bulimic syndromes: (1) multiple categories with distinct severity dimensions (as proposed in *DSM-5*); (2) a single category with multiple severity dimensions; or (3) a combination of categories with one or more severity dimensions. Results supported a hybrid model with three categorically distinct diagnostic categories and a single latent severity dimension for bulimic syndromes. Specifically, Keel et al. concluded that severity might be best

captured by the total number of symptoms endorsed across bulimic diagnoses rather than separate severity dimensions for each diagnosis.

However, one problem with previous studies is that there have either been no external criteria by which to judge the clinical utility of the severity model (e.g., Keel et al., 2013) or the criterion was another measure of eating pathology (Berner et al., 2013; Grilo et al., 2015a, 2015b, 2015c). Moreover, although Keel et al. (2013) tested whether severity was best represented by a single or multiple latent dimension(s), there were a limited number of variables included in the model, and several viable alternative models have not been tested.

Other research has indicated that duration of illness may impact eating-disorder recovery and quality-of-life (Padierna, Quintana, Horcajo, Madrazo, & Ecenarro, 2004). Specifically, individuals who suffered with an eating disorder for over five years were less likely to benefit from treatment and reported a lower quality-of-life than individuals with shorter duration of illnesses. However, conflicting results have also been found showing that duration of eating disorder does not significantly predict poor quality-of-life (Bamford & Sly, 2010). Nonetheless, it is possible that the influence of individual eating-disorder features on impairment varies over the course of an eating disorder; thus, additional research is needed to clearly evaluate the impact of duration of illness on eating disorder "severity."

Variables That May Influence Eating Disorder Severity Prediction

Some studies that examined the utility of *DSM-5* severity indices (and proposed alternative methods by which to judge severity) did not control for common confounding variables shown to impact eating-disorder severity (Berner et al., 2013; De Young et al., 2013; Edler et al., 2007; Grilo et al., 2015a, 2015b, 2015c; Keel, Crosby, Hildebrandt, Haedt-Matt, & Gravener, 2013). Neuroticism is a predisposition to experience strong negative emotional

reactions to stress (McCrae, 1990) and is associated with eating disorder symptomatology (Cassin & von Ranson, 2005; Schmitz, Kugler, & Rollnik, 2003; Widiger & Trull, 1992). Specifically, individuals with eating disorders have reported higher levels of neuroticism than normal controls (Brewerton, Hand, & Bishop Jr, 1993; Diaz-Marsa, Carrasco, & Saiz, 2000; Podar, Jaanisk, Allik, & Harro, 2007). In addition, research has identified neuroticism or negative affect to precede binge-eating episodes (Chua, Touyz, & Hill, 2004; Davis & Jamieson, 2005; Haedt-Matt & Keel, 2011; Rush, Becker, & Curry, 2009; Vanderlinden, Grave, Vandereycken, & Noorduin, 2001). The association between dysregulated affect and binge eating has been described by the Affect Regulation Model, which posits that individuals binge eat to reduce negative affect (see Gross, 2007 for comprehensive review). Additionally, a recent study showed that the interaction of neuroticism and negative affect predicted weekly fluctuations in binge-eating episode frequency (Zander & De Young, 2014). Neuroticism's impact on binge-eating frequency raises concerns when considering the new severity index for BED, which is based on the number of binge episodes per week. In order to better test the utility of the new *DSM-5* severity indicators, controlling for levels of neuroticism is warranted.

Important variables that should also be controlled for through statistical analyses when examining *DSM-5* eating disorder severity indices include psychiatric medication use and treatment seeking. The main goal of treatment, whether pharmacological or psychological, is to decrease symptomatology and increase chances for recovery. To obtain non-confounded models of eating-disorder severity, it is important to statistically control for the effect of treatment.

Proposed Approach for Assessing Eating Disorder Severity Indices

Psychosocial dysfunction is defined by significant impairment in a variety of life domains caused by psychopathology. Psychosocial dysfunction may represent a clinically useful criterion

for assessing "severity" in eating disorders. The Global Assessment of Functioning Scale (GAF) was removed from *DSM-5* based on empirical research that showed that the validity of GAF scores correlated more strongly with symptom severity than psychosocial impairment (Smith et al., 2011; Soderberg, Tungstrom, & Armelius, 2014; Von Korff, Andrews, & Delves, 2011). The *DSM-5* currently recommends that mental health professionals should utilize the World Health Organization Disability Assessment Schedule 2.0 (WHO-DAS 2.0; Üstün et al., 2010) to assess global functioning and impairment because the WHO-DAS 2.0 provides a more reliable and valid index of psychosocial functioning based on activities, behaviors, and participation in life and society.

To address the limited empirical support for the *DSM*-5's eating disorder severity indices, the goal of the current study was to evaluate the *DSM-5's* severity indices for eating disorders, as well as other potential indices of severity across time in a community-recruited sample of individuals with bulimic syndromes using a reliable and valid measure of functioning and impairment. Based on the past literature, two potential indices of severity for eating disorders, outside of *DSM-5*'s defined indices, were identified, including: body dissatisfaction and duration-of-illness. Based on prior research (Grilo et al., 2015b, 2015c), I hypothesized that symptoms that reflected body dissatisfaction or overvaluation of weight or shape would represent the strongest predictor of psychosocial dysfunction. In support of this hypothesis, past research has shown that the frequency of body checking (e.g., repeatedly trying on different outfits because one did not like how one looked, pinching one's body fat, weighing oneself, looking at one's reflection in mirrors or storefront windows, etc.) significantly predicted quality-of-life impairment (Latner, Mond, Vallance, Gleaves, & Buckett, 2012) and that weight and shape concerns were significant unique predictors of impairment in college students with clinically

significant disordered eating (Hovrud & De Young, 2015). I hypothesized that duration-of-illness would also emerge as a strong predictor of psychosocial impairment, given that past studies have shown individuals with long-term eating disorders report lower quality-of-life scores than those with shorter duration (Padierna et al., 2004). I also hypothesized that current *DSM-5* severity indices would not significantly predict psychosocial impairment given the limited supporting research that used external criteria to judge the clinical utility of these severity models (Grilo et al., 2015a, 2015b, 2015c). Finally, I hypothesized that psychosocial functioning would mediate the association between disordered eating symptoms (independent variables) of the same construct from Time 2 to Time 3, given that research has shown that disordered eating behaviors are associated with impairments in quality-of-life (Doll et al., 2005; Mond et al., 2005; Padierna et al., 2000; Rie et al., 2005).

