# Risk factors across the eating disorders

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## Abstract

This study sought to examine risk and onset patterns in anorexia nervosa (AN), bulimia nervosa (BN), and binge eating disorder (BED). Women with AN (*n*=71), BN (*n*=66), BED (*n*=160) and non-psychiatric controls (*n*=323) were compared retrospectively on risk factors, symptom onset, and diagnostic migration. Eating disorder groups reported greater risk exposure than non-psychiatric controls. AN and BED differed on premorbid personality/behavioral problems, childhood obesity, and family overeating. Risk factors for BN were shared with AN and BED. Dieting was the most common onset symptom in AN, whereas binge eating was most common in BN and BED. Migration between AN and BED was rare, but more frequent between AN and BN and between BN and BED. AN and BED have distinct risk factors and onset patterns, while BN shares similar risk factors and onset patterns with both AN and BED. Results should inform future classification schemes and prevention programs.

## Keywords

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## 1. Introduction

Anorexia nervosa (AN), bulimia nervosa (BN) and binge eating disorder (BED) constitute the specific eating disorders defined in the Diagnostic and Statistical Manual of Mental Disorders, 5th Edition (DSM-5; American Psychiatric Association [APA], 2013). They are characterized to varying degrees by aberrant eating, dysregulated body weight, and shape and weight overconcern, and are accompanied by significant physical and psychosocial morbidities (Treasure et al., 2010). Over the past several decades, retrospective and prospective, longitudinal studies have enhanced our understanding of risk factors and onset patterns for eating disorders (Killen et al., 1996; McKnight Investigators, 2003; Jacobi et al., 2004; Stice et al., 2008, 2011). However, few studies have compared risk factors or onset patterns across disorders, and to our knowledge no study provides a direct comparison across AN, BN and BED. Thus, the field has inferred similarities and differences across diagnoses on these dimensions by assembling findings from diverse studies; however, such strategies do not provide rigorous empirical comparisons and are vulnerable to error given the diversity of methodologies used. The current study involves a direct comparison of risk factors and onset patterns across AN, BN and BED designed to inform classification approaches and the development of prevention and early intervention programs.

The term "risk factor" refers to a measurable characteristic that precedes the onset of a disorder (Kraemer et al., 1997). Eating disorders have multiple biological and psychosocial risk factors, including genetic and environmental factors (Jacobi et al., 2004). "Variable" risk factors are those that can change spontaneously or with intervention, and are hence most relevant to prevention and early intervention. In our previous work using case control study designs (Fairburn et al., 1997, 1998, 1999; Striegel-Moore et al., 2005; Pike et al., 2008), we have studied "general" variable risk factors that predict the onset of any psychiatric disorder, and "specific" variable risk factors that predict the onset of eating disorders in particular. Stressful life events, including exposure to physical and/or sexual abuse, certain familial experiences (e.g., problematic parenting, parent psychopathology), and negative affectivity did not differ between individuals with eating disorders or other psychiatric disorders, indicating that they are general psychiatric risk factors. In the same way, one can operationalize certain risk factors as common to eating disorders in general, and others as specific to particular eating disorder diagnoses. Shape- and weight-related concerns, dietary restraint, and family history of an eating disturbance are among the most well-established risk factors for partial- and full-syndrome eating disorders (Killen et al., 1996; Jacobi et al., 2004; Stice et al., 2008), but assessing these factors has yielded little prognostic information in terms of predicting the development of specific eating disorder diagnoses.

To our knowledge, no studies to date have directly addressed shared and specific risk factors and onset patterns among individuals with AN, BN, and BED. Nevertheless, individual risk factor studies focusing on specific eating disorder diagnoses have identified several risk factors for each disorder. For example, AN has been associated with childhood feeding problems and premorbid perfectionism (Pike et al., 2008; Nicholls and Viner, 2009). Perfectionism has also been documented in BN (Fairburