Published in final edited form as:

Int J Eat Disord. 2016 July ; 49(7): 651–662. doi:10.1002/eat.22508.

The incremental validity of the episode size criterion in binge-eating definitions: An examination in women with purging syndromes

K. Jean Forney, M.S.¹, Lindsay P. Bodell, Ph.D.², Alissa A. Haedt-Matt, Ph.D.³, and Pamela K. Keel, Ph.D.^{1,*}

¹Department of Psychology, Florida State University, Tallahassee, Florida

²Department of Psychiatry, University of Pittsburgh School of Medicine, Pittsburgh, Pennsylvania

³Department of Psychology, Illinois Institute of Technology, Chicago, Illinois

Abstract

Objective—Of the two primary features of binge eating, loss of control (LOC) eating is well validated while the role of eating episode size is less clear. Given the ICD-11 proposal to eliminate episode size from the binge-eating definition, the present study examined the incremental validity of the size criterion, controlling for LOC.

Method—Interview and questionnaire data come from four studies of 243 women with bulimia nervosa (n=141) or purging disorder (n=102). Hierarchical linear regression tested if the largest reported episode size, coded in kilocalories, explained additional variance in eating disorder features, psychopathology, personality traits, and impairment, holding constant LOC eating frequency, age, and body mass index (BMI). Analyses also tested if episode size moderated the association between LOC eating and these variables.

Results—Holding LOC constant, episode size explained significant variance in disinhibition, trait anxiety, and eating disorder-related impairment. Episode size moderated the association of LOC eating with purging frequency and depressive symptoms, such that, in the presence of larger eating episodes, LOC eating was more closely associated with these features. Neither episode size nor its interaction with LOC explained additional variance in BMI, hunger, restraint, shape concerns, state anxiety, negative urgency, or global functioning.

Discussion—Taken together, results support the incremental validity of the size criterion, in addition to and in combination with LOC eating, for defining binge-eating episodes in purging syndromes. Future research should examine the predictive validity of episode size in both purging and non-purging eating disorders (e.g., binge eating disorder) to inform nosological schemes.

Binge eating is a prevalent feature of eating disorders associated with a host of negative outcomes, including weight gain, (1) eating disorder maintenance, (2,3) and impairment. (4)

*Correspondence to: Pamela K. Keel, Ph.D., Department of Psychology, Florida State University, 1107 W. Call St., Tallahassee, FL, 32306. keel@psy.fsu.edu.

Parts of this manuscript were presented at the International Conference on Eating Disorders, New York, New York, March 2014 and at the meeting of the Society for the Study of Ingestive Behaviors, Denver, Colorado, July 2015.

The Diagnostic and Statistical Manual, 5th Edition (DSM-5) defines binge eating by the presence of two core features: consuming an amount of food that is “definitely larger” than what most people would eat and experiencing a sense of loss of control (LOC) over eating, also called an objective binge episode (OBE). (5) Clinical experience and the research literature demonstrate that these two features may, and do, occur independently of one another. (6,7) That is, some individuals eat an unusually large amount of food without experiencing a sense of loss of control, called objective overeating (OOE). Others experience LOC while eating a small or normal amount of food, called a subjective binge episode (SBE). While the presence of LOC is a well-validated component of the DSM-5 binge-eating definition, the validity of the size criterion is less clear because it has been examined to a lesser extent. (8) This may be a result of historical patterns in the definition of binge eating. Since the inclusion of bulimia in the DSM-III, the definition of binge eating has always included large amounts of food (9) whereas LOC was added as part of the definition more recently in the DSM-IV. (10) Given variations in clinical presentation, the clear clinical significance of LOC for both “subjective” and “objective” binge episodes, and the arbitrary nature of the size criterion threshold, the current ICD-11 proposal removes the size criterion in definitions of binge eating in bulimia nervosa (BN) and binge eating disorder, (11,12) collapsing OBEs and SBEs into one group. This may be premature, however, as the absence of data does not prove an absence of validity or utility for the size criterion. Thus, the purpose of the current study was to evaluate the incremental validity of the size criterion by examining the pathology associated with eating a “definitely large amount of food,” independent of LOC eating.

Research on the second feature of binge-eating episodes, LOC eating, supports its contribution to the host of negative outcomes associated with binge eating. LOC eating, across both OBEs and SBEs, is associated with more severe eating pathology in samples of adults with eating disorders (13,14) and community samples of adolescents (15) and adults. (16,17) Similarly, LOC eating is associated with increased psychopathology in adult community (17) and eating disorder (14) samples. Specifically, LOC eating is associated with anxiety in community-based child samples, (15,18) and depressive symptoms in community child (15,19) and adult samples, (20,21) as well as adult eating disorder samples. (14) Perhaps because of the elevated eating pathology and general psychopathology, LOC eating is associated with poorer quality of life in community-based (20,22) and eating disorder (14,23) adult samples. Prospective studies of community children and adolescents have found that the presence of LOC eating predicts more severe eating pathology (24) and weight gain. (25) Given that these findings mirror the correlates of binge eating, LOC eating appears to be a core component of the psychopathology and impairment associated with binge eating, leading some authors to conclude that LOC eating is the primary pathological feature of binge eating. (15,20,26) Although these studies examined LOC eating across OBEs and SBEs, episode size was not measured and included as a covariate. This makes the independent contributions of LOC and episode size unclear as the two may be correlated.

Studies of individuals with eating disorders suggest that an unusually large episode size may be associated with more frequent purging, (27) greater eating pathology, (13) fewer cognitive distortions, (7), and more (28) or less impulsivity. (7) Additionally, in community adult samples, overeating is associated with increased disinhibition and hunger. (29) One

longitudinal study following adolescents into young adulthood found that eating an unusually large amount of food was associated with the onset of depressive symptoms, (30) and drug use in women. (1) In the context of eating disorders, overeating is associated with increased body mass index (BMI), (26,27,31) although this finding was not replicated in a community sample of those at-risk for overweight and overweight adolescents, perhaps due to range restriction. (15) Despite relatively robust associations with BMI in eating disorder groups, there does not appear to be an association between overeating and weight/shape concerns in community samples of overweight adolescent (15,32) or adult samples. (33) Likewise, in overweight community adolescent samples, overeating is not associated with greater eating pathology (15) or with greater depressive and anxiety symptoms. (15,32)

Despite some evidence regarding the clinical relevance of episode size, efforts to accumulate this evidence have been limited by failure to control for severity of LOC, (13,15,26,27,30,31,33) failure to include OOE to fully dissociate episode size from LOC, (7,20,26,27,31) and reliance on participants to determine what constitutes an unusually large amount of food. (1,26,29,30,33) Compounding this last limitation, studies have examined episode size as a categorical distinction rather than examining associations across the full range of eating episode sizes. (1,13,15,26,28–30,32,33) Categorical approaches introduce error by ignoring variability in episode size among those grouped together (e.g., assuming equivalence between someone who ate a slice of pizza and a pint of ice cream and someone who ate an extra-large deep dish pizza and a gallon of ice cream) and creating distinctions at an arbitrary threshold (e.g., assuming a meaningful difference between someone who ate a pint of ice cream and someone who ate a pint of ice cream and a slice of pizza). Additionally, a uniform threshold cannot consider natural variability in energy needs based upon such features as sex, age, physical activity, and muscle mass.

In addition to weak evidence regarding the unique contributions of eating episode size and LOC eating to eating pathology and comorbidity, interactions between these features have not been examined. The association of LOC eating with eating pathology and comorbidity may depend upon the episode size, in which case eliminating episode size from the definition of binge eating may lose clinically important information. Thus, research is needed to examine both unique, direct effects of episode size on eating pathology and comorbidity and possible interactions between episode size and LOC eating on these variables before episode size is deemed clinically irrelevant.

The current study sought to understand the incremental validity of the size criterion for binge-eating episodes by examining the unique associations of episode size, controlling for LOC, with indicators of clinical significance in a sample of women with bulimia nervosa (BN) and purging disorder (PD) who exhibited a range of both LOC eating and episode size. Although women with PD do not engage in objectively large eating episodes, many women with PD experience a loss of control over eating while eating a small or normal amount of food. (14) Thus, the inclusion of BN and PD allowed a full range of eating episode sizes to permit examination of the potential consequences of adopting the ICD-11 proposal. Notably, with the ICD-11 proposal, the majority of women with PD would be subsumed into the BN category. Thus, we are able to identify how the ICD-11 proposal may impact the utility of the diagnostic criteria. Based on findings that the presence of unusually large eating episodes

is associated with increased BMI, (26,27,31) more severe eating pathology, (13,27) increased disinhibition and hunger, (29) the onset of depressive symptoms, (30) and impulsivity and impulsive behaviors, (1,28) we hypothesized that eating episode size would explain additional variance in these variables, even after controlling for LOC severity. Finally, we examined if episode size interacted with LOC frequency in explaining variation in these features.

Methods

Participants

Data were drawn from women with BN purging subtype ($n=141$) and PD ($n=102$) who participated in one of four studies examining psychological and/or biological correlates of eating pathology in BN and PD. (34–37) Recruitment methods across studies were identical and involved mass e-mails to college women, posters and paid advertisements in the community. For inclusion in the present study, participants met criteria either for DSM-5 BN or research criteria for PD, (38) which is differentiated from BN by the *absence* of recurrent OBEs (e.g., episodes > 1000 kcal). Additionally, all participants were required to have a minimally normal weight (i.e., BMI greater than 18.5 kg/m^2) and to engage in purging behaviors (i.e., self-induced vomiting, laxative misuse, diuretic misuse) at least 12 times over the past three months. Participants were a mean (SD) age of 22.83 (5.34) years old (range 18–43) and had a mean (SD) BMI of 22.35 (1.86) kg/m^2 (range 18.52–27.59). BN and PD participants did not differ on age ($t(240) = -.04, p = .97$) or on BMI ($t(240) = .23, p = .82$). Racial background was as follows: 81.9% White ($n = 199$), 8.6% Asian or Pacific Islander ($n = 21$), 4.5% Black ($n = 11$), 3.7% Hispanic ($n = 9$), and 1.2% ($n=3$) unknown. BN and PD did not differ on ethnic/racial background ($X^2(3) = 1.12, p = .77$).

Procedure

Although participants came from four different studies, key study procedures were the same across studies. Individuals interested in the parent studies completed phone screens to establish initial eligibility before attending the first study visit to confirm eligibility. After giving informed consent, participants completed diagnostic interviews and self-report questionnaires and had height and weight measured. Data from the current study come from these interview and questionnaire measures. The Institutional Review Boards of the universities where these studies occurred approved all study procedures.

Measures

Because data were drawn from four studies, some measures were not available for all participants. Differences in sample size are noted in Table 1.

Eating Pathology—The Eating Disorder Examination (EDE) (39) is a semi-structured interview used to establish eating disorder diagnoses. All assessments were completed by masters-level interviewers who were trained by and received ongoing supervision from the corresponding author (PK). The EDE assesses eating disorder symptoms and features over the 12 weeks prior to the interview. A key advantage of the EDE is that it assesses eating episode size independently of LOC eating, such that presence and frequency of OBEs,

SBEs, and OOE's may be evaluated. Table 2 presents the frequencies of combinations of OBE, SBE, and OOE presentations, highlighting the heterogeneity in the sample. *LOC eating frequency* was operationalized as the sum of both OBEs and SBEs over the 28 days prior to assessment (range 0 – 120). The intraclass correlation (ICC) for LOC eating frequency, available for three of the parent studies, (35–37) was excellent at .99. *Purging frequency* was calculated as the total number of self-induced vomiting, laxative misuse, and diuretic misuse episodes over the past 28 days (range 1 – 136). ICC for purging frequency, available for three of the parent studies, (35–37) was excellent at .99. Interrater reliability for diagnosis in the parent studies from which these data come was excellent (Kappa Range: .91– 1.00 (35–37) and 100% agreement (34)). In addition to using the EDE to operationalize the key variables of LOC eating frequency and eating episode size, the EDE total score was used as a measure of eating disorder severity, Cronbach's alpha = .84.

Episode size was defined as the largest eating episode over the past 28 days described, whether OBE, SBE, OOE, or "normal" eating, and coded in terms of kcals (range 40 – 12,144 kcal, median = 1720). For participants for whom episode number was coded "too great to be counted" (n = 2 for LOC eating; n = 1 for self-induced vomiting), analyses used the highest frequency reported by other participants. Eating episodes were probed to provide detailed information about food consumed including serving sizes, types of food, and food brands, using standard EDE procedures. Kilocalories were calculated using Bowes & Church's Food Values of Portions Commonly Used (40,41) or using commercially available nutrition information (e.g., www.mcdonalds.com for McDonald's Big Mac). 10% of participants were randomly drawn from all four parent studies for interrater reliability; the ICC was excellent at .88 for episode size. Further supporting the validity of this assessment, an *ad lib* test meal of frozen yogurt was administered to a subset of participants (n = 60); frozen yogurt consumption was correlated with largest eating episode size with a moderate effect size, $r = .45$, $p < .001$. (37)

The Body Shape Questionnaire (BSQ) (42) assesses body image disturbance over the past 28 days and differentiates between "probable" and "non-probable" eating disorder cases. It has a reported 3-week test-retest reliability of .88. (43) Cronbach's alpha was .95.

The Three-Factor Eating Questionnaire (TFEQ) (44) is a 51-item self-report questionnaire that assesses cognitive restraint of eating, disinhibition around food, and hunger. The Disinhibition and Hunger subscales have distinguished between individuals with BN and PD participants in prior studies. (35,36,45) Internal consistency was good for the Restraint (alpha = .83), Disinhibition (alpha = .83), and Hunger subscales (alpha = .84).