Methods

Participants

Participants included individuals enrolled in an ongoing longitudinal research study (PI: Kelsie Forbush). Participants (N=189; 19.9% men) were recruited from two Midwestern communities through filers, newspaper advertisements, bus advertisements, and from mass emails sent to students, faculty, and staff at two large Midwestern universities. Participants were included if they were ≥ 14 years of age; fluent English speakers; and if they met *DSM-5* criteria for AN, BN, or BED, or if they met two or more symptoms of an eating disorder that caused clinically significant impairment (i.e., an 'other specified eating disorder'). Clinically significant impairment was considered present if the participant had a score ≥ 16 on the Clinical Impairment Assessment Questionnaire (CIA; Bohn et al., 2008), because a cut score of 16 has been shown to have maximal sensitivity and specificity for distinguishing individuals with eating disorders from

individuals without eating disorders (Bohn et al., 2008). Eating disorder diagnoses were confirmed in-person using semi-structured clinical interviews.

Participants were excluded if they were younger than 14 years old; had a diagnosis of an intellectual disability or neurological disorder; or had a body mass index (BMI) ≤ 14. Participant demographics and diagnostic data for baseline are shown in Table 1. Checks for inconsistent responding were included within the study (e.g., endorsing binge-eating behavior on one measure and denying binge eating on another measure). Data were omitted for two participants at Time 2 and two participants at Time 3 due to invalid responding. This resulted in valid data for 189 participants at Time 1, 100 participants at Time 2, and 80 participants at Time 3.

The mean age (SD) of the sample was 25.15 (8.94), and participants had a mean (SD) BMI of 26.67 (7.71) at baseline. The majority of sample was Caucasian (71.9%). Other races/ethnicities included were: Hispanic or Latino/a (9.5%), African American (6.3%), Asian (17.9%), Native-American/Alaskan Native (1.0%), Native-Hawaiian/Pacific Islander (0.5%), Multi-Racial (4.7%), or any other race or ethnicity (3.1%). Consistent with prior community and clinically based studies, the most common diagnostic categories for current eating disorders were OSFED (45.0%) and BN (42.4%). Other current eating disorders included were AN (3.1%) and BED (8.4%). The majority of current OSFED cases were subthreshold forms of BN (28.3%). The mean duration of illness (SD) was 7.68 (8.05) years.

Procedures

All participants completed written informed consent prior to each assessment, and study procedures were approved by the appropriate university institutional review boards.

Baseline. Information about symptoms of *DSM-5* eating, mood, and anxiety disorders were collected by self-reports and semi-structured clinical interviews (see list of measures on

next page). Height and weight measurements were collected using self-reported data and verified using digital scales and wall-mounted stadiometers. Self-reported and objectively measured height ($r = 0.987$; $p < .01$) and weight ($r = 0.830$; $p < .01$) measurements were significantly correlated at baseline. Baseline sessions took approximately three to four hours to complete and participants were compensated $70.00 for participation.

Follow-ups. Two self-report follow-up online surveys were administered via Qualtrics, an online survey system, to collect symptom information about *DSM-5* eating, mood, and anxiety disorders, and psychosocial functioning. Height and weight were based on self-reported data at follow-up. Participants received a link to each survey via email. The second study link was e-mailed to participants 6-9 months after baseline and the third student link was sent 3-6 months after the submission of their second survey. Participants who completed the baseline assessment (and gave permission to be re-contacted) were sent up to three reminder e-mails to encourage them to participate in the follow-up surveys. Each online survey took approximately twenty minutes to complete and participants were compensated $10.00 for the completion of the first follow-up (Time 2) and $15.00 for participation in the second survey (Time 3).

Measures

The Eating Pathology Symptoms Inventory (EPSI; Forbush et al., 2013) is a self-report measure of eating-disorder psychopathology and includes eight scales that assess Body Dissatisfaction, Binge Eating, Restricting, Cognitive Restraint, Muscle Building, Purging, Excessive Exercise, and Negative Attitudes Toward Obesity. Body Dissatisfaction assesses negative feelings about an individual's body weight or shape (e.g., I *thought my butt was too big*). Binge Eating assesses symptoms of objective binge eating (e.g., *I ate until I was uncomfortably full*). Restricting assesses successful caloric restriction in order to lose weight

(e.g., *I skipped two meals in a row*). Cognitive Restraint assesses attempts to restrict intake, regardless of whether these attempts are successful (e.g., *I tried to exclude "unhealthy" foods from my diet*). Muscle Building assesses negative feelings about an individual's muscle size and attempts to increase muscle mass (e.g., *I thought about taking steroids as a way to get more muscular*). Purging assesses inappropriate compensatory behaviors designed to expel calories from the body (e.g., *I made myself vomit in order to lose weight*). Excessive Exercise assesses pathological exercising behaviors (e.g., *I pushed myself extremely hard when I exercised*). Negative Attitudes toward Obesity assesses individuals' negative thoughts about individuals with overweight or obesity (e.g., *I was disgusted by the sight of an overweight person wearing tight clothes*).

Research conducted across a range of samples has indicated that EPSI scale scores have strong internal consistency, convergent validity, discriminant validity, and test-retest reliability (Forbush, Wildes, & Hunt, 2014; Forbush et al., 2013). The self-report version of the EPSI was completed at baseline and administered at each follow-up session. The EPSI was used to examine five predictor variables, including the total scale score for Body Dissatisfaction, Binge Eating, Restricting, Purging, and Excessive Exercise from the self-report version.

Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I; First et al., 2001) is a semi-structured clinical interview to assess and diagnose *DSM-IV* disorders (APA, 2000). At baseline, participants were interviewed using the eating-disorders modules of the SCID-I to assess current and lifetime symptoms of eating disorders. Skip-out rules were ignored to collect information on all clinically significant symptoms of an eating disorder that might have otherwise been missed. The diagnostic criteria for the eating disorders module were adjusted to reflect changes to *DSM-5*.

Interviewers included trained master's- and bachelor's-level assessors. Assessors were trained using didactics, role-playing, and observation. Assessors were required to complete an annual 16-hour training retreat split into three days; complete readings on the development of the SCID-I; and score two audiotaped interviews from the lab with ≥ 80% reliability. Assessors were required to attend weekly consensus meetings discuss eating disorder diagnoses, ask questions, and prevent interviewer drift. Inter-rater reliability ratings based on audiotape ratings were conducted on a random sample (n = 22) of baseline SCID-I interviews. All eating disorder diagnoses had Conger's kappa values ranging from .89 to 1.00, indicating excellent inter-rater reliability. Information collected at baseline from the SCID-I was used to determine eating disorder status at baseline.

NEO Five-Factor Inventory-3 (NEO-FFI-3; Costa & MacCrae, 1992) is a 60 item self-report measure of personality with five domains, including Neuroticism, Extraversion, Openness to Experience, Agreeableness, and Conscientiousness. Only one domain, Neuroticism, was used in the current study. The Neuroticism facet is assessed by twelve items, which have been shown to have acceptable (α = .79) internal consistency. The NEO-FFI-3 is a widely used personality inventory with substantial empirical data to support its construct and criterion-related validity (Costa & MacCrae, 1992). The NEO-FFI-3 Neuroticism facet was included as a covariate in analyses to control for levels of Neuroticism at each time point.

Body Mass Index (BMI) was calculated for each participant at baseline using digital scales and wall-mounted stadiometers. BMI was calculated by dividing each participants' weight in kilograms by the square of his or her height in meters (kg/m^2). The National Institutes of Health (1998) divides BMI into four categories: (1) underweight is defined as a BMI less than 18.5; (2) normal weight is defined as a BMI between 18.5 and 24.99; (3) overweight is defined

as a BMI between 25.0 and 29.99; and (4) obesity is defined as a BMI of 30 or higher. BMI was calculated for follow-up sessions based on self-reported height and weight measurements. BMI was used as a predictor variable in the analyses.

World Health Organization Disability Assessment Schedule 2.0 (WHO-DAS 2.0; Üstün et al., 2010) is a self-report assessment that measures functioning and disability in major life domains within the last month. The WHO-DAS 2.0 includes 36 items that cover six domains including Cognition, Mobility, Self-Care, Getting Along, Life Activities, and Participation. Cognition assesses individuals' ability to understand and communicate, including concentration, memory, and maintaining conversations. Mobility assesses individuals' ability to move and get around, such as standing for long periods or walking long distances. Self-Care assesses individuals' ability to attend to personal hygiene, dressing and eating, and to live alone. Getting Along assesses individuals' ability to interact with other people, such as making new friends. Life Activities assesses individuals' ability to carry out responsibilities at home, work, and school. Participation assesses individuals' ability to engage in community, civil, and recreational activities. Instructions were modified to focus on the impact of individuals' eating disorder on psychosocial functioning that might have otherwise been missed.

Research conducted across a range of samples, including members of the general population in good health, individuals with physical disorders, individuals with chronic conditions, individuals with mental or emotional disorders, and individuals with problems related to drug or alcohol use, has demonstrated that WHO-DAS 2.0 domains have good internal consistency, convergent validity, and test-retest reliability (Garin et al., 2010; Üstün et al., 2010). The WHO-DAS 2.0 was completed at both follow-up time points. The WHO-DAS 2.0 summed

score was used as the outcome variable in the analyses. As indicated above, the WHO-DAS 2.0 is now being used instead of the GAF for *DSM*-5 to assess psychosocial dysfunction.

Statistical Analysis

Descriptive statistics, independent samples *t*-tests, and simple regression models were analyzed using SPSS Version 24 (IBM, 2013). Independent samples *t*-tests were used to test between-group differences on BMI and EPSI scales at each time point among individuals in different weight categories, seeking treatment, and use of psychiatric medication. Simple regression models were used to test the direct effect of control variables on psychosocial dysfunction.

Additional data analyses were analyzed using Mplus Version 7.31 (Muthén & Muthén, 1998-2012) using full information maximum likelihood estimation (MLR) to recover missing data. An autoregressive, longitudinal, cross-lagged panel model in a structural equation modeling (SEM) framework was used to identify significant predictors of psychosocial dysfunction among individuals with eating disorders (see Figure 1). This method has been shown to work well in large (e.g., three or more variables across three or more time points) longitudinal data sets (Burkholder & Harlow, 2003). Independent variables included BMI, duration-of-illness, and EPSI Body Dissatisfaction, Binge Eating, Restricting, Purging, and Excessive Exercise scales (see Table 2 for more information). Given that the *DSM-5* severity indices developed for each eating disorder were based on different symptoms, measures of BMI were tested for AN, EPSI Restricting, Purging, and Excessive Exercise scales were tested as ICBs for BN, and the EPSI Binge Eating scale was tested for BED. Control variables included Neuroticism, the number of months between assessments, history of psychological treatment, and use of psychiatric medication. Control variables were included in separate autoregressive cross-lagged panel

models. Modification indices, which identify areas of localized model strain, were used to improve model fit by correlating relevant items when theoretically supported. Given that multiple predictor variables are included in the models, standardized regression coefficients were used to determine the strongest predictor variable. The Bayesian Information Criterion (BIC) was used to compare model fit. When comparing BIC values, lower BIC values indicate a better fitting model, as lower values balance goodness-of-fit with model parsimony (Muthén, Muthén, & Asparouhov, 2016). Individual model fit was determined by assessing the (a) overall model chi-square; (b) comparative fit index (CFI); (c) Tucker-Lewis Fit Index (TLI); and (d) root-mean-square error of approximation (RMSEA), using criteria established by Hu and Bentler (1999). Hu and Bentler (1999) concluded that a cutoff value close to .95 for CFI and TLI, and a cutoff value close to .06 for RMSEA suggest relatively good fit of the hypothesized model and the observed data.

Results

Descriptive statistics are presented in Table 3. Independent samples *t*-tests indicated that overweight or obese participants reported significantly higher levels of BMI and binge eating; whereas, normal weight participants reported higher levels of restricting and excessive exercise. Participants using psychiatric medication reported significantly higher levels of binge eating, body dissatisfaction, and purging than individuals not using medication. Independent samples *t*-tests also indicated that treatment-seeking participants reported significantly higher levels of body dissatisfaction, whereas non-treatment seeking participants reported higher levels of excessive exercise.

Skew and kurtosis were also examined for all study variables and are presented in Table 4. EPSI Purging, Restricting, Body Dissatisfaction, and Excessive Exercise scales were non-

normally distributed throughout the study. Variables that reported significant skew and/or kurtosis were log-transformed to reduce skew.

The direct effect of control variables on psychosocial dysfunction was examined. Control variables included Neuroticism, the number of months between assessments, history of psychological treatment, and use of psychiatric medication. Regression analyses revealed a significant positive association between psychosocial dysfunction and Neuroticism at baseline (β = 1.20, t = 3.04, p = 0.004) and Time 2 (β = 1.72, t = 4.84, p = 0.000), as well as a significant negative association between psychosocial dysfunction and time between assessments at Time 3 (β = -13.13, t = -3.67, p = 0.001). Results did not reveal any other significant direct effects of control variables on psychosocial dysfunction.

Standardized regression coefficients were used to determine the strongest predictor variable in each model. EPSI Binge Eating at Time 1 appeared as the strongest predictor variable of psychosocial dysfunction at Time 2 in Model 2 (β = 0.451; p = 0.025), Model 3 (β = 0.544; p = 0.000), Model 4 (β = 0.377; p = 0.053), and Model 5 (β = 0.402; p = 0.022). In Model 1, EPSI Restricting at Time 1 appeared as the strongest predicter of psychosocial dysfunction at Time 2 (β = 0.297; p = 0.009). EPSI Body Dissatisfaction at Time 2 appeared as the strongest predictor variable of psychosocial dysfunction at Time 3 in Model 1 (β = 0.298; p = 0.001), Model 3 (β = 0.147; p = 0.310), Model 4 (β = 0.142; p = 0.082), and Model 5 (β = 0.123; p = 0.103). In Model 2, BMI at Time 2 was the strongest predictor of psychosocial dysfunction at Time 3 (β = 0.156; p = 0.235).

The Bayesian Information Criterion (BIC), overall model chi-square, CFI, TLI, and RMSEA are reported in Table 5 for all five models. BIC was used to compare the relative fit of path models, which differed on control variables included. Individual model fit was determined

by assessing overall model chi-square, CFI, TLI, and RMSEA. Modification indices in all models, except Model 1, indicated one theoretically supported area of model strain, with modification indices ranging from 10.19 to 15.78 in these models. Because binge eating and purging behaviors often co-occur in individuals with certain eating disorders, it made theoretical sense to allow correlations between these variables. Thus, except for Model 1, the EPSI Binge Eating scale was correlated with EPSI Purging in all models.

Model 1, which omitted control variables, demonstrated sub-optimal fit to the data based on the model's chi-square and RMSEA, and reported the lowest BIC value in all five models (see Table 5, Model 1). However, the model's CFI and TLI approached Hu and Bentler's (1999) recommended cut-off value. Model 1 results showed that the EPSI Restricting (p = .012) scale at Time 1 significantly predicated psychosocial dysfunction at Time 2 and the EPSI Body Dissatisfaction at Time 2 significantly predicted psychosocial dysfunction at Time 3 (p = .001). Duration of illness at Time 2 also significantly predicted psychosocial dysfunction at Time 3 (p = .033).

Model 2, which controlled for the number of months between follow-ups, resulted in sub-optimal model fit based on the model's chi-square, CFI, TLI, and RMSEA, and Model 2's BIC value was the largest of all five models (see Table 5, Model 2). Results showed that baseline EPSI Binge Eating significantly predicated psychosocial dysfunction at Time 2 (p = .020). No independent variables from Time 2 significantly predicted psychosocial dysfunction at Time 3.

In Model 3, Neuroticism and the number of months between assessments were statistically controlled to better identify hypothesized associations above-and-beyond negative affect (see Table 5, Model 3). Results indicated sub-optimal fit based on the model's chi-square, CFI, TLI, and RMSEA values and resulted in one of the lower BIC values. Both baseline EPSI

Binge Eating (p = .001) and Restricting (p = .002) scales significantly predicted psychosocial dysfunction at Time 2; however, no variables predicted dysfunction at Time 3.

Finally, medication use (Model 4) and treatment seeking (Model 5) were controlled (in addition to the number of months between assessments) (see Table 5). Both Model 4 and Model 5 had poor chi-square, CFI, TLI, and RMSEA values as well as large BIC values. When controlling for medication use, no independent variables from Time 2 or Time 3 significantly predicated psychosocial dysfunction. When controlling for treatment seeking, the EPSI Binge Eating scale at Time 1 predicted dysfunction at Time 2 (p = .044) and EPSI Body Dissatisfaction at Time 2 significantly predicted dysfunction at Time 3 (p = .023).

Psychosocial functioning was included in each model as a mediator between variables of the same construct across Time 1 to Time 3. Results showed that psychosocial functioning did not mediate the association of the same construct across time in any of the five models tested.

Discussion

Eating disorders are associated with significant impairments in social adjustment and quality-of-life (Allen et al., 2013; Hudson et al., 2007; Kendler et al., 1991; Stice, Marti, & Rohde, 2013; Walters & Kendler, 1995). To better identify factors associated with psychosocial impairment in individuals with eating disorders, a goal of this study was to identify mechanisms that may help to define eating-disorder severity. I hypothesized that symptoms that reflected body dissatisfaction or overvaluation of weight or shape would represent the strongest predictor of psychosocial dysfunction. This hypothesis was not supported in the current study. Body dissatisfaction significantly predicted psychosocial dysfunction and had the largest standardized regression coefficients when omitting control variables (Model 1), when controlling for treatment seeking (Model 4), and when controlling for medication use (Model 5). These results

differ from previous studies examining the clinical utility of *DSM-5* severity indicators supporting overvaluation of weight or shape as a severity specifier (Grilo et al., 2015a, 2015b); however, past studies examined the association between overvaluation and eating-disorder psychopathology versus psychosocial dysfunction in important life domains. In addition, independent sample *t*-tests in this study revealed study participants seeking treatment and/or using psychiatric medication reported significantly higher levels of body dissatisfaction than participants who were not in treatment or prescribed psychiatric medication. It makes sense that body dissatisfaction would be a significant predictor of psychosocial dysfunction in Models 4 and 5 given that there were significant differences in the study sample that were controlled for in the analyses.

