



Published in final edited form as:

*Compr Psychiatry*. 2013 July ; 54(5): 506–516. doi:10.1016/j.comppsy.2012.12.006.

## RESPONSE PATTERNS ON INTERVIEW AND QUESTIONNAIRE VERSIONS OF THE EATING DISORDER EXAMINATION AND THEIR IMPACT ON LATENT STRUCTURE ANALYSES

Kelly C. Berg, Ph.D.<sup>1</sup>, Sonja A. Swanson, Sc.M.<sup>2</sup>, E. Colleen Stiles-Shields, M.S.W.<sup>3</sup>, Kamryn T. Eddy, Ph.D.<sup>4</sup>, Carol B. Peterson, Ph.D.<sup>1</sup>, and Daniel Le Grange, Ph.D.<sup>2</sup>

<sup>1</sup>Department of Psychiatry, University of Minnesota, Minneapolis, MN

<sup>2</sup>Department of Epidemiology, Harvard School of Public Health, Boston, MA

<sup>3</sup>Department of Psychiatry, The University of Chicago, Chicago, IL

<sup>4</sup>Department of Psychiatry, Massachusetts General Hospital, Harvard Medical School, Boston, MA

### Abstract

**Objective**—The purpose of this investigation was to compare the latent structures of the interview (EDE) and questionnaire (EDE-Q) versions of the Eating Disorder Examination.

**Methods**—Participants were 280 children, adolescents, and young adults seeking eating disorder treatment. Two separate latent structure analyses (LSAs) were conducted; one used variables from the EDE as indicators and the other used the corresponding variables from the EDE-Q as indicators.

**Results**—The EDE and EDE-Q models both yielded four-class solutions. Three of the four classes from the EDE-Q model demonstrated moderate to high concordance with their paired class from the EDE model. Using the EDE-Q to detect the EDE, the sensitivity and specificity of measuring certain classes varied from poor (18.6%) to excellent (93.7%). The overall concordance was moderate ( $\kappa=.49$ ).

**Discussion**—These data suggest that LSAs using the EDE and EDE-Q may be directly compared; however, differences between results may represent inconsistencies in response patterns rather than true differences in psychopathology.

### 1. Introduction

In anticipation of the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) [1], researchers have used latent structure analysis<sup>1</sup> (LSA) [2, 3] to empirically derive subtypes of psychopathology, including eating disorders [4]. The goal of LSA is to use observable data to identify the underlying or latent classes that explain

© 2012 Elsevier Inc. All rights reserved.

**Publisher's Disclaimer:** This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

<sup>1</sup>*Latent class analysis (LCA)* and *latent profile analysis (LPA)* are two specific techniques subsumed under the general term *latent structure analysis (LSA)*. Overall, there is disagreement over the usage of these terms, particularly across fields of study (e.g., statistics, social sciences), and to avoid confusion, the general term *latent structure analysis* will be used in the current study to encompass all types of latent structure analyses.

patterns of observed variables, making it particularly well-suited to informing classification schemes. LSA examines the relationships between the observed variables (indicators) included in the analysis and then identifies the latent classes that best explain those relationships. LSA is based on the assumption that an exhaustive set of mutually exclusive unobservable latent classes exist and that while membership in those classes may not be measured directly, class membership is related to other variables that are observable. In the field of eating disorders, these types of analyses have been widely used to inform DSM-5 [4]. Since 1998, 31 LSA studies have been conducted in the field of eating disorders to identify broad classes of eating disorders as well as subtypes of those classes (see Table 1).

Although LSA provides a powerful tool for researchers interested in classification, results cannot be generalized without replication. Unfortunately, despite the growing number of LSA studies, inconsistent methodologies make model comparison across studies difficult [5]. For example, the use of different samples (e.g., clinical versus community) or different indicators (e.g., cognitive symptoms versus behavioral symptoms versus personality dimensions) could dramatically change the latent classes of psychopathology identified in the analyses. One of the most prolific inconsistencies among LSA studies is the use of different instruments to assess the variables used as indicators [5]. In the field of eating disorders, the inconsistent measurement of indicators is particularly problematic. Of the 31 LSA studies that have been conducted in the field of eating disorders, the most commonly used assessment was the Eating Disorder Examination [6, 7], a clinician-administered interview, which was utilized in just nine (26.7%) of the studies.

An even more fundamental discrepancy across LSA studies is the inconsistent use of semi-structured interviews versus self-report questionnaires to measure the variables used as indicators. Of the 31 LSA studies in eating disorders, 16 used indicators measured by semi-structured interviews [e.g., 8, 9] whereas the other 15 used indicators measured by self-report questionnaires [e.g., 10, 11] (see Table 1). Given that LSA requires relatively large samples to estimate the structures reliably [12], data are often used from samples of convenience rather than studies specifically designed for LSA. Thus, these inconsistencies in methodology are undoubtedly due, in part, to issues of practicality. However, instrument selection is one of the more easily controlled variables in research and yet, little attention has been paid to whether LSA studies using different types of assessments, specifically semi-structured interviews versus self-report questionnaires, can be directly compared.

The inconsistent use of semi-structured interview- and questionnaire-based assessments in LSA studies is potentially problematic as the two methods of measurement can result in differential response patterns. For example, when used to assess symptoms of mental illness, significant differences between semi-structured interviews and self-report questionnaires have been found for symptom presence [e.g., 13], severity [e.g., 14], and frequency [e.g., 15, 16]. Additionally, even when two instruments are nearly identical, the method of measurement can have a substantial impact on participants' responses. For example, the interview (EDE) and questionnaire (EDE-Q) versions of the Eating Disorder Examination have both demonstrated validity as measures of eating disorder cognitions and are nearly identical with regard to content, language, and rating scales. Yet, respondents score on average 0.3 to 0.6 standard deviations higher on the EDE-Q subscales than the EDE subscales and some studies have reported respondents scoring as much as 1.76 standard deviations higher on the EDE-Q subscales [17, 18].

Unfortunately, most research on the convergence of scores on semi-structured interviews and self-report questionnaires has focused on the convergence of individual construct scores across respondents (e.g., convergence of scores on the Restraint subscale of the EDE and EDE-Q in a particular sample) rather than individual differences in response patterns across

multiple constructs (e.g., Do respondents who score higher on the EDE-Q Restraint subscale than the EDE Restraint subscale also score higher on the other EDE-Q subscales? How do differences in response patterns between semi-structured interviews and self-report questionnaires impact diagnoses?). However, a few studies have demonstrated that differences between scores on semi-structured interviews and self-report questionnaires can result in poor diagnostic agreement [e.g., 19–21], including recent research on the diagnostic concordance of the EDE and EDE-Q [22]. Overall, these research findings illustrate that both individual scores and overall response patterns can vary between semi-structured interview- and questionnaire-based instruments, even when both instruments have been validated. Given that LSA specifically evaluates observed associations between indicators, inherent differences between individuals' response patterns on semi-structured interviews and self-report questionnaires may result in discrepancies between the latent models of semi-structured interviews and self-report questionnaires, rendering model comparisons unusable. However, whether differences between the response patterns on semi-structured interviews and self-report questionnaires ultimately result in discrepancies between LSA models is unclear because there is no published research comparing the latent structures of semi-structured interviews and self-report questionnaires.

### 1.1 Overview of the Current Study

The purpose of the present study was to compare the latent structure of an investigator-based semi-structured interview to that of a self-report questionnaire, holding the sample and indicators used constant. The Eating Disorder Examination (EDE) [6, 7], a clinician-administered interview that assesses cognitive and behavioral symptoms associated with eating disorders, is one of the most commonly used and widely researched assessments of eating disorder pathology [23]. A questionnaire version of the EDE (EDE-Q) [24, 25] was also developed because the interview is lengthy to administer and requires significant assessor training. The interview and questionnaire versions of the EDE include nearly identical item content, wording, and rating scales and research has found significant correlations between EDE and EDE-Q scores [17]. As a result, the EDE and EDE-Q are thought to measure the same constructs with the only difference between them being the method with which the instruments are administered. Given the structural similarities between the two instruments, the EDE and EDE-Q present a unique opportunity to examine whether differences in response patterns on semi-structured interviews and self-report questionnaires result in discrepancies between the latent structures of these types of instruments.

## 2. Methods

### 2.1 Participants

Data were collected from 280 treatment-seeking patients at The University of Chicago Eating Disorders program between 1999 and 2009. The Eating Disorders program is an outpatient treatment and research clinic serving children, adolescents, and young adults through empirically-supported eating disorder treatment. Participants ranged in age from 8 to 25 years, with a mean age of  $16.37 \pm 3.27$  years. The majority of participants were female (92.1%) and most identified as Caucasian (70.7%), followed by Latino (12.5%), and African American (9.3%), with other groups constituting less than 8% of the sample. Based on clinical interview administered by trained graduate level interviewers, most participants were diagnosed with eating disorder not otherwise specified (EDNOS; 61.6%), followed by bulimia nervosa (BN; 20.4%), anorexia nervosa (AN; 16.5%), and no eating disorder diagnosis (1.4%). With regard to eating disorder diagnosis, the composition of the sample was consistent with findings from other eating disorder clinics [26].

## 2.2 Measures

**2.2.1 Eating Disorder Examination**—The Eating Disorder Examination (EDE) [6,7] is a semi-structured interview used to assess both the cognitive and behavioral symptoms related to eating disorders. The cognitive symptoms of eating disorders (e.g., fear of weight gain, overevaluation of shape) are assessed for the past 28 days using a 7-point Likert scale ranging from 0 to 6, with higher scores indicating more severe eating disorder pathology. The behavioral symptoms of eating disorders are assessed by counting the specific frequencies with which binge eating and compensatory behaviors have occurred during the past three months. However, for this study, only data from the past month was used to be consistent with the data compiled by the EDE-Q.

After a comprehensive review of the published research that has used LSA to classify subtypes of eating disorders in a broad eating disorder sample, indicators were selected that met the following criteria: 1) the indicator was used in more than one study and 2) the indicator could be measured by both the EDE and EDE-Q. The following individual items that assess cognitive symptoms of eating disorders were used as indicators and dichotomized such that scores of 0–3 were coded as “symptom absent” and scores of 4–6 were coded as “symptom present”: “fear of weight gain,” “overevaluation of shape,” “overevaluation of weight,” “dietary rules,” and “preoccupation with shape and weight”. Variables were dichotomized to stay consistent with the majority of LSA studies that have been conducted in eating disorders and the point of dichotomization was chosen to reflect the threshold for clinical significance indicated by the authors of the EDE [7]. Given that the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders, text revision (DSM-IV-TR) [27] does not distinguish between overevaluation of shape and overevaluation of weight, these two items were combined such that if the respondent scored 4–6 on either item, the item was coded as “symptom present”.

The following items assessing behavior frequency were also used as indicators and dichotomized such that scores of 0–3 (i.e., <1x/week) were coded as “symptom absent” and scores 4 (i.e., 1x/week) were coded as “symptom present”: frequency of Objective Bulimic Episodes (OBEs), frequency of Subjective Bulimic Episodes (SBEs), and frequency of purging behaviors (i.e., total frequency of self-induced vomiting, laxative use, and diuretic use). The threshold of once per week was chosen for the behavioral symptoms to correspond with the criteria proposed in DSM-5 for BN and binge eating disorder (BED). The frequency of excessive exercise and fasting were not used as indicators because the EDE-Q does not assess fasting and there is little data on the reliability and validity of the items that assess frequency of excessive exercise on the EDE and EDE-Q [28].

The five cognitive items chosen as indicators in the current study have demonstrated inter-rater reliability, with coefficients ranging from .90 to 1.00 [29]. Behavioral frequency scores for Objective Bulimic Episodes and self-induced vomiting have also demonstrated test-retest reliability (.70–.97) and inter-rater reliability (.98–1.0). Previous research has found that scores on the EDE can be used to distinguish between cases and non-cases of eating disorders [16, 30, 31]. A comprehensive review of the psychometric properties of the EDE and EDE-Q can be found in Berg et al. (2012).

**2.2.2 Eating Disorder Examination-Questionnaire**—The Eating Disorder Examination-Questionnaire (EDE-Q) [24, 25] is a 36-item self-report questionnaire based on the EDE interview. Each item pair from the EDE and EDE-Q includes nearly identical language and is rated on the same scales (e.g., cognitive symptoms are rated on a 7-point Likert scale ranging from 0–6, behavioral symptoms are measured with a frequency count). The primary differences between the EDE and EDE-Q are that the EDE allows the interviewer to ask additional questions to clarify responses and the EDE-Q only assesses

symptoms during the past 28 days. The same items that were chosen as indicators for the EDE model were chosen as indicators in the EDE-Q model: “fear of weight gain,” “overevaluation of shape,” “overevaluation of weight,” “dietary rules,” and “preoccupation with shape and weight”, frequency of Objective Bulimic Episodes (OBEs), frequency of Subjective Bulimic Episodes (SBEs), and frequency of purging behaviors. These items have demonstrated significant, positive correlations with their item pairs from the EDE [17], and the behavioral frequency scores for Objective Bulimic Episodes and self-induced vomiting have also demonstrated test-retest reliability (.68–.92) [28]. Scores on the EDE-Q have demonstrated the ability to distinguish between non-eating disorder and eating disorder cases [16]. Additional detail regarding the psychometric properties of the EDE and EDE-Q can be found in Berg et al. (2012).