Related Psychopathology and Personality Features—The Beck Depression Inventory (BDI) (46) is a 21-item self-report questionnaire that assesses the cognitive and somatic symptoms of depression. Internal consistency was good, alpha = .91.

The State-Trait Anxiety Inventory (STAI) (47) is a 40-item measure of both current anxiety (state) and anxiety proneness (trait). Higher test-retest reliability in the Trait (.91) compared to the State (.73) scale (36) support the distinctiveness of the state and trait scales. (48) Cronbach's alpha was .94 for state anxiety and .93 for trait anxiety.

The Negative Urgency subscale of the UPPS Impulsive Behavior Scale (UPPS) (49) has been used to examine associations between aspects of impulsive behavior and eating disorder symptoms in both clinical and nonclinical samples. (50,51) Negative urgency, in particular, is associated with LOC eating in PD (14) and is a risk factor for binge eating (52). Cronbach's alpha was .87 for negative urgency.

Impairment and Functioning—The Clinical Impairment Assessment (CIA) (53) is a 16-item questionnaire that specifically assesses impairment due to an eating disorder. CIA scores are positively correlated with clinicians' rating of impairment, and scores differentiated those with a diagnosed eating disorder from those who no longer met criteria. (53) Cronbach's alpha was .91.

The Global Assessment of Functioning (GAF) from the Structured Clinical Interview for DSM-IV (SCID) (54) is an interviewer rating of general functioning on a scale from 1 (poor) to 100 (good).

Data Analyses

LOC eating frequency, purging frequency, and age were log transformed due to kurtosis. Variables were inspected for outliers, and replacement values were entered as needed. Pearson correlations were used to examine the associations of LOC frequency and episode size with measures of eating pathology, related psychopathology, personality, and impairment. When episode size was significantly associated with outcomes, the independent effect of episode size on measures of eating pathology, related psychopathology, personality and impairment was tested in hierarchical linear regression models using standardized variables of interest (i.e., LOC eating frequency and episode size). In the first step, covariates and LOC eating frequency were entered. In the second step, episode size was entered, and in the third step, the interaction of LOC eating and episode size was entered. The change in R^2 was evaluated at each step to test if episode size or the interaction of episode size and LOC eating explained significant variance. If the addition of the interaction term was significant, the interaction was probed at one standard deviation above and below the mean for eating episode size. We also probed the interaction at one standard deviation above and below the mean for LOC eating to understand the incremental validity of LOC eating when examining episode size. Parent study was group coded and used as a covariate in all hierarchical regression analyses to control for any systematic differences across studies. BMI and age were used as covariates in all hierarchical regression models. Because these represent cross-sectional analyses, we also ran analyses examining the incremental validity of LOC eating above and beyond episode size. We evaluated the change in R^2 after adding LOC eating to regression models containing episode size, parent study, BMI, and age. Given our interest in how episode size and LOC eating are associated with greater pathology, we used one-tailed tests with an alpha level of .01 when examining main effects of episode size and LOC eating. Because we did not have *a priori* hypotheses regarding the interaction of LOC eating and episode size, we used a two-tailed test with an alpha level of .01

Results

Table 1 presents means and standard deviations as well as correlations of LOC eating frequency and episode size with eating pathology, related psychopathology, personality, and impairment. LOC eating frequency and episode size showed a significant moderate association, $r = .41$, $p < .001$, underscoring the importance of examining their independent contributions to the clinical significance of binge-eating episodes.

Eating Pathology

LOC eating was associated with purging frequency with a moderate to large effect size, whereas episode size was associated with a small to medium effect size (see Table 1). The multivariable model controlling for parent study, BMI, age, and LOC eating frequency explained a moderate to large amount of variance in purging frequency ($R^2 = .21$, $F(6, 234) = 10.56$, $p < .001$; see Table 3). The addition of eating episode size did not explain significantly more variance in purging frequency ($R^2 = .00$, $F(1, 233) = .04$, $p = .42$). However, the addition of the interaction term explained a moderate amount of variance in purging frequency ($R^2 = .10$, $F(1, 232) = 35.33$, $p < .001$) (see Figure 1). Probing this interaction indicated that at smaller episode size, LOC eating frequency was positively associated with purging frequency ($\beta = .22$, $t(232) = 3.04$, $p = .003$, $f^2 = .04$) and at larger eating episode size, LOC eating frequency was more strongly associated with purging frequency ($\beta = .995$, $t(232) = 8.93$, $p < .001$, $f^2 = .34$). Probing this interaction the alternate way revealed that at lower LOC eating frequency, episode size was negatively associated with purging frequency ($\beta = -.50$, $t(232) = -4.77$, $p < .001$, $f^2 = .10$) and at higher LOC eating frequency, episode size was positively associated with purging frequency ($\beta = .27$, $t(232) = 3.64$, $p < .001$, $f^2 = .06$).

Both LOC eating frequency and episode size demonstrated small to moderate correlations with eating disorder severity as measured by the EDE total score (see Table 1). As shown in Table 3, LOC eating frequency and covariates explained a small to moderate amount of variance in eating disorder severity in the multiple regression model ($R^2 = .08$, $F(6, 234) = 3.52$, $p = .001$). The addition of episode size was associated with a small effect size that failed to reach statistical significance ($R^2 = .01$, $F(1, 233) = 3.26$, $p = .04$), and the addition of the LOC by episode size interaction did not explain significantly more variance ($R^2 = .003$, $F(1, 232) = .73$, $p = .39$).

Both LOC eating and episode size demonstrated small to moderate correlations with shape and weight concerns (see Table 1). In the regression model, parent study, BMI, age, and LOC eating frequency explained a moderate amount of variance in shape and weight concerns ($R^2 = .11$, $F(6, 233) = 4.66$, $p < .001$; see Table 3). The addition of episode size explained a small amount of variance, but failed to reach statistical significance ($R^2 = .02$, $F(1, 232) = 4.04$, $p = .023$). The addition of the episode size by LOC eating interaction did not explain significantly more variance in shape and weight concerns ($R^2 = .00$, $F(1, 231) = .003$, $p = .96$).

At the bivariate level, LOC eating was correlated with disinhibition scores at a large effect size and episode size was correlated at a moderate to large effect size (see Table 1). LOC

eating frequency and covariates explained a large amount of variance in disinhibition ($R^2 = .36$, $F(5, 221) = 24.82$, $p < .001$; see Table 3). The addition of episode size significantly explained a small to moderate amount of variance ($R^2 = .06$, $F(1, 220) = 24.30$, $p < .001$). The interaction of episode size and LOC eating explained a small amount of variance that failed to reach statistical significance ($R^2 = .01$, $F(1, 219) = 3.35$, $p = .07$).

A different pattern was observed with hunger. LOC eating frequency was correlated with hunger at a moderate to large effect size and episode size was correlated with hunger at a moderate effect size (see Table 1). In the multiple regression model, LOC eating frequency and covariates explained a moderate to large amount of variance in hunger scores ($R^2 = .22$, $F(5, 221) = 12.27$, $p < .001$; see Table 3). The addition of episode size explained a small amount of variance but did not reach statistical significance ($R^2 = .01$, $F(1, 220) = .01$, $p = .04$) and the episode size by LOC eating interaction ($R^2 = .00$, $F(1, 219) = .03$, $p = .86$) did not explain additional variance in hunger scores.

Finally, LOC eating demonstrated a negative small to moderate correlation with restraint and episode size demonstrated a small, non-significant negative correlation with restraint. LOC eating had a small, negative non-significant correlation with BMI and episode size demonstrated a negligible relationship with BMI. Thus, restraint and BMI were not examined in hierarchical regression models.

Related Psychopathology and Personality

LOC eating frequency demonstrated a moderate to large correlation with depressive symptoms and episode size demonstrated a small to moderate correlation (see Table 1). In the multiple regression model, LOC eating frequency, parent study, age, and BMI explained a moderate to large amount of variance in depressive symptoms ($R^2 = .16$, $F(5, 220) = 8.42$, $p < .001$; see Table 4). The addition of episode size explained a small amount of variance that did not reach statistical significance ($R^2 = .01$, $F(1, 219) = 3.17$, $p = .04$). The addition of the interaction term significantly explained a small to moderate amount of variance ($R^2 = .03$, $F(1, 218) = 7.03$, $p = .009$) (see Figure 1 for depiction of a similar interaction). Probing this interaction indicated that at smaller eating episode size, LOC frequency was positively associated with depressive symptoms ($\beta = .23$, $t(218) = 2.76$, $p = .006$, $f^2 = .04$) and at larger eating episode size, the relationship between LOC frequency and depressive symptoms was stronger ($\beta = .60$, $t(218) = 5.01$, $p < .001$, $f^2 = .12$). Alternatively, probing this interaction at lower LOC eating frequency revealed that episode size was not significantly associated with depressive symptoms ($\beta = -.14$, $t(218) = -1.16$, $p = .25$, $f^2 = .01$) and at higher LOC eating frequency, episode size was positively associated with depressive symptoms ($\beta = .25$, $t(218) = 3.03$, $p = .003$, $f^2 = .04$).

LOC eating and episode size were both correlated with state anxiety at small to moderate effect sizes (see Table 1). LOC eating and covariates explained a moderate amount of variance in state anxiety ($R^2 = .10$, $F(6, 227) = 4.10$, $p < .001$; see Table 4). The addition of episode size did not reach statistical significance but was associated with a small effect size ($R^2 = .01$, $F(1, 226) = 3.36$, $p = .03$). The addition of the episode size and LOC eating frequency did not explain a significant amount of variance ($R^2 = .00$, $F(1, 225) = .00$, $p = .99$).

LOC eating demonstrated a moderate correlation and episode size demonstrated a small to moderate correlation with trait anxiety (see Table 1). In the multiple regression model, parent study, age, BMI, and LOC eating frequency explained a moderate to large amount of variance in trait anxiety ($R^2 = .15$, $F(6, 225) = 6.48$, $p < .001$; see Table 4). The addition of episode size significantly explained a small amount of variance ($R^2 = .02$, $F(1, 224) = 6.47$, $p = .006$). The addition of the episode size by LOC eating interaction also explained a small amount of variance that fell short of reaching statistical significance ($R^2 = .02$, $F(1, 223) = 5.67$, $p = .018$).

Finally, LOC eating demonstrated a large correlation with negative urgency and episode size demonstrated a small to moderate correlation that failed to reach statistical significance (see Table 1). Thus, negative urgency was not examined further in a hierarchical regression model.

Impairment and Functioning

Both LOC eating and episode size demonstrated moderate to large correlations with eating disorder-related impairment (see Table 1). LOC eating frequency, parent study, age, and BMI explained a large amount of variance in eating disorder-related impairment ($R^2 = .28$, $F(4, 68) = .64$, $p < .001$; see Table 4). The addition of episode size explained a small to moderate amount variance at the threshold for statistical significance ($R^2 = .06$, $F(1, 67) = 5.71$, $p = .010$). The addition of the interaction term did not explain additional variance ($R^2 = .003$, $F(1, 66) = .25$, $p = .62$).

Finally, LOC eating demonstrated a moderate to large negative correlation with global functioning and episode size demonstrated a small to moderate negative correlation. LOC eating frequency, age, BMI, and parent study explained a moderate to large amount of variance ($R^2 = .22$, $F(6, 230) = 10.94$, $p < .001$). The addition of episode size did not explain additional variance ($R^2 = .00$, $F(1, 229) = .06$, $p = .41$) nor did the addition of the interaction term ($R^2 = .001$, $F(1, 228) = .20$, $p = .65$).

Examining the Incremental Validity of LOC Eating

We also ran analyses testing the incremental validity of LOC, holding constant episode size and covariates. The addition of LOC eating explained a moderate to large amount of variance in purging frequency ($R^2 = .15$, $F(1, 233) = 44.36$, $p < .001$). Similarly, holding constant episode size and covariates, the addition of LOC eating explained a small to moderate amount of variance in EDE total scores ($R^2 = .03$, $F(1, 233) = 8.37$, $p = .002$). The same pattern was observed with shape concerns ($R^2 = .04$, $F(1, 232) = 10.37$, $p < .001$). The addition of LOC eating to the model containing episode size and covariates explained a moderate to large amount of variance in disinhibition ($R^2 = .18$, $F(1, 220) = 69.96$, $p < .001$) and hunger ($R^2 = .14$, $F(1, 220) = 39.39$, $p < .001$).

Concerning depressive symptoms, the addition of LOC eating to the model containing episode size and covariates explained a moderate amount of variance in depressive symptoms ($R^2 = .09$, $F(1, 219) = 23.27$, $p < .001$). LOC eating explained a small amount of additional variance in state anxiety ($R^2 = .02$, $F(1, 226) = 5.72$, $p = .009$) and a small to moderate amount of variance in trait anxiety ($R^2 = .05$, $F(1, 224) = 14.63$, $p < .001$).

In a model containing episode size and covariates, the addition of LOC eating frequency explained a moderate amount of variance in eating disorder-related impairment, ($R^2 = .11$, $F(1, 67) = 11.18$, $p < .001$). In contrast, the addition of LOC eating frequency explained a small to moderate amount of variance in global functioning, ($R^2 = .05$, $F(1, 229) = 14.10$, $p < .001$).