I also hypothesized that duration-of-illness would emerge as a strong predictor of psychosocial impairment. Duration-of-illness significantly predicted psychosocial impairment in Model 1; however, this association was non-significant after control variables were added to the model. In addition, standardized regression coefficients revealed that duration-of-illness did not appear as a strong predictor variable in any of the models tested. The lack of association between psychosocial dysfunction and illness duration is supported by literature showing that eating-disorder duration does not significantly predict poor quality-of-life (Bamford & Sly, 2010). However, conflicting findings have been reported in other studies (Padierna et al., 2004). Our results suggest that illness duration predicts psychosocial dysfunction due to its association with other factors, such as neuroticism and treatment seeking.

My final hypothesis was that that psychosocial functioning would mediate the association between disordered eating symptoms (independent variables) of the same construct across time, given that research has shown that disordered eating behaviors are associated with impairments

in quality-of-life (Doll et al., 2005; Mond et al., 2005; Padierna et al., 2000; Rie et al., 2005). Psychosocial functioning was included in each model as a mediator between variables of the same construct across Time 1 to Time 3 and results showed that psychosocial functioning did not mediate the association of the same construct across time in any of the models tested. These insignificant findings may have been the result of the assumed directional association between eating-disorder symptoms and psychosocial dysfunction. It would be interesting for future studies to investigate bidirectional associations between psychosocial functioning and eating-disorder symptoms.

An additional study goal was to evaluate the current *DSM-5* severity indices for AN, BN, and BED. I hypothesized that the current eating disorder severity indices would not significantly predict psychosocial impairment, given the limited research on these severity ratings. Results showed that BMI was not a significant predictor of psychosocial dysfunction; however, EPSI Restricting and Binge Eating scales were significant predictors. In addition, standardized regression coefficients for the EPSI Binge Eating scale showed that Binge Eating was one of the strongest predictor variables of psychosocial dysfunction throughout the models tested. These findings provide some support for the current eating disorder severity indices and are supported by previous literature examining *DSM-5*'s severity criteria in bulimic syndromes (Grilo et al., 2015a, 2015b, 2015c).

Interestingly, the association between binge-eating frequency and psychosocial dysfunction was not limited to individuals meeting diagnostic criteria for BED in the current study. These results align with past studies that have shown associations between binge eating and lower quality-of-life in community samples of individuals with eating disorders (Hay, 2003; Mond, Hay, Rodgers, & Owen, 2006) and impairment in college students with clinically

significant disordered eating (Hovrud & De Young, 2015). The significant association between binge-eating frequency and psychosocial dysfunction provides important insights into potential future alternative approaches to defining eating-disorder severity. Specifically, it is possible that binge eating may represent an important trans-diagnostic construct for predicting psychosocial impairment across bulimic syndromes.

Results also suggested that the frequency of restrictive eating behaviors (after controlling for negative affect) might represent an additional trans-diagnostic dimension of severity among individuals with bulimic syndromes. In support of this finding, a recent network analysis study of eating disorder symptoms found that two symptoms assessing dietary restraint/restricting emerged as 'key players,' such that their removal from the network of eating disorder symptoms resulted in fracturing of the network into smaller, disconnected components (Forbush, Siew, & Vitevitch, 2016); thus, restraint may play an important role in maintaining eating-disorder psychopathology over time. Past studies have also suggested that elevated frequency of restrictive eating behaviors in individuals diagnosed with the binge-purge subtype of AN (ANbp) does not appear to be a simple compensatory response to binge eating and may represent a non-weight based construct of severity (De Young et al., 2013). Taken together, findings from the current study yielded two potential alternative dimensions that are important to consider when assessing eating disorder severity in bulimic syndromes – binge eating and dietary restriction.

Although my findings provided important insights into novel alternative methods for assessing eating-disorder severity, it is important to note that fit indices suggested that no model demonstrated a good fit to the data. Thus, the utilization of eating-disorder symptoms to define eating disorder severity (such as those included in this paper) may not represent the best approach for measuring severity. In addition to using eating-disorder symptoms to predict future

psychosocial functioning, it may be beneficial for future studies to include symptoms of mood and anxiety disorders in severity models given the high comorbidity rates among mood, anxiety, and eating disorders (Blinder, Cumella, & Sanathara, 2006; Herzog, Keller, Sacks, & Lavori, 1992; Kaye, Bulik, Thornton, Barbarich, & Masters, 2014). Inclusion of disorders and processes associated with mood and anxiety disorders is supported by research that shows that mood and anxiety disorders better predict diagnostic "migration" among eating disorder diagnoses (e.g., movement within eating-disorder subtypes or between eating-disorder diagnoses over time) than baseline eating disorder diagnoses (Vrabel, Rosenvinge, Hoffart, Martinsen, & Rø, 2008). The inclusion of negative affect processes within severity models for eating disorders was also supported by results of the current study, which found that negative affect significantly predicted psychosocial impairment.

Certain limitations of this study are worth noting. First, the study included a limited number of individuals meeting *DSM-5* criteria for AN (Table 1) and generalizability of study results to individuals with AN is uncertain. However, lifetime prevalence of AN was higher and previous research using ecological momentary assessments (e.g., individuals report eating-disorder symptoms throughout the day using handheld computers) have suggested restricting as a non-weight-based indicator of severity in individuals with ANbp (De Young et al., 2013). Thus, it is possible that similar findings might be seen in future studies that are comprised of a larger number of individuals meeting diagnostic criteria for AN. Second, study procedures resulted in a sub-optimal retention rate between baseline and follow-ups (Time 2 = 52.9%; Time 3 = 42.3%); thus, it is possible that the most severe cases dropped out of the study, which could have affected our findings. Independent sample *t*-tests did not find significant differences between participants that continued participation and those that dropped-out based on sex, age, or race/ethnicity.