**2.2.3 Physical Assessment**—The weight and height of each participant was measured at the time of assessment. Weight was measured using a calibrated digital or balance-beam scale and all patients were weighed in light, indoor clothing. Height was obtained using a calibrated stadiometer. Percent of ideal body weight (%IBW) was defined as current weight divided by 50<sup>th</sup> centile weight. The 50<sup>th</sup> centile weight for adolescent participants (age 19) was calculated using the Center for Disease Control’s Child and Adolescent Body Mass Index Percentile Calculator which takes into account both age and gender [32]. For adult participants (age 20), the 50<sup>th</sup> centile weight was based on gender and defined as the median weight published in the 1959 Metropolitan Life Insurance Tables [33]. As an indicator, %IBW was categorized ordinally (i.e., <85%, 85–94.9%, 95%) and used in both models.

### 2.3 Procedure

All participants completed the EDE, EDE-Q, and physical assessments prior to the start of treatment. These assessments were conducted on the same day and the order in which the assessments were given was not controlled or counterbalanced. Written consent for patients over 18 years of age or parental/guardian consent and adolescent assent for patients under 18 years of age were obtained for the use of these data in analysis. Refusal to consent did not alter the assessment or treatment of any patients. This study was approved by the Institutional Review Board at The University of Chicago Medical Center.

### 2.4 Statistical Method

**2.4.1 Latent Structure Analyses**—Latent structure models were fit in parallel for the EDE and EDE-Q data using the software MPlus Version 5.1 [34]. Using the eight indicator variables as described above, models were fit with increasing numbers of classes. In both the EDE set of models and the EDE-Q set of models, the optimal number of classes was determined by minimizing the sample-size adjusted Bayesian Information Criterion (aBIC) [35], with additional consideration given to both the Bayesian Information Criterion (BIC) [36] and Akaike Information Criterion (AIC) [37]. The choice of the aBIC in model selection was based off a simulation study indicating that the aBIC achieved over 80% accuracy<sup>2</sup> in sample sizes of 300 for unbalanced four-class structures, whereas the BIC and AIC had much poorer accuracy at such sample sizes for the same designs [38]. Other than the use of equivalent constructs for indicator variables, no restrictions were made on the EDE and EDE-Q models in these analyses (e.g., consistent number of classes). Across the 15 indicators used in the EDE and EDE-Q models, the amount of missing data ranged from 0.0% to 7.7% (median=1.1%) for each individual indicator. There was no significant difference between the EDE and EDE-Q models with regard to the amount of missing data.

<sup>2</sup>In simulation studies, “over 80% accuracy” indicates that the criterion chose the correct number of classes more than 80% of the time.

For both models, all available information (i.e., not listwise deletion) was used under a maximum likelihood framework with robust standard errors.

**2.4.2 Comparison of Latent Structure Analyses**—Once the best-fitting latent structure models were finalized for the EDE and EDE-Q data, participants were assigned to classes in each model based on their maximum posterior probabilities. These class assignments were used to assess the concordance of the structures, including estimates of the pair-wise sensitivity and specificity of the classes using the EDE model as the “gold standard” as well as kappa and total classification accuracy. For ease of discussion as well as in the estimation of kappa and total classification accuracy, each of the latent classes from the EDE model were matched to classes in the EDE-Q model. These pairs were subjectively matched after reviewing the identified symptom patterns and concordance of class assignments for all pairings.

### 3. Results

#### 3.1 Concordance of Model Structures

LSA models using 1- to 6-class solutions were evaluated separately for the EDE and EDE-Q models. In both sets of models, AIC and ABIC were lowest for a 4-class solution whereas BIC was lowest for a 2-class solution (see Table 2). Given that aBIC is heavily favored in sample sizes under 300 when class sizes are not expected to be balanced [38]; the 4-class solution was chosen for both the EDE and EDE-Q models. For ease of discussion, in the EDE model, the latent classes were labeled 1 through 4 whereas in the EDE-Q model, the latent classes were labeled A through D.

#### 3.2 Concordance of Symptom Patterns

Latent classes (LC) -1, -2, and -4 from the EDE model were characterized by relatively similar symptom classes as LC-A, -B, and -D from the EDE-Q model respectively. In contrast, LC-3 and LC-C did not appear to represent the same symptom classes (see Table 3 and Figure 1). LC-1/LC-A<sup>3</sup> consisted primarily of individuals who endorsed clinically significant eating disorder cognitions (with the exception of “preoccupation with shape/weight”, the probability of endorsement = .70) and denied regular binge eating or purging (probability of endorsement = .25). LC-2/LC-B was characterized by individuals who endorsed both clinically significant eating disorder cognitions (probability of endorsement = .69) and regular binge eating and purging (probability of endorsement > .50). Finally, LC-4/LC-D was typified by a denial of any eating disorder symptoms (probability of endorsement < .25). Although LC-3 and LC-C were both characterized by atypical symptom classes, there was little concordance between the two symptom classes. With regard to weight status, LC-2/LC-B and LC-3/LC-C were primarily characterized by normal-weight individuals whereas individuals assigned to LC-1/LC-A or LC-4/LC-D were more likely to be low-weight.

#### 3.3 Concordance of Class Assignments

In the EDE model, 66 (23.6%) participants were assigned to LC-1, 102 (36.4%) to LC-2, 43 (15.4%) to LC-3, and 69 (24.6%) to LC-4. Similarly, 81 (28.9%) participants were assigned to LC-A, 98 (35.0%) to LC-B, 23 (8.2%) to LC-C, and 78 (27.9%) to LC-D in the EDE-Q model. When using the EDE-Q to detect class assignment in the EDE model, the specificities for all four classes were high, ranging from 80.4% to 93.7%, but the sensitivities of the classes varied substantially, ranging from 18.6% to 84.1% (see Tables 5

<sup>3</sup>When two classes are meant to be interpreted as a pair, the notation “LC-1/LC-A” will be used. When two classes are meant to be interpreted as two individual classes, the following notation will be used: “LC-1 and LC-A”.

and 6). Overall, there was moderate agreement between the two models with regard to class assignments ( $\kappa=.49$ ; total classification accuracy=63.2%).

## 4. Discussion

The purpose of the current study was to compare the latent structure of the EDE interview to the EDE-Q, specifically examining the concordance of their model structures, the symptom patterns of the identified latent classes, and class assignments. With regard to the number of classes, there was perfect concordance between the EDE and EDE-Q models as 4-class solutions provided the best fit for both models. With regard to the concordance of symptom patterns, three of the four latent classes from the EDE model appeared to be characterized by similar symptom patterns as their counterparts from the EDE-Q model. Although the symptom patterns of these three pairs of classes were not identical, the differences between the two models appeared to be minimal. For example, the probability that fear of weight gain was endorsed in LC-1 was .732 whereas the probability that it was endorsed in LC-A was .911. Given that it was still overwhelmingly likely that fear of weight gain was endorsed in both latent classes, this discrepancy did not appear to result in overall differences between the symptom patterns of LC-1 and LC-A. Thus, descriptively, it appears that three distinct symptom patterns were identified by the LSA regardless of whether participants' symptoms were assessed by the EDE or the EDE-Q. In contrast, there appeared to be poor concordance between the symptom patterns of LC-3 and LC-C. Both latent classes appear to be characterized by atypical symptom patterns; however, they did not appear to represent the same symptom presentation. With regard to the concordance of class assignments, three of the four pairs of latent classes demonstrated moderate to strong concordance. In contrast, there was poor concordance of class assignments for LC-3/LC-C.

### 4.1 Implications

These results have implications for understanding whether there are differences between overall response patterns on the EDE and EDE-Q. If participants' response patterns on EDE and EDE-Q were exactly the same, the results of the two LSA models, including the symptom patterns and class memberships<sup>4</sup>, should be identical. Additionally, because the EDE and EDE-Q were completed by the same participants on the same day, differences between the two models cannot be due to true differences in psychopathology. In other words, participants could not have presented with one type of pathology while completing the EDE and a different type of pathology while completing the EDE-Q. Thus, any differences between the LSA models identified in the current study would indicate differences in participants' overall response patterns on the EDE and EDE-Q.

Although a 4-class solution was derived for both the EDE and EDE-Q models, differences emerged between the symptom patterns of the classes as well as the class memberships of the two models. For example, although the EDE and EDE-Q models identified three pairs of classes with relatively similar symptom patterns, class membership was not identical across the two models. Additionally, the fact that LC-3 and LC-C were both characterized by atypical symptom patterns is not sufficient to explain the poor concordance between the two classes. As stated above, if the response patterns on the EDE and EDE-Q were identical, one would expect to find identical symptom patterns across the two models even if the classes represented atypical symptom patterns; thus, these data suggest that there were differences between participants' overall response patterns on the EDE and EDE-Q.

---

<sup>4</sup>Although class membership in LSA is based on posterior probabilities and is thus vulnerable to error, if all participants' response patterns were identical on the EDE and EDE-Q, participants would be assigned to the exact same classes in the two models.

These results also have implications for researchers interested in comparing latent structure models of eating disorder pathology across studies [5], an objective that may be particularly relevant to understanding the validity of DSM-5 classification [4]. Overall, the results demonstrate that similar classes of eating disorder pathology may be derived regardless of whether the indicators were measured by the EDE or EDE-Q. These findings provide preliminary evidence that latent structure models using items from the EDE as indicators can be directly compared to latent structure models using items from the EDE-Q. However, these data also suggest that comparisons of latent structure models across studies should be interpreted cautiously. For example, the symptom patterns of LC-4/LC-D were characterized by a denial of any clinically significant eating disorder symptoms, which is a symptom pattern that has been derived in other LSA studies [11, 39, 40]. The consistency with which this symptom pattern has been found may suggest several possible interpretations. First, it may indicate that this symptom presentation is a valid eating disorder subtype, which would imply that there are some individuals with eating disorders whose symptom presentations are not captured by current conceptualizations of eating disorders. Second, it may suggest that there are individuals suspected of having an eating disorder who do not, in fact, have one. However, a third explanation may be that there is a subgroup of individuals with eating disorders who will (either inadvertently or deliberately) deny or minimize symptoms consistently, regardless of the method of measurement.

Although these data demonstrate that LSA models may identify similar classes of eating disorders regardless of whether the EDE or EDE-Q is used to measure the indicators, these data also illustrate that the method of measurement can lead to discrepancies between LSA models. For example, despite using the same indicators, which were nearly identical with regard to wording and rating scales, the two models in the current study identified a discordant pair of classes (i.e., LC-3 and LC-C). The lack of concordance between LC-3 and LC-C is particularly striking given that the EDE and EDE-Q models were based on measurements of the same sample taken on the same day, which precludes the possibility that the two models each identified a unique subtype of eating disorder pathology. Rather, as stated above, it is hypothesized that discrepancies between the EDE and EDE-Q models reflect different response patterns on the two instruments. Thus, the results from this study illustrate that discrepancies between LSA models may simply represent inconsistencies between response patterns the instruments used to measure the indicators rather than true differences in pathology.

#### 4.2 Strengths and Limitations

This study has several strengths. It is the first to examine whether the EDE and EDE-Q yield the same latent classes in a latent structure analysis. This is critically important for model comparison given the inconsistent use of semi-structured interviews versus self-report questionnaires across eating disorder LSA studies. The EDE and EDE-Q provide a unique opportunity to compare the latent structures of a semi-structured interview and a self-report questionnaire because the two are nearly identical with regard to item content, wording, and scoring. Additionally, the two assessments were completed by the same participants and on the same day. Thus, any differences between the latent classes yielded by the EDE and EDE-Q would be attributable to discrepancies between response patterns on the EDE and the EDE-Q rather than methodological inconsistencies or true differences in pathology. Finally, data were collected from a heterogeneous clinic sample of children, adolescents and young adults reporting a broad range of eating disorder symptoms, thereby potentially increasing the generalizability of the findings.

There were also limitations to this study. Although the primary goal of the current study was to examine and describe the concordance between the latent structures of the EDE and EDE-Q, direct statistical comparisons are not feasible using LSA and comparisons of the EDE and



EDE-Q models in this study are descriptive and not inferential. Additionally, these data do not indicate what factors accounted for the concordance or lack of concordance between the two models. It is possible that the reliability of the instruments may have placed constraints on the extent to which the latent structures of the EDE and EDE-Q could converge. It is also worth noting that the EDE and EDE-Q scores were dichotomized prior to analysis to remain consistent with previous investigations using LSA in the field of eating disorders. Although the points of dichotomization were chosen because they are thought to represent the thresholds of clinical significance, the point of dichotomization could have impacted the extent to which the models converged. In addition, the majority of the participants were Caucasian females seeking treatment, limiting the generalizability of these findings. Finally, individuals' class memberships were based on posterior probabilities and should not be considered absolute. Although approximately 80% of the participants were correctly classified (entropy for EDE model=.73, entropy for EDE-Q model=.82), misclassifications could have increased or decreased the concordance of the two models.

These data may have also been impacted by methodological limitations. First, because the primary objective of this study was to explore a methodological issue with LSA rather than to identify classes of eating disorders, validation analyses were not conducted on the identified classes. Thus, the classes identified in this study should not necessarily be interpreted as valid classes of eating disorders<sup>5</sup>. Second, data were collected in the context of an intake assessment at an outpatient clinic, which precluded the possibility of controlling or counterbalancing the order in which the EDE and EDE-Q were administered. It is generally believed that administering the EDE prior to the EDE-Q would inflate the concordance of the two assessments because definitions of key variables are provided during the interview (e.g., binge eating) that may bias responding on the questionnaire. Thus, because it is impossible to know what percentage of the time the EDE was given first, these results may represent either an overestimation or an underestimation of the concordance of the EDE and EDE-Q models. Second, although the amount of missing data was minimal, the presence of any missing data may have impacted the results. However, because there was no apparent pattern of omissions (e.g., more missing data on the EDE or the EDE-Q), it is unlikely that missing data affected one model more than the other.

One final issue worth noting is that 9.3% of the sample was 12-years-old or younger. It is possible that age may impact the concordance of response patterns on the EDE and EDE-Q and, consequently, the concordance of the EDE and EDE-Q models. However, at this point, there is no empirical evidence to support this hypothesis. To our knowledge, only one study has examined whether age impacts the extent to which scores on the EDE and EDE-Q are discrepant [41], finding that age did not predict discrepancy scores in an adult sample. Furthermore, recent research using a sample of 8,594 females demonstrated stability of latent classes across the following age groups: (a) 9–12 years-old, (b) 13–15 years-old, (c) 16–18 years old, (d) 19–22 years old, (e) 23–26 years old [42]. Given these data, we had no a priori hypotheses about whether the concordance of the LSA models would be stable across different age groups. Although there were concerns that the younger participants (ages 8–12) would have difficulty understanding the EDE-Q content, correlations between scores on the EDE and EDE-Q were found to be similar between the younger (ages 8–12; range of  $r$ s=.20–.94) and older participants (ages 13–25; range of  $r$ s=.25–.71). These results indicate that the younger participants responded similarly on the EDE and EDE-Q (or at least as similarly as older participants), which suggests that the two instruments were understood equally well. Additionally, a post hoc analysis indicated that participants assigned to LC-4 and LC-D were significantly younger (mean ages=15.01 and 15.05 years

---

<sup>5</sup>The replication of three of the four classes could be considered a type of validation analysis.

respectively) than those assigned to LC-2, -3, -B, and -C (mean ages=17.00, 17.53, 17.73, and 17.17 years respectively;  $F(3, 276)=7.75, p<.001$ ). Given that the concordance between LC-4/LC-D was the strongest, these data may suggest that the response patterns on semi-structured interviews and self-report questionnaires are more concordant in younger samples. However, due to the modest sample size, subgroup analyses were not possible. Further research is needed to examine response pattern discrepancies across different age groups.

### 4.3 Conclusions

Despite these limitations, this study represents the first comparison of the latent structure of the EDE and the EDE-Q in particular, and the first time an LSA model using indicators from a semi-structured interview has been compared to an LSA model using indicators from a self-report questionnaire more generally. As such, these data represent a critically important step towards understanding the extent to which findings from different LSA investigations can be integrated across studies. Overall, the results suggest that LSA models using the EDE and EDE-Q may yield similar results. However, caution should also be used when interpreting similarities between study results (e.g., consistently finding a subgroup that denies eating disorder behaviors and cognitions) as such findings may provide support for the validity of specific subtypes of eating disorders and consequently, possible DSM-5 eating disorder diagnoses; or, such similarities may merely indicate that certain subgroups of patients will endorse or deny certain eating disorder symptoms regardless of how the symptoms are assessed. Furthermore, discrepancies between findings may indicate true differences in psychopathology between samples (e.g., cultural differences in eating disorder pathology or differences in eating disorder pathology reported in community versus clinical samples); or, such discrepancies may reflect differences in response patterns across different methods of measurement. Given that these results only represent one comparison of the latent structures of a semi-structured interview and a self-report questionnaire, further research is needed before these results can be generalized to other samples, instruments, and fields of psychopathology.

### References

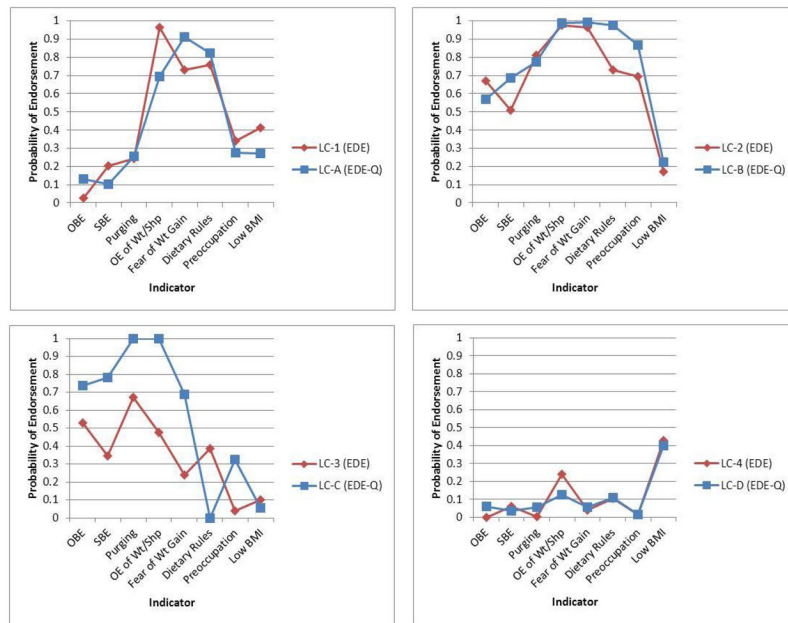
1. American Psychiatric Association. DSM-5 Development. 2012. Available at: [www.dsm5.org](http://www.dsm5.org)
2. Lazarsfeld, PF.; Henry, NW. Latent Structure Analysis. Boston, MA: Houghton Mifflin; 1968.
3. McCutcheon, AL. Latent Class Analysis. Newbury Park, CA: Sage Publications; 1987.
4. Striegel-Moore, RH.; Walsh, BT.; Wonderlich, SA.; Mitchell, JE., editors. Developing an Evidence-Based Classification of Eating Disorders: Scientific Findings for DSM-5. Arlington, VA: American Psychiatric Association; 2011.
5. Crow, SJ.; Swanson, SA.; Peterson, CB.; Crosby, RD.; Wonderlich, SA.; Mitchell, JE. Latent structure analyses of eating disorder diagnoses: Critical review of results and methodological issues. In: Striegel-Moore, RH.; Walsh, BT.; Wonderlich, SA.; Mitchell, JE., editors. Developing an Evidence-Based Classification of Eating Disorders: Scientific Findings for DSM-5. Arlington, VA: American Psychiatric Association; 2011. p. 103-120.
6. Fairburn, CG.; Cooper, Z.; O'Connor, M. Eating Disorder Examination. In: Fairburn, CG., editor. Cognitive Behavior Therapy and Eating Disorders. 16.0D. New York: Guilford Press; 2008.
7. Fairburn, CG.; Cooper, Z. The Eating Disorder Examination. In: Fairburn, CG.; Wilson, GT., editors. Binge Eating: Nature, Assessment, and Treatment. 12. New York: Guilford Press; 1993. p. 317-360.
8. Bulik CM, Sullivan PF, Kendler KS. An empirical study of the classification of eating disorders. *American Journal of Psychiatry*. 2000; 157:886–895. [PubMed: 10831467]
9. Keel PK, Fichter M, Quadflieg N, Bulik CM, Baxter MG, Thornton L, et al. Application of a latent class analysis to empirically define eating disorder phenotypes. *Archives of General Psychiatry*. 2004; 61:192–200. [PubMed: 14757596]