Discussion

The present study sought to examine the incremental validity of the size criterion for binge-eating episodes. Three patterns of associations were observed in examining relationships between episode size and LOC eating frequency (summarized in Figure 2). In the first and most commonly observed pattern, LOC eating frequency, but not episode size, was associated with clinical variables, including eating disorder severity, shape concerns, restraint, hunger, state anxiety, impulsivity, and global functioning. In the second pattern, holding LOC eating constant, episode size explained additional variance in disinhibition around food, trait anxiety, and eating disorder-related impairment. In the third pattern, LOC frequency interacted with episode size to have differential associations with purging frequency and depressive symptoms. Thus, some hypotheses were supported while others were not. Results reinforce the clinical significance and salience of LOC eating in purging syndromes as the key feature of binge eating. Additionally, for almost half of the variables examined (including purging frequency and current depressive symptoms, which may serve as indicators of severity and distress, respectively) episode size demonstrated either direct associations with outcome variables or moderated associations between LOC eating and outcome variables. Taken together, results reinforce the clinical significance of LOC eating and support the incremental validity of the size criterion in defining binge episodes among those who purge.

The strength of association between LOC eating and purging frequency varied by episode size. Specifically, our results indicate that, at larger episode sizes, LOC eating is more strongly associated with purging frequency whereas this association is weaker with smaller eating episode sizes. This interaction also can be interpreted as the relationship between episode size and purging frequency varied by LOC eating. At more frequent LOC eating levels, episode size was positively associated with purging frequency whereas when LOC eating was less frequent, episode size was negatively associated with purging frequency. This pattern may reflect the presence of two groups among individuals who purge, based, in part, on our recruitment of participants for the parent studies from which data came. The first group reflects those with BN, for whom binge eating episodes are a known trigger of purging. The second group may comprise individuals with PD whose purging behavior is triggered by the act of eating in the presence of shape concerns or other triggers, such as changes in positive and negative affect (34) rather than “pathological overeating” or feelings of LOC. Importantly, regardless of our approach to recruitment, if PD and BN merely represented the same disorder of LOC eating and compensatory behavior residing on different ends of an irrelevant continuum of eating episode size, one would not expect this interaction. This has important implications for intervention and identifying maintenance factors for purging behaviors in the context of various purging syndromes.

A significant interaction between LOC eating and episode size also was found when examining depressive symptoms, such that the relationship between LOC eating and depressive symptoms was stronger in the presence of larger eating episodes. Examining the combination of episode size and LOC eating, rather than only episode size or only LOC eating, aids in understanding the association of dysregulated eating with this indicator of distress. As depressive symptoms (55) are associated with worse eating disorder outcomes, these results support the validity of distinguishing between eating disorder groups based on the combination of LOC eating and large eating episode sizes as currently indicated in the DSM-5.

Results indicate that larger eating episodes are associated with greater disinhibition around food, independent of LOC eating, consistent with evidence that individuals with BN report more disinhibition than individuals with PD. (35,56,57) This also is consistent with associations between disinhibition and eating large amounts of food in non-clinical samples (29) and suggests the construct of disinhibition is more than simply feeling out of control. Instead, it appears to index a behavioral indicator of loss of control, in which a person actually consumes greater quantities of food. Episode size explained additional variance in trait anxiety, independent of LOC eating. This is particularly relevant in treatment contexts as trait anxiety predicts poorer treatment outcome. (58) Finally, independent associations of episode size with eating disorder distress, but not general functioning, suggest that larger episodes are particularly distressing for those with eating disorders and thus objectively large eating episodes may be important targets when working to increase patients' quality of life.

We were unable to replicate previous findings regarding associations of large eating episodes with hunger. (29) This finding does not fit with previous reports of greater hunger in BN compared to PD, (35,36) which had been attributed to differences in episode size. Thus, other features that differ between these groups likely account for the differences in hunger. Although the link between impulsivity and binge eating is well established, (51) findings regarding the relationship of episode size and impulsivity are mixed. (7,28) Our results suggest that the negative urgency facet of impulsivity is particularly linked to LOC, not episode size. Finally, we were unable to replicate results regarding associations between episode size and higher BMI, (26,27,31) perhaps due to restricted range of BMI in the parent studies (i.e., BMI between 18.5 – 26.5 kg/m²).

This study has a number of strengths. Our analytic approach allowed us to simultaneously consider both episode size and LOC eating dimensionally in order to examine the unique contributions of these features, which is particularly important as size and LOC eating were correlated with a moderate to large effect size. In addition, our large sample provided adequate power to detect small to moderate effect sizes. However, there are limitations to acknowledge. As these are secondary analyses, results may not generalize to non-normal weight eating disorders or eating disorders without purging behaviors. As individuals with BN were required to experience LOC and individuals with PD were not required to experience LOC, recruitment ensured non-independence between episode size and LOC. These results should be replicated in samples that are not selected based upon the presence/absence of binge-eating episodes, among eating disorders without purging behaviors, and in

more weight-diverse samples. This study relied on self-report of food recall during structured clinical interview assessments and on lay estimation of caloric intake, which introduces error. Additionally, the use of kilocalories as a measure of episode size reflects a combination of energy density and the volume of food consumed. This provides an imperfect metric of the large amounts of food specified in the DSM-5, which may include large amounts of non-calorically dense foods (e.g., 5 apples). Prospective, ecological momentary assessment and behavioral measures (e.g., *ad lib* meals) may provide more accurate measures of LOC frequency and episode size, respectively. However, the assessment methods in this study represent those used in clinical settings and thus increase the generalizability of findings to a highly relevant context. Because this study represents a secondary analysis of data, we were unable to examine confounds that may influence food intake such as muscle mass, physical activity, and actual dietary restriction. Similarly, a person's "largest" eating episode may not be representative of their "typical" eating episode, but we were unable to measure "typical" eating episodes due to the nature of secondary data analysis. Finally, LOC frequency and largest eating episode size are not parallel measures of severity. Although LOC frequency matches the method of assessment in prior studies, (17,20) future research would benefit from the use of the newer dimensional measures of LOC severity (59,60) as a parallel to largest eating episode size.

Taken together, results indicate that assessing the size of eating episodes provides information about eating disorder severity, disinhibition, depressive symptoms, anxiety, and eating disorder-related impairment that is not fully captured by the presence or severity of LOC eating. Episode size, when considered in conjunction with LOC eating, provides incremental validity in understanding eating pathology and should be retained in nosological schemes. This conclusion is in line with the DSM-5 definition of binge-eating episodes and evidence linking disruptions in gut hormone functioning to eating unusually large amounts of food. (36,61) Adopting the ICD-11 proposal would collapse together individuals with objective and subjective binge episodes. This functionally combines the diagnoses of BN and PD, despite evidence that these groups meaningfully differ on severity, comorbidity, impairment and possible maintenance factors (e.g., gut hormone function) (38). Adopting the ICD-11 proposal also may lead to a decrease in the reliability of the BN diagnosis, given evidence that subjective binge episodes are more difficult to assess reliably. (62) By retaining the size criterion, the diagnosis of BN maintains good reliability and remains more homogeneous. An alternative to this approach would be to consider binge episode size as a specifier among those who purge. Including binge episode size as a specifier would protect reliability and homogeneity of BN as a diagnosis and distinguish between BN and PD, consistent with data supporting the concurrent validity of this distinction. (38) However, not all individuals with PD experience LOC eating episodes, (14) and these cases would be missed by either adopting the ICD-11 proposal or by adding episode size as a specifier among those identified on the basis of LOC eating. To expand on the current findings regarding the clinical utility of the dichotomous size criterion, future research should examine the predictive validity of episode size, LOC eating, and their interaction on illness maintenance in order to inform diagnostic systems and treatment targets.

Acknowledgments

Funding: This research was supported by National Institute of Mental Health Grants F31 MH105082 (Forney), F31 MH08456 (Haedt-Matt), R01 MH61836 (Keel), R03 MH61320 (Keel), an American Psychological Association dissertation research award (Haedt-Matt) and an Academy for Eating Disorders student research grant (Haedt-Matt).

References

1. Sonnevile KR, Horton NJ, Micali N, Crosby RD, Swanson SA, Solmi F, et al. Longitudinal associations between binge eating and overeating and adverse outcomes among adolescents and young adults: Does loss of control matter? *JAMA Pediatrics*. 2013; 167(2):149–155. [PubMed: 23229786]
2. Loeb KL, Le Grange D, Hildebrandt T, Greif R, Lock J, Alfano L. Eating disorders in youth: diagnostic variability and predictive validity. *Int J Eat Disord*. 2011; 44(8):692–702. [PubMed: 22072406]
3. Richard M. Effective treatment of eating disorders in Europe: Treatment outcome and its predictors. *Eur Eat Disord Rev*. 2005; 13(3):169–179.
4. 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]
5. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5. Arlington, VA: American Psychiatric Association; 2013.
6. Grilo CM, Masheb RM, Wilson GT. A comparison of different methods for assessing the features of eating disorders in patients with binge eating disorder. *J Consult Clin Psychol*. 2001; 69(2):317. [PubMed: 11393608]
7. Brownstone LM, Bardone-Cone AM, Fitzsimmons-Craft EE, Printz KS, Le Grange D, Mitchell JE, et al. Subjective and objective binge eating in relation to eating disorder symptomatology, negative affect, and personality dimensions. *Int J Eat Disord*. 2013; 46(1):66–76. [PubMed: 23109272]
8. Wolfe BE, Baker CW, Smith AT, Kelly-Weeder S. Validity and utility of the current definition of binge eating. *Int J Eat Disord*. 2009; 42(8):674–686. [PubMed: 19610126]
9. American Psychiatric Association. Diagnostic and statistical manual (DSM-III). Washington, DC: 1980.
10. American Psychiatric Association. DSM-IV-TR: Diagnostic and statistical manual of mental disorders, text revision. American Psychiatric Association; 2000.
11. Uher R, Rutter M. Classification of feeding and eating disorders: Review of evidence and proposals for ICD-11. *World Psychiatry*. 2012; 11(2):80–92. [PubMed: 22654933]
12. Al-Adawi S, Bax B, Bryant-Waugh R, Claudino AM, Hay P, Monteleone P, et al. Revision of ICD–status update on feeding and eating disorders. *Adv Eat Disord*. 2013; 1(1):10–20.
13. Birgegård A, Clinton D, Norring C. Diagnostic issues of binge eating in eating disorders. *Eur Eat Disord Rev*. 2013; 21(3):175–183. [PubMed: 23440765]
14. Forney KJ, Haedt-Matt AA, Keel PK. The role of loss of control eating in purging disorder. *Int J Eat Disord*. 2014; 47(3):244–251. [PubMed: 24185981]
15. Shomaker LB, Tanofsky-Kraff M, Elliott C, Wolkoff LE, Columbo KM, Ranzenhofer LM, et al. Salience of loss of control for pediatric binge episodes: Does size really matter? *Int J Eat Disord*. 2010; 43(8):707–716. [PubMed: 19827022]
16. Jenkins PE, Hoste RR, Meyer C, Blissett JM. Eating disorders and quality of life: A review of the literature. *Clin Psychol Rev*. 2011; 31(1):113–121. [PubMed: 20817335]
17. Latner JD, Hildebrandt T, Rosewall JK, Chisholm AM, Hayashi K. Loss of control over eating reflects eating disturbances and general psychopathology. *Behav Res Ther*. 2007; 45(9):2203–2211. [PubMed: 17229399]
18. Goossens L, Soenens B, Braet C. Prevalence and characteristics of binge eating in an adolescent community sample. *J Clin Child Adolesc Psychol*. 2009; 38(3):342–353. [PubMed: 19437295]

19. Tanofsky-Kraff M, Shomaker LB, Olsen C, Roza CA, Wolkoff LE, Columbo KM, et al. A prospective study of pediatric loss of control eating and psychological outcomes. *J Abnorm Psychol.* 2011; 120(1):108–118. [PubMed: 21114355]
20. Vannucci A, Theim KR, Kass AE, Trockel M, Genkin B, Rizk M, et al. What constitutes clinically significant binge eating? Association between binge features and clinical validators in college-age women. *Int J Eat Disord.* 2013; 46(3):226–232. [PubMed: 23386591]
21. Colles SL, Dixon JB, O'Brien PE. Loss of control is central to psychological disturbance associated with binge eating disorder. *Obesity.* 2008; 16(3):608–614. [PubMed: 18239550]
22. Jenkins PE, Conley CS, Rienecke Hoste R, Meyer C, Blissett JM. Perception of control during episodes of eating: Relationships with quality of life and eating psychopathology. *Int J Eat Disord.* 2012; 45(1):115–119. [PubMed: 21344466]
23. Latner JD, Vallance JK, Buckett G. Health-related quality of life in women with eating disorders: Association with subjective and objective binge eating. *J Clin Psychol Med S.* 2008; 15(2):148–153.
24. Hilbert A, Hartmann AS, Czaja J, Schoebi D. Natural course of preadolescent loss of control eating. *J Abnorm Psychol.* 2013; 122(3):684. [PubMed: 24016009]
25. Tanofsky-Kraff M, Yanovski SZ, Schvey NA, Olsen CH, Gustafson J, Yanovski JA. A prospective study of loss of control eating for body weight gain in children at high risk for adult obesity. *Int J Eat Disord.* 2009; 42(1):26–30. [PubMed: 18720473]
26. Mond J, Latner J, Hay P, Owen C, Rodgers B. Objective and subjective bulimic episodes in the classification of bulimic-type eating disorders: Another nail in the coffin of a problematic distinction. *Behav Res Ther.* 2010; 48(7):661–669. [PubMed: 20434132]
27. Jones A, Helverskov J, Rokkedal K, Clausen L. A comparison of eating disorder symptomatology in a clinical population of adolescents with subjective versus objective bulimic episodes. *Adv Eat Disord.* 2015; 3(2):124–132.
28. Keel PK, Mayer SA, Harnden-Fischer JH. Importance of size in defining binge eating episodes in bulimia nervosa. *Int J Eat Disord.* 2001; 29(3):294–301. [PubMed: 11262508]
29. Mailloux G, Bergeron S, Meilleur D, D'Antono B, Dubé I. Examining the associations between overeating, disinhibition, and hunger in a nonclinical sample of college women. *Int J Behav Med.* 2014; 21(2):375–384. [PubMed: 23532565]
30. Skinner HH, Haines J, Austin SB, Field AE. A prospective study of overeating, binge eating, and depressive symptoms among adolescent and young adult women. *J Adolescent Health.* 2012; 50(5):478–483.
31. Watson HJ, Fursland A, Bulik CM, Nathan P. Subjective binge eating with compensatory behaviors: A variant presentation of bulimia nervosa. *Int J Eat Disord.* 2013; 46(2):119–126. [PubMed: 22911884]
32. Goldschmidt AB, Jones M, Manwaring JL, Luce KH, Osborne MI, Cunnig D, et al. The clinical significance of loss of control over eating in overweight adolescents. *Int J Eat Disord.* 2008; 41(2):153–158. [PubMed: 18095271]
33. Striegel-Moore RH, Wilson GT, Wilfley DE, Elder KA, Brownell KD. Binge eating in an obese community sample. *Int J Eat Disord.* 1998; 23(1):27–37. [PubMed: 9429916]
34. Haedt-Matt AA, Keel PK. Affect regulation and purging: An ecological momentary assessment study in purging disorder. *J Abnorm Psychol.* 2015; 124(2):399. [PubMed: 25688426]
35. Keel PK, Haedt A, Edler C. Purging disorder: An ominous variant of bulimia nervosa? *Int J Eat Disord.* 2005; 38(3):191–199. [PubMed: 16211629]
36. Keel PK, Wolfe BE, Liddle RA, De Young KP, Jimerson DC. Clinical features and physiological response to a test meal in purging disorder and bulimia nervosa. *Arch Gen Psychiatry.* 2007; 64(9):1058–1066. [PubMed: 17768271]
37. Keel, P.; Hildebrandt, B.; Haedt-Matt, A.; McCormick, L.; Appelbaum, J.; Jimerson, D. Examination of PYY response in purging disorder, bulimia nervosa, and healthy controls: Novel link between physiology and behavior. Presented at the International Conference on Eating Disorders; Boston, MA. 2015.
38. Keel PK, Striegel-Moore RH. The validity and clinical utility of purging disorder. *Int J Eat Disord.* 2009; 42(8):706–719. [PubMed: 19642215]