Significant differences were found between study participants and drop-outs on the EPSI Purging scale. It is possible that purging would significantly predict psychosocial dysfunction if more study participants who engaged in purging behavior continued participation. Third, participants' eating-disorder symptoms were collected through self-report measures at each follow-up assessment, and it is possible that participants did not accurately report the frequency of their behaviors or weight, introducing possible measurement error in the study results. On the other hand, because this study used online assessments to collect responses to self-report measures, the additional anonymity may have reinforced honest responses and reduce embarrassment. Fourth, the study sample was too small to compare group differences based on weight category (e.g., underweight, normal weight, overweight or obese) or eating-disorder diagnoses; eating-disorder diagnosis may have played a role in variables that predicted psychosocial dysfunction given that results from independent *t*-tests indicated significant differences in EPSI scales in these groups. However, because the majority of study participants met *DSM-5* criteria for full- and sub-threshold BN, findings can be applied across bulimic syndromes (e.g., syndromes involving binge eating episodes). Fifth, because eating disorder symptoms were collected via self-report, I was unable to collect information about the frequency of combined ICBs. Thus, the exact *DSM-5* severity index for BN could not be tested, which combines across various forms of ICBs frequencies (e.g., excessive exercise, restricting, self-induced vomiting). However, to the extent that there are important differences among ICB behaviors for predicting psychosocial dysfunction, my results could also be viewed as providing greater clarity about certain forms of ICBs in terms of their relationship with future severity. Finally, because psychosocial impairment was tested with a questionnaire assessing difficulties due to health conditions, it is possible that improved model fit would have emerged when using a measure of impairment

developed specifically for eating disorder populations. Instructions for the WHO-DAS 2.0 were modified to focus on the impact of individuals' eating disorder on psychosocial functioning that might have otherwise been missed; however, other measurements of impairment exist that were validated in eating disorder samples, such as the CIA (Bohn et al., 2008) or the Eating Disorders Quality of Life (EDQOL; Engel, Wittrock, Crosby, Wonderlich, Mitchell, & Kolotkin, 2006) instrument.

Despite these limitations, the current study has several strengths. This study was one of the first to evaluate the clinical utility of *DSM-5*'s eating disorder severity indicators (Grilo et al., 2015a, 2015b, 2015c), and the first to use a prospective longitudinal design to predict psychosocial impairment at future time-points. Most studies testing *DSM-5*'s or alternative severity indicators were based on cross-sectional designs that did not test for directional effects (Berner et al., 2013; Edler et al., 2007; Grilo et al., 2015a, 2015b, 2015c; Keel et al., 2013). This study sample included community participants meeting diagnostic criteria for *DSM-5* eating disorders. Using a community sample allowed for generalizations across bulimic syndromes and provided important insights to psychosocial impairment in a naturalistic sample of individuals with eating disorders (as opposed to studies using clinical or university samples). I used a measure of eating-disorder symptoms with good discriminant validity, which provided the opportunity to identify specific trans-diagnostic constructs that were associated with psychosocial impairment, in contrast to diagnostic measures that include more than one symptom within each criterion. Lastly, analyses of impairment were based on the WHO-DAS2.0, an external criterion to judge the clinical utility of these severity models that was not another measure of eating pathology, which has been shown to demonstrate better psychometric properties compared to other existing measures of psychosocial impairment or quality-of-life.

Identifying the associations between eating disorder symptoms and psychosocial impairment may contribute to an improved understanding of the mechanisms underlying the etiology, course, and maintenance of eating disorders, and aid in the development in new methods for characterizing severity in future editions of the *DSM*. For example, current eating disorder severity ratings are based on different symptoms for each disorder despite significant symptom overlap among eating disorders. Findings from this study suggest that DSM-5 eating disorder symptom based models of severity do not significantly predict psychosocial dysfunction. Notably, this study highlights the need for future research to: (1) evaluate current *DSM-5* severity indices for eating disorders using structured clinical interviews and (2) test the role of integrated mood, anxiety, and eating disorder symptom based models of severity.

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Table 1
Participant Demographics and Eating Disorder Diagnoses at Baseline

		Mean		*SD*	
Age		25.15		8.94	
BMI		26.67		7.71	
		N		Percentage	
Ethnicity	Non-Hispanic	171		89.5	
	Hispanic	18		9.5	
Race	Caucasian	136		71.9	
	African American	12		6.3	
	Asian	34		17.9	
	Native American/Alaskan Native	2		1.0	
	Native-Hawaiian/Pacific Islander	1		0.5	
	Multi-Racial	9		4.7	
	Other or Declined	6		3.1	
SCID Diagnoses	Disorder	Lifetime[a]		Current	
		N	Percentage	*N*	Percentage
Eating Disorders	Anorexia Nervosa	30	10.9	6	3.1
	Restricting type	13	4.7	4	2.1
	Binge-eating/purging type	17	6.2	2	1.0
	Bulimia Nervosa	88	32.0	81	42.4
	Binge Eating Disorder	55	20.0	16	8.4
	Other Specified Feeding or Eating Disorder	102	37.1	86	45.0
	Subthreshold Bulimia Nervosa	13	4.7	54	28.3
	Subthreshold Anorexia Nervosa	22	8.0	9	4.7
	Subthreshold Binge Eating Disorder	9	3.3	4	2.1
	Other	59	21.5	19	4.2

Note: SCID = Structured Clinical Interview for DSM-IV Axis I Disorders. Other Specified Feeding or Eating Disorders include presentations in which symptoms of a feeding or eating disorder cause clinically significant distress or impairment, but do not meet full criteria for AN, BN, or BED. Lifetime diagnoses exclude current eating disorder diagnosis. [a]Lifetime eating disorder diagnoses were missing for one participant due to study dropout before the baseline session could be completed.