10. Pinheiro AP, Bulik CM, Sullivan PF, Machado PPP. An empirical study of the typology of bulimic symptoms in young portuguese women. *International Journal of Eating Disorders*. 2008; 41:251–258. [PubMed: 18095310]
11. Mitchell JE, Crosby RD, Wonderlich SA, Hill L, Le Grange D, Powers P, et al. Latent profile analysis of a cohort of patients with eating disorder not otherwise specified. *International Journal of Eating Disorders*. 2007; 40:595–598.
12. Yang C. Evaluating latent class analysis models in qualitative phenotype identification. *Computational Statistics and Data Analysis*. 2006; 50:1090–1104.
13. Prinstein MJ, Nock MK, Spirito A, Grapentine WL. Multimethod assessment of suicidality in adolescent psychiatric inpatients. *Journal of the American Academy of Child & Adolescent Psychiatry*. 2001; 40:1053–1061. [PubMed: 11556629]
14. Woodruff-Borden J, Jeffery SE, Bourland SL, Brothers AJ, Albano AM. Patient self-report in the assessment of panic disorders: Comparison with interview-derived clinician ratings. *Journal of Nervous and Mental Disease*. 2000; 188:308–310. [PubMed: 10830570]
15. Wilfley DE, Schwartz MB, Spurrell EB, Fairburn CG. Assessing the specific psychopathology of binge eating disorder patients: Interview or self-report? *Behav Res Ther*. 1997; 35(12):1151–1159. [PubMed: 9465449]
16. Mond JM, Hay PJ, Rodgers B, Owen C, Beumont PJV. Validity of the Eating Disorder Examination Questionnaire (EDE-Q) in screening for eating disorders in community samples. *Behav Res Ther*. 2004; 42(5):551–567. [PubMed: 15033501]
17. Berg KC, Peterson CB, Frazier P, Crow SJ. Convergence of scores on the interview and questionnaire versions of the Eating Disorder Examination: A meta-analytic review. *Psychological Assessment*. 2011; 23:714–724. [PubMed: 21517194]
18. Grilo CM, Masheb RM, Wilson GT. Different methods for assessing the features of eating disorders in patients with binge eating disorder: A replication. *Obesity Research*. 2001; 9:418–422. [PubMed: 11445665]
19. Zimmerman M, Coryell WH. Diagnosing personality disorders in the community: A comparison of self-report and interview measures. *Archives of General Psychiatry*. 1990; 47:527–531. [PubMed: 2350205]
20. Reeves JC, Large RG, Honeyman M. Parasuicide and depression: A comparison of clinical and questionnaire diagnoses. *Australian and New Zealand journal of psychiatry*. 1985; 19:30–33. [PubMed: 3859282]
21. Angus LE, Marziali E. A comparison of three measures for the diagnosis of borderline personality disorder. *The American Journal of Psychiatry*. 1988; 145:1453–1454. [PubMed: 3189609]
22. Berg KC, Stiles-Shields EC, Swanson SA, Peterson CB, Lebow J, Le Grange D. Diagnostic concordance of the interview and questionnaire versions of the Eating Disorder Examination. *International Journal of Eating Disorders*. 2011 Aug 8. Advance online publication. 10.1002/eat20948
23. Wilson, GT. Assessment of Binge Eating. In: Fairburn, CG.; Wilson, GT., editors. *Binge Eating: Nature, Assessment, and Treatment*. 12. New York: Guilford Press; 1993.
24. Fairburn CG, Beglin SJ. Assessment of eating disorders: Interview or self-report questionnaire? *Int J Eat Disord*. 1994; 16(4):363–370. [PubMed: 7866415]
25. Fairburn, CG.; Beglin, S. *Eating Disorder Examination Questionnaire*. In: Fairburn, CG., editor. *Cognitive Behavior Therapy and Eating Disorders*. New York: Guilford Press; 2008.
26. Fairburn CG, Cooper Z, Bohn K, O'Connor ME, Doll HA, Palmer RL. The severity and status of eating disorder NOS: Implications for DSM-V. *Behaviour research and therapy*. 2007; 45:1705–1715. [PubMed: 17374360]
27. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. 4. Washington, D.C: APA; 2000. Text Revision (DSM-IV-TR)
28. 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. *International Journal of Eating Disorders*. 2012; 45:438.
29. Cooper Z, Fairburn CG. 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.

30. Cooper Z, Cooper PJ, Fairburn CG. The validity of the Eating Disorder Examination and its subscales. *The British journal of psychiatry*. 1989; 154(6):807–812. [PubMed: 2597887]
31. Wilfley DE, Schwartz MB, Spurrell EB, Fairburn CG. Using the Eating Disorder Examination to identify the specific psychopathology of binge eating disorder. *Int J Eat Disord*. 2000; 27(3):259–269. [PubMed: 10694711]
32. Centers for Disease Control and Prevention. BMI Percentile Calculator for Child and Teen. 2010. Available at: <http://apps.nccd.cdc.gov/dnpabmi/>
33. Halmi KA, Agras WS, Crow SJ, Mitchell J, Wilson GT, Bryson SW, et al. Predictors of treatment acceptance and completion in anorexia nervosa. *Archives of General Psychiatry*. 2005; 62:776–781. [PubMed: 15997019]
34. Muthen, LK.; Muthen, BO. Mplus. 2008.
35. Sclove LS. Application of model-selection criteria to some problems in multivariate analysis. *Psychometrika*. 1987; 52:333–343.
36. Schwartz G. Estimating the dimension of a model. *Annals of Statistics*. 1978; 6:461–464.
37. Information theory and an extension of the maximum principle. 2nd International Symposium on Information Theory; 1973.
38. Swanson SA, Lindenber K, Bauer S, Crosby RD. A Monte Carlo investigation of factors influencing latent class analysis: An application to eating disorder research. *International Journal of Eating Disorders*. 2011 Aug 31. Advanced online publication.
39. Eddy KT, Crosby RD, Keel PK, Wonderlich SA, Le Grange D, Hill L, et al. Empirical identification and validation of eating disorder phenotypes in a multisite clinical sample. *The Journal of Nervous and Mental Disease*. 2009; 197:41–49. [PubMed: 19155809]
40. Eddy KT, Le Grange D, Crosby RD, Hoste RR, Doyle AC, Smyth A, et al. Diagnostic classification of eating disorders in children and adolescents: How does DSM-IV-TR compare to empirically-derived categories? *Journal of the American Academy of Child & Adolescent Psychiatry*. 2010; 49:277–287. [PubMed: 20410717]
41. Black CMD, Wilson GT. Assessment of eating disorders: Interview versus questionnaire. *Int J Eat Disord*. 1996; 20(1):43–50. [PubMed: 8807351]
42. Swanson, SA.; Horton, N.; Crosby, RD.; Micali, N.; Sonneville, K.; Eddy, K.; Field, A. A latent class analysis to empirically examine eating disorders through development stages. Poster presented at the Society for Epidemiologic Research (SER). 45th Annual Meeting; 2012, June;
43. Cain AS, Epler AJ, Steinley D, Sher KJ. Stability and change in patterns of concerns related to eating, weight, and shape in young adult women: A latent transition analysis. *Journal of Abnormal Psychology*. 2010; 119:255–267. [PubMed: 20455598]
44. Crosby, RD.; Fichter, MM.; Quadflieg, N.; Wonderlich, SA. Validating eating disorder classification models with mortality and recovery data. In: Striegel-Moore, RH.; Walsh, BT.; Wonderlich, SA.; Mitchell, JE., editors. *Developing an Evidence-Based Classification of Eating Disorders: Scientific Findings for DSM-5*. Arlington, VA: American Psychiatric Association; 2011. p. 133-144.
45. Crow SJ, Swanson SA, Peterson CB, Crosby RD, Wonderlich SA, Mitchell JE. Latent class analysis of eating disorders: Relationship to mortality. *Journal of Abnormal Psychology*. 2012; 121:225–231. [PubMed: 21707126]
46. Dechartres A, Huas C, Godart N, Pousset M, Pham A, Divac SM, et al. Outcomes of empirical eating disorder phenotypes in a clinical female sample: Results from a latent class analysis. *Psychopathology*. 2011; 44:12–20. [PubMed: 20980783]
47. Duncan AE, Bucholz KK, Neuman RJ, Agrawal A, Madden PA, Heath AC. Clustering of eating disorder symptoms in a general population female twin sample: A latent class analysis. *Psychological Medicine*. 2007; 37:1097–1107. [PubMed: 17472759]
48. Duncan AE, Neuman RJ, Kramer J, Kuperman S, Hesselbrock V, Reich T, et al. Are there subgroups of bulimia nervosa based on comorbid psychiatric disorders? *International Journal of Eating Disorders*. 2005; 37:19–25. [PubMed: 15690461]
49. Jacobs MJ, Roesch S, Wonderlich SA, Crosby RD, Thornton L, Wilfley DE, et al. Anorexia nervosa trios: Behavioral profiles of individuals with anorexia nervosa and their parents. *Psychological Medicine*. 2009; 39:451–461. [PubMed: 18578898]

50. Keel, PK.; Holm-Denoma, J.; Crosby, RD.; Haedt, AA.; Gravener, JA.; Joiner, TE. Latent structure of bulimia syndromes: An empirical approach utilizing latent profile analyses and taxometric analyses. In: Striegel-Moore, RH.; Walsh, BT.; Wonderlich, SA.; Mitchell, JE., editors. *Developing an Evidence-Based Classification of Eating Disorders: Scientific Findings for DSM-5*. Arlington, VA: American Psychiatric Association; 2011. p. 145-166.
51. Krug I, Root T, Bulik C, Granero R, Penelo E, Jimenez-Murcia S, et al. Redefining phenotypes in eating disorders based on personality: A latent profile analysis. *Psychiatry Research*. 2011; 188:439–445. [PubMed: 21664698]
52. Myers TC, Wonderlich SA, Crosby RD, Mitchell JE, Steffen KJ, Smyth J, et al. Is multi-impulsive bulimia a distinct type of bulimia nervosa: Psychopathology and EMA findings. *International Journal of Eating Disorders*. 2006; 39:655–661. [PubMed: 16927382]
53. Olmsted, MP.; Wonderlich, SA.; McFarlane, T.; Crosby, RD. Empirical taxonomy of patients with eating disorders. In: Striegel-Moore, RH.; Walsh, BT.; Wonderlich, SA.; Mitchell, JE., editors. *Developing an Evidence-Based Classification of Eating Disorders: Scientific Findings for DSM-5*. Arlington, VA: American Psychiatric Association; 2011. p. 121-132.
54. O'Toole, JK.; DeSocio, JE.; Munoz, DJ.; Crosby, RD. Eating disorders in children and adolescents. In: Striegel-Moore, RH.; Walsh, BT.; Wonderlich, SA.; Mitchell, JE., editors. *Developing an Evidence-Based Classification of Eating Disorders: Scientific Findings for DSM-5*. Arlington, VA: American Psychiatric Association; 2011. p. 203-220.
55. Peterson CB, Crow SJ, Swanson SA, Crosby RD, Wonderlich SA, Mitchell JE, et al. Examining the stability of DSM-IV and empirically derived eating disorder classification: Implications for DSM-5. *Journal of Consulting and Clinical Psychology*. 2011; 79:777–783. [PubMed: 22040286]
56. Richardson J, Steiger H, Schmitz N, Joobar R, Bruce KR, Israel M, et al. Relevance of the 5-HTTLCR polymorphism and childhood abuse to increased psychiatric comorbidity in women with bulimia-spectrum disorders. *Journal of Clinical Psychology*. 2008; 69:981–990.
57. Steiger H, Richardson J, Schmitz N, Israel M, Bruce KR, Gauvin L. Trait-defined eating-disorder subtypes and history of child abuse. *International Journal of Eating Disorders*. 2010; 43:428–432. [PubMed: 19536883]
58. Steiger H, Richardson J, Schmitz N, Joobar R, Israel M, Bruce KR, et al. Association of trait-defined eating disorder sub-phenotypes with (biallelic and triallelic) 5-HTTLCR variations. *Journal of Psychiatric Research*. 2009; 43:1086–1094. [PubMed: 19383563]
59. Striegel-Moore RH, Franko DL, Thompson D, Affenito S, May A, Kraemer HC. Exploring the typology of night eating syndrome. *International Journal of Eating Disorders*. 2008; 41:411–418. [PubMed: 18306340]
60. Striegel-Moore RH, Franko DL, Thompson D, Barton B, Schreiber GB, Daniels SR. An empirical study of the typology of bulimia nervosa and its spectrum variants. *Psychological Medicine*. 2005; 35:1563–1572. [PubMed: 16219114]
61. Sullivan PF, Kessler RC, Kendler KS. Latent Class Analysis of Lifetime Depressive Symptoms in the National Comorbidity Survey. *American Journal of Psychiatry*. 1998; 155:1398–1406. [PubMed: 9766772]
62. Thomas, JJ.; Crosby, RD.; Wonderlich, SA.; Striegel-Moore, RH.; Becker, AE. A latent profile analysis of the typology of bulimic symptoms in an indigenous pacific population. 2011. p. 365-206.
63. Wade TD, Crosby RD, Martin NG. Use of latent profile analysis to identify eating disorder phenotypes in an adult Australian twin cohort. *Archives of General Psychiatry*. 2006; 63:1377–1384. [PubMed: 17146012]
64. Wagner A, Barbarich-Marsteller NC, Frank GK, Bailer UF, Wonderlich SA, Crosby RD, et al. Personality traits after recovery from eating disorders: Do subtypes differ? *International Journal of Eating Disorders*. 2006; 39:276–284. [PubMed: 16528697]
65. Wildes JE, Marcus MD, Crosby RD, Ringham RM, Marin Dapelo M, Gaskill JA, et al. The clinical utility of personality subtypes in patients with anorexia nervosa. *Journal of Consulting and Clinical Psychology*. 2011; 79:665–674. [PubMed: 21767000]