39. Cooper Z, Fairburn C. The Eating Disorder Examination: A semi-structured interview for the assessment of the specific psychopathology of eating disorders. *Int J Eat Disord.* 1987; 6(1):1–8.
40. Pennington, JAT. Bowes & Church's food values of portions commonly used. 17. Philadelphia: Lippincott; 1998.
41. Pennington, JAT.; Spungen, J. Bowes & Church's Food Values of Portions Commonly Used. 19. Philadelphia: Lippincott Williams & Wilkins; 2009.
42. Cooper PJ, Taylor MJ, Cooper Z, Fairburn CG. The development and validation of the Body Shape Questionnaire. *Int J Eat Disord.* 1987; 6(4):485–494.
43. Rosen JC, Jones A, Ramirez E, Waxman S. Body shape questionnaire: Studies of validity and reliability. *Int J Eat Disord.* 1996; 20(3):315–319. [PubMed: 8912044]
44. 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]
45. Smith KE, Crowther JH. An exploratory investigation of purging disorder. *Eating Behav.* 2013; 14(1):26–34.
46. Beck AT, Ward CD, Mendelson M, Mock J, Erbaugh J. An inventory for measuring depression. *Arch Gen Psychiatry.* 1961; 4(6):561–571. [PubMed: 13688369]
47. Spielberger, CD.; Gorsuch, RL.; Lushene, R.; Vagg, PR.; Jacobs, GA. The State-Trait Anxiety Inventory. Palo Alto, CA: Consulting Psychologists Press; 1983.
48. Spielberger CD, Vagg PR. Psychometric Properties of the STAI: A Reply to Ramanaiah, Franzen, and Schill. *J Pers Assess.* 1984; 48(1):95. [PubMed: 6707862]
49. Whiteside SP, Lynam DR, Miller JD, Reynolds SK. Validation of the UPPS impulsive behaviour scale: A four-factor model of impulsivity. *Eur J Pers.* 2005; 19(7):559–574.
50. Anestis MD, Smith AR, Fink EL, Joiner TE. Dysregulated eating and distress: Examining the specific role of negative urgency in a clinical sample. *Cognitive Ther Res.* 2009; 33(4):390–397.
51. Fischer S, Smith GT, Cyders MA. Another look at impulsivity: A meta-analytic review comparing specific dispositions to rash action in their relationship to bulimic symptoms. *Clin Psychol Rev.* 2008; 28(8):1413–1425. [PubMed: 18848741]
52. Pearson CM, Combs JL, Zapolski TCB, Smith GT. A longitudinal transactional risk model for early eating disorder onset. *J Abnorm Psychol.* 2012; 121(3):707–718. [PubMed: 22428790]
53. Bohn K, Doll HA, Cooper Z, O'Connor M, Palmer RL, Fairburn CG. The measurement of impairment due to eating disorder psychopathology. *Behav Res Ther.* 2008; 46(10):1105–1110. [PubMed: 18710699]
54. First, M.; Spitzer, RL.; Gibbon, M.; Williams, JBW. Structured Clinical Interview for DSM-IV Axis I Disorders-- patient ed. New York: New York State Psychiatric Institute; 1995. SCID-I/P
55. Koch S, Quadflieg N, Fichter M. Purging disorder: A comparison to established eating disorders with purging behaviour. *Eur Eat Disord Rev.* 2013; 21(4):265–275. [PubMed: 23629831]
56. Roberto CA, Grilo CM, Masheb RM, White MA. Binge eating, purging, or both: Eating disorder psychopathology findings from an internet community survey. *Int J Eat Disord.* 2010; 43(8):724–731. [PubMed: 19862702]
57. Keel PK, Wolfe BE, Gravener JA, Jimerson DC. Co-morbidity and disorder-related distress and impairment in purging disorder. *Psychol Med.* 2008; 38:1435–1442. [PubMed: 17825120]
58. Dalle Grave R, Calugi S, Marchesini G. Compulsive exercise to control shape or weight in eating disorders: Prevalence, associated features, and treatment outcome. *Compr Psychiatry.* 2008; 49(4): 346–352. [PubMed: 18555054]
59. Blomquist KK, Roberto CA, Barnes RD, White MA, Masheb RM, Grilo CM. Development and validation of the Eating Loss of Control Scale. *Psychol Assess.* 2014; 26(1):77. [PubMed: 24219700]
60. Latner JD, Mond JM, Kelly MC, Haynes SN, Hay PJ. The loss of control over eating scale: Development and psychometric evaluation. *Int J Eat Disord.* 2014; 47(6):647–659. [PubMed: 24862351]
61. Dossat AM, Bodell LP, Williams DL, Eckel LA, Keel PK. Preliminary examination of glucagon-like peptide-1 levels in women with purging disorder and bulimia nervosa. *Int J Eat Disord.* 2014; 47(6):647–659. [PubMed: 24862351]

62. Berg KC, Peterson CB, Frazier P, Crow SJ. Psychometric evaluation of the eating disorder examination and eating disorder examination-questionnaire: A systematic review of the literature. *Int J Eat Disord.* 2012; 45(3):428–438. [PubMed: 21744375]

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

Figure 1.