Table 2
Variables Matched to Measures

Variables	Measure	Description
Independent Variables		
Body Mass Index	Self-report	Participants' self-reported height and weight measurements at each time point. Research staff also calculated BMI in-person at baseline.
Body Dissatisfaction	EPSI	Participants completed the EPSI assessment at each time point, which assesses body dissatisfaction, binge eating, restricting, self-induced vomiting, and laxative and diuretic misuse, and excessive exercise over the past three months.
Binge Eating	EPSI	
Restricting	EPSI	
Purging	EPSI	
Excessive Exercise	EPSI	
Duration of Illness	SCID-I	At baseline, participants were interviewed using the SCID-I. Duration of illness was calculated based on the onset of the participant's first eating disorder diagnoses.
Dependent Variables		
Psychosocial Dysfunction	WHO-DAS 2.0	Psychosocial dysfunction was assessed at Time 2 and Time 3.
Covariates		
Neuroticism	NEO-FFI-3	Neuroticism was assessed at each time point.
Time Between Baseline and Follow-up	Completion dates	The number of months between baseline and Time 2, and between Time 2 and Time 3 were calculated based on completion dates.
Treatment Seeking	Self-report	Participants' self-reported treatment seeking and medication use were calculated as dichotomous variables.
Medication	Self-report	

Note: EPSI = Eating Pathology Symptoms Inventory. SCID-I = Structured Clinical Interview for DSM-IV Axis I Disorders. WHO-DAS 2.0 = World Health Organization Disability Assessment Schedule 2.0. NEO-FFI-3 = NEO Five-Factory Inventory-3.

Table 3
Descriptive Statistics for Body Mass Index (BMI) and the Eating Pathology Symptoms Inventory (EPSI)

	Baseline												Effect Sizes	
	Normal Weight[a]		Overweight or Obese[b]		Medication Use[c]		No Medication Use[d]		Treatment Seeking[e]		Non-Treatment Seeking[f]			
Scale	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	d[g]	d[h]
BMI	21.86_a	1.65	32.69_b	7.23	28.29_c	9.58	26.14_c	7.00	26.73_d	8.87	26.63_d	7.31	-2.07	0.26
BE	22.75_a	6.20	25.81_b	5.39	25.17_c	7.08	23.48_c	5.99	23.45_d	6.63	24.06_d	6.18	-0.53	0.26
BD	25.57_a	6.05	27.01_a	6.28	27.72_c	6.42	25.49_c	6.31	25.90_d	7.46	26.08_d	5.99	-0.23	0.35
R	15.53_a	5.34	11.76_b	4.40	14.96_c	5.38	13.78_c	5.46	15.25_d	5.74	13.63_d	5.29	0.77	0.22
P	7.80_a	2.88	6.99_a	11.25	8.96_c	3.77	6.95_c	8.92	6.24_d	14.75	7.88_d	2.81	0.10	0.29
EE	14.20_a	5.83	12.04_b	5.66	12.35_c	6.21	13.17_c	5.76	12.20_d	5.97	13.26_d	5.83	0.38	-0.14
	Time 2													
BMI	22.50_a	1.84	32.62_b	7.34	28.85_c	8.73	27.12_c	7.27	27.14_e	7.86	27.46_e	7.50	-1.89	0.22
BE	20.83_a	7.21	21.69_a	6.50	24.26_c	7.23	20.00_d	6.39	22.79_e	8.30	24.06_e	6.18	-0.13	0.62
BD	24.73_a	8.19	25.57_a	7.17	28.61_c	5.48	23.85_d	7.95	27.75_e	6.88	26.08_f	5.99	-0.11	0.70
R	12.33_a	5.41	11.18_a	4.47	13.74_c	5.07	11.68_c	5.46	13.83_e	5.98	13.63_e	5.29	0.23	0.39
P	7.70_a	2.47	7.94_a	3.23	9.22_c	3.98	7.42_d	2.52	8.96_e	4.20	7.88_e	2.81	-0.08	0.54
EE	13.60_a	5.84	10.88_b	5.63	13.57_c	6.44	11.66_c	5.80	12.38_e	6.91	13.26_f	5.83	0.47	0.31
	Time 3													
BMI	22.24_a	1.83	32.80_b	7.51	28.49_c	9.45	27.30_c	7.40	27.43_d	8.04	27.58_d	7.86	-1.93	0.14
BE	20.34_a	7.26	20.60_a	6.61	21.33_c	6.30	19.87_c	7.09	19.91_d	6.52	20.34_d	6.89	-0.04	0.22
BD	23.38_a	7.50	23.03_a	7.77	25.52_c	7.69	22.78_c	6.97	25.91_d	7.65	22.16_e	6.96	0.05	0.37
R	11.79_a	4.23	10.03_a	3.69	11.86_c	4.60	11.00_c	4.69	12.18_d	5.49	10.89_d	4.19	0.44	0.19
P	7.52_a	2.37	6.83_a	1.78	7.48_c	2.32	7.11_c	2.14	7.55_d	2.74	7.04_d	1.89	0.33	0.17
EE	10.72_a	5.52	10.58_a	6.06	9.57_c	5.78	10.67_c	5.46	10.09_d	5.95	11.07_d	5.84	0.02	-0.20

Note. Raw means are reported in the table. Independent *t*-tests were used to compare scale scores between normal weight and overweight or obese college students, students using psychiatric medication and no medication use, and treatment seeking and non-treatment seeking students. For this comparison, means not sharing the same subscript within a row differ from one another at $p < .05$. BMI = Body Mass Index. BE = EPSI Binge Eating scale. BD = EPSI Body Dissatisfaction scale. R = EPSI Restricting scale. P = EPSI Purging scale. EE = EPSI Excessive Exercise scale. [a]n = 88 at Time 1; 40 at Time 2; 29 at Time 3. [b]n = 90 at Time 1; 49 at Time 2; 40 at Time 3. [c]n = 46 at Time 1; 23 at Time 2; 21 at Time 3. [d]n = 143 at Time 1; 74 at Time 2; 56 at Time 3. [e]n = 51 at Time 1; 24 at Time 2; 22 at Time 3. [f]n = 138 at Time 1; 76 at Time 2; 58 at Time 3. [g]Cohen's d for normal weight vs. overweight or obese participants. [h]Cohen's d for psychiatric medication use vs. no medication use participants. [i]Cohen's d for treatment seeking vs. non-treatment seeking participants.