66. Wonderlich SA, Crosby RD, Engel SG, Mitchell JE, Smyth J, Miltenberger R. Personality-based clusters in bulimia nervosa: Differences in clinical variables and ecological momentary assessment. *Journal of Personality Disorders*. 2007; 21:340–357. [PubMed: 17536944]
67. Wonderlich SA, Crosby RD, Joiner T, Peterson CB, Bardone-Cone AM, Klein M, et al. Personality subtyping and bulimia nervosa: Psychopathological and genetic correlates. *Psychological Medicine*. 2005; 35:649–657. [PubMed: 15918341]



**Figure 1.** Comparison of the EDE and EDE-Q class plots. EDE = Eating Disorder Examination; EDE-Q = Eating Disorder Examination-Questionnaire; LC = latent class; OBE = objective bulimic episode; SBE = subjective bulimic episode; OE of Wt/Shp = overevaluation of weight and shape; Fear of Wt Gain = fear of weight gain; BMI = body mass index.

**Table 1**

Type of Assessment Used to Measure the Variables Used as Indicators in Latent Structure Analyses of Eating Disorders

Study	Method of Measurement	
	Interview	Questionnaire
Bulik et al. (2000) [8]	x	
Cain et al. (2010) [43]		x
Crosby et al. (2011) [44]	x	
Crow et al. (2012) [45]		x
Dechartres et al. (2011) [46]		x
Duncan et al. (2007) [47]	x	
Duncan et al. (2005) [48]	x	
Eddy et al. (2009) [39]		x
Eddy et al. (2010) [40]	x	
Jacobs et al. (2009) [49]		x
Keel et al. (2004) [9]	x	
Keel et al. (2011) [50]	x	
Krug et al. (2011) [51]		x
Mitchell et al. (2007) [11]		x
Myers et al. (2006) [52]	x	
Olmsted et al. (2011) [53]	x	
O'Toole et al. (2011) [54]	x	
Peterson et al. (2011) [55]	x	
Pinheiro et al. (2008) [10]		x
Richardson et al. (2008) [56]	x	
Steiger et al. (2010) [57]		x
Steiger et al. (2009) [58]		x
Striegel-Moore et al. (2008) [59]	x	
Striegel-Moore et al. (2005) [60]	x	
Sullivan et al. (1998) [61]	x	
Thomas et al. (2011) [62]		x
Wade et al. (2006) [63]	x	
Wagner et al. (2006) [64]		x
Wildes et al. (2011) [65]		x
Wonderlich et al. (2007) [66]		x
Wonderlich et al. (2005) [67]		x



**Table 2**

Fit Indices for Latent Structure Models with 1- to 6-Class Solutions

Number of Classes	aBIC	BIC	AIC
EDE			
1	3037.06	3065.60	3032.88
2	2752.71	<b>2812.96</b>	2743.90
3	2730.05	2822.01	2716.60
4	<b>2715.26</b>	2838.93	<b>2697.17</b>
5	2726.63	2882.01	2703.90
6	2733.25	2920.33	2705.88
EDE-Q			
1	3013.45	3041.99	3009.28
2	2531.65	<b>2591.90</b>	2522.83
3	2501.18	2593.14	2487.73
4	<b>2480.94</b>	2604.61	<b>2462.85</b>
5	2485.78	2641.15	2463.05
6	2491.04	2678.13	2463.67

*Note:* Minimum value for each fit index is in boldface.

aBIC=adjusted Bayesian Information Criterion; BIC=Bayesian Information Criterion; AIC=Akaike Information Criterion; EDE=Eating Disorder Examination; EDE-Q=Eating Disorder Examination-Questionnaire.

**Table 3**

Probability of Endorsement of Eating Disorder Pathology in Latent Classes 1-4 of the EDE Model and Latent Classes A-D of the EDE-Q Model

	EDE				EDE-Q			
	LC-1 (n = 66)	LC-2 (n = 102)	LC-3 (n = 43)	LC-4 (n = 69)	LC-A (n = 81)	LC-B (n = 98)	LC-C (n = 23)	LC-D (n = 78)
Objective Bulimic Episodes	0.024	0.670	0.530	0.000	0.130	0.570	0.740	0.060
Subjective Bulimic Episodes	0.202	0.508	0.346	0.059	0.101	0.688	0.783	0.036
Purging Behaviors	0.242	0.810	0.671	0.002	0.254	0.775	1.000	0.056
Overevaluation of Shape/Weight	0.964	0.975	0.477	0.241	0.696	0.987	1.000	0.127
Fear of Weight Gain	0.732	0.962	0.238	0.041	0.911	0.990	0.688	0.055
Dietary Rules	0.757	0.729	0.385	0.105	0.821	0.974	0.000	0.107
Preoccupation with Shape/Weight	0.340	0.693	0.039	0.015	0.277	0.867	0.327	0.014
Low Body Mass Index	0.414	0.169	0.099	0.427	0.272	0.222	0.055	0.401

Note: EDE=Eating Disorder Examination; EDE-Q=Eating Disorder Examination-Questionnaire; LC = latent class.

**Table 4**

Number of participants assigned to each latent class pairing

	EDE				TOTAL
	LC-1	LC-2	LC-3	LC-4	
LC-A	<b>39</b>	17	15	10	81
LC-B	18	<b>72</b>	08	00	98
LC-C	02	12	<b>08</b>	01	23
LC-D	07	01	12	<b>58</b>	78
TOTAL	66	102	43	69	280

Note: N's for latent profile pairings that were identified as the best match between the EDE and EDE-Q are presented in boldface.

EDE = Eating Disorder Examination; LC = latent class; EDE-Q = Eating Disorder Examination - Questionnaire.

**Table 5**

Sensitivity and Specificity of the EDE-Q to detect EDE class membership

EDE-Q	EDE											
	LC-1		LC-2		LC-3		LC-4					
	Sn	Sp	Sn	Sp	Sn	Sp	Sn	Sp				
LC-A	<b>59.1%</b>	<b>80.4%</b>	16.7%	64.0%	34.9%	72.2%	14.5%	66.4%				
LC-B	27.3%	62.6%	<b>70.6%</b>	<b>85.4%</b>	18.6%	62.0%	00.0%	53.6%				
LC-C	03.0%	90.2%	11.8%	93.8%	<b>18.6%</b>	<b>93.7%</b>	01.4%	89.6%				
LC-D	10.6%	66.8%	01.0%	56.7%	27.9%	72.2%	<b>84.1%</b>	<b>90.5%</b>				

Note: The best combination of sensitivity and specificity are presented in boldface.

EDE-Q = Eating Disorder Examination - Questionnaire; EDE = Eating Disorder Examination; LC = latent class; Sn = sensitivity; Sp = specificity.