	Loss of Control Eating	Eating Episode Size	LOC x Size Interaction
Purging Frequency	Yes	No	Yes
EDE Total Score	Yes	No	No
BSQ	Yes	No	No
Disinhibition	Yes	Yes	No
Hunger	Yes	No	No
BDI	Yes	No	Yes
State Anxiety	Yes	No	No
Trait Anxiety	Yes	Yes	No
Negative Urgency	Yes	No	No
CIA	Yes	Yes	No
GAF	Yes	No	No

Figure 2.

Summary of Unique Associations of Loss of Control Eating Frequency and Eating Episode Size with Eating Pathology, Related Psychopathology, Personality, and Distress and Impairment Note: LOC = loss of control eating, EDE = Eating Disorder Examination, BSQ = Body Shape Questionnaire, BDI = Beck Depression Inventory, CIA = Clinical Impairment Assessment, GAF = Global Assessment of Functioning.

Table 1

Means, Standard Deviations, and Correlations of Loss of Control Eating Frequency and Eating Episode Size with Eating Pathology, Related Psychopathology, Personality, and Distress and Impairment

	N	Mean	SD	LOC frequency r	Episode Size r
LOC frequency	243	23.28	21.69	--	.41**
Episode Size	243	2207.63	1818.43	--	--
Purging frequency	243	29.28	23.13	.46**	.19*
EDE total	243	3.64	.92	.23**	.19*
BSQ	242	140.80	28.72	.23**	.20*
Restraint	229	16.06	3.96	-.17	-.10
Disinhibition	229	11.07	3.80	.57**	.46**
Hunger	229	7.81	3.81	.43**	.27**
Body Mass Index	242	22.35	1.86	-.10	-.04
BDI	227	14.29	9.98	.38**	.25**
State Anxiety	236	42.49	12.20	.22**	.18*
Trait Anxiety	233	48.11	11.50	.33**	.26**
Negative Urgency	87	2.62	.61	.51**	.24
CIA	73	21.75	9.49	.49**	.44**
GAF	238	58.14	9.14	-.36**	-.15*

Note: LOC = loss of control eating, EDE = Eating Disorder Examination, BSQ = Body Shape Questionnaire, BDI = Beck Depression Inventory, CIA = Clinical Impairment Assessment, GAF = Global Assessment of Functioning. Represent one-tailed tests

* $p < .01$,

** $p < .001$

Table 2

Frequencies (n) of combinations of loss of control eating and unusually large eating episodes

	SBE	OOE	
		Yes	No
OBE	Yes	7	82
	No	7	46
No OBE	Yes	2	76
	No	1	16

Note: The number of objective overeating episodes were coding as missing for 6 participants. OBE = objective binge episodes; SBE = subjective binge episode; OOE = objective overeating episode

Hierarchical Multiple Regression Analyses Examining Influence of Loss of Control Frequency, Episode Size, and their Interaction on Eating Pathology

Table 3

Step 1*	Purging Frequency			EDE Total			Body Shape Questionnaire			Disinhibition			Hunger		
	β	<i>t</i>	<i>p</i>	β	<i>t</i>	<i>p</i>	β	<i>t</i>	<i>p</i>	β	<i>t</i>	<i>p</i>	β	<i>t</i>	<i>p</i>
Parent Study	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
Group Code 1	-.02	-.29	.62	.12	1.66	.05	.22	3.04	.002	-.03	-.40	.66	.02	.31	.38
Group Code 2	.02	.27	.39	.14	1.87	.03	.14	1.93	.03	-.12	-1.89	.97	-.05	-.75	.77
Group Code 3	.01	.21	.42	.11	1.67	.05	.08	1.21	.11	--	--	--	--	--	--
BMI	.05	.82	.21	.10	1.55	.12	.10	1.54	.06	.11	1.97	.03	.03	.46	.32
Age	.03	.52	.30	-.02	-.22	.59	-.05	-.68	.75	-.02	-.36	.64	-.13	-2.01	.98
LOC Frequency	.45	7.31	<.001	.26	3.86	<.001	.28	4.29	<.001	.61	10.71	<.001	.47	7.55	<.001
Step 2*															
Episode Size	.01	.20	.42	.12	1.81	.04	.14	2.01	.02	.28	4.93	<.001	.11	1.71	.04
Step 3**															
LOC X Size	.36	5.94	<.001	-.06	-.85	.39	-.004	-.06	.96	-.10	-1.83	.07	-.01	-.17	.86

Note: BMI = body mass index; LOC = loss of control eating; EDE = Eating Disorder Examination;

* using one-tailed test;

** using two-tailed test;

Table 4

Hierarchical Multiple Regression Analyses Examining Influence of Loss of Control Frequency, Episode Size, and their Interaction on Related Psychopathology, Personality, Impairment, and Functioning

Step 1 *	Beck Depression Inventory			STAI-State Anxiety			STAI-Trait Anxiety			Clinical Impairment Assessment			Global Assessment of Functioning		
	β	<i>t</i>	<i>p</i>	β	<i>t</i>	<i>p</i>	β	<i>t</i>	<i>p</i>	β	<i>t</i>	<i>p</i>	β	<i>t</i>	<i>p</i>
Parent Study	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
Group Code 1	.06	.78	.22	.09	1.25	.11	.10	1.38	.08	.10	.86	.19	.05	.77	.78
Group Code 2	.06	.84	.20	.10	1.44	.08	.13	1.91	.03	--	--	--	-.18	-2.63	.005
Group Code 3	--	--	--	.10	1.45	.07	.06	.95	.17	--	--	--	.06	.92	.82
BMI	-.07	-1.08	.86	-.18	-2.70	.99	-.13	-2.07	.98	-.12	-1.12	.87	.16	2.64	.99
Age	-.11	-1.66	.95	-.10	-1.47	.93	-.10	-1.48	.93	.17	1.49	.07	-.10	-1.64	.05
LOC Frequency	.39	5.98	< .001	.22	3.31	< .001	.33	5.04	< .001	.51	4.87	< .001	-.26	-4.19	< .001
Step 2 *															
Episode Size	.12	1.78	.04	.13	1.83	.03	.17	2.54	.006	.28	2.39	.01	-.02	-.24	.41
Step 3 **															
LOC X Size	.18	2.65	.009	.00	-.003	.99	.16	2.38	.02	-.06	-.50	.62	-.03	-.45	.65

Note: BMI = body mass index; LOC = loss of control eating; STAI = State Trait Anxiety Inventory;

* using one-tailed test;

** using two-tailed test;