Table 4
Skewness and Kurtosis for Body Mass Index (BMI) and the Eating Pathology Symptoms Inventory (EPSI)

	Baseline		Time 2		Time 3	
Scale	z_1	z_2	z_1	z_2	z_1	z_2
BMI	1.50	2.59	1.51	2.67	1.49	2.63
BE	-0.06	0.22	-0.03	-0.20	0.38	-0.13
BD	-0.59	-0.48	-0.50	-0.68	-0.27	-0.71
R	0.65	0.14	0.82	0.31	0.78	0.76
P	-10.78	138.18	1.76	2.49	2.31	5.63
EE	0.34	-0.92	0.59	-0.79	0.82	-0.58

Note. BMI = Body Mass Index. BE = EPSI Binge Eating scale. BD = EPSI Body Dissatisfaction scale. R = EPSI Restricting scale. P = EPSI Purging scale. EE = EPSI Excessive Exercise scale. z_1 is the skewness and z_2 is the kurtosis.

Table 5
Model Fit Indices After Modifications

Model	X^2	*df*	*CFI*	*TLI*	*RMSEA*	*BIC*
Model 1	330.266*	168	0.920	0.890	0.206	2526.723
Model 2	942.380*	199	0.502	0.340	0.219	5298.183
Model 3	662.266*	268	0.738	0.641	0.173	3553.890
Model 4	669.806*	268	0.741	0.645	0.170	3704.685
Model 5	988.942*	268	0.530	0.355	0.219	4365.904

Note. Model 1 included all predictor variables omitting controls; Model 2 included all predictor variables while controlling for the time between assessments by number of months; Model 3 included all predictor variables while controlling for the time between assessments and neuroticism; Model 4 included all predictor variables while controlling for the time between assessments and medication use; Model 5 included all predictor variables while controlling for the time between assessments and treatment seeking. CFI = comparative fit index. TLI = Tucker-Lewis index. RMSEA = root-mean-square error of approximation. BIC = Bayesian information criterion.
* $p < .001$.

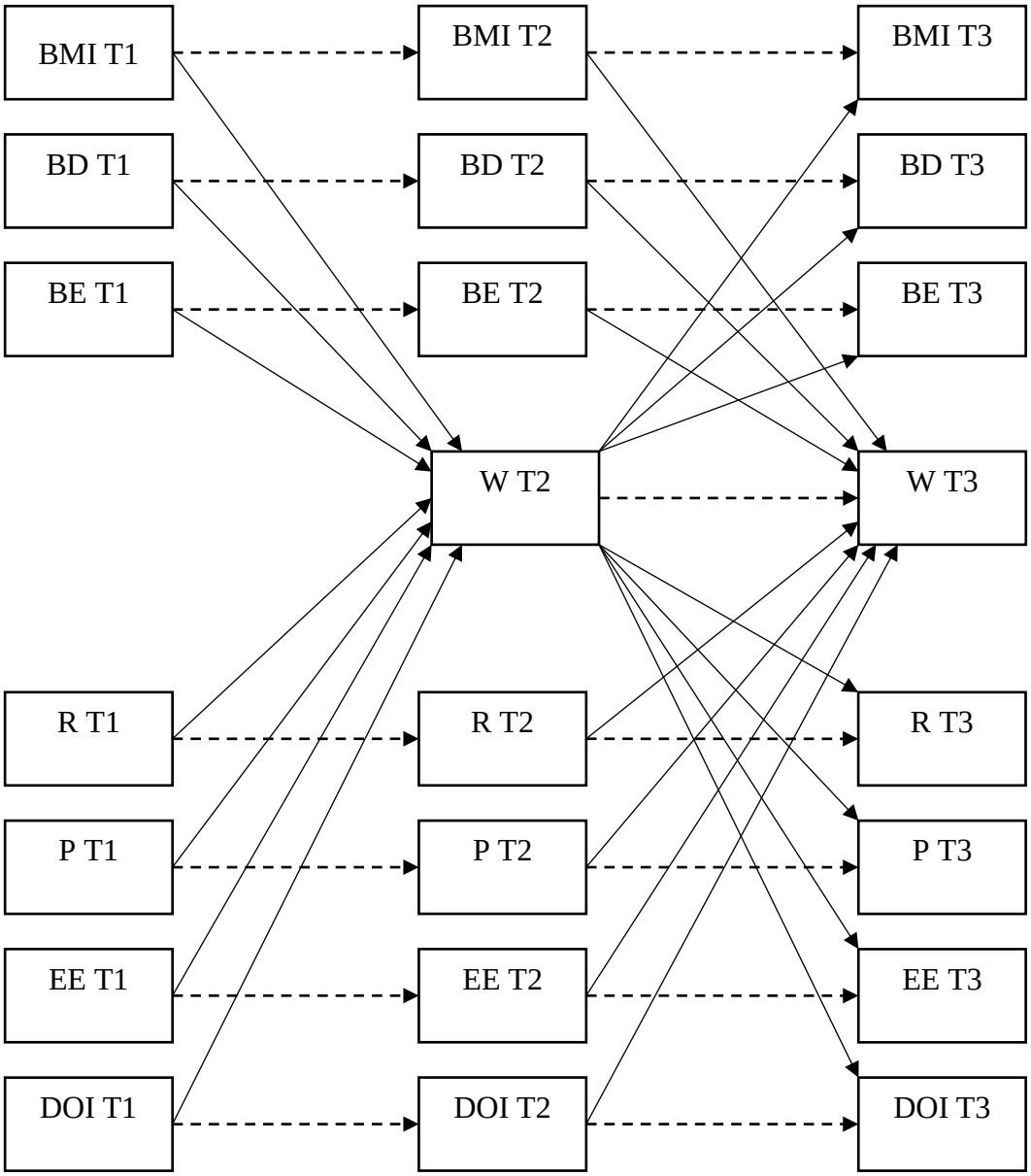

Figure 1. Conceptual autoregressive, cross-lagged, longitudinal panel path model. Dashed lines represent within-construct regression paths, while solid lines represent regression paths connecting different measures. Control variables were regressed on to all independent variables within that time point (Neuroticism, the number of months between assessments, history of psychological treatment, and use of psychiatric medication). BMI = Body Mass Index. BD = EPSI Body Dissatisfaction. BE = EPSI Binge Eating. R = EPSI Restricting. P = EPSI Purging. EE = EPSI Excessive Exercise. DOI = Duration of illness. W = WHODAS2.0 psychosocial impairment sum score. T1 = Time 1/Baseline. T2 = Time 2. T3 = Time 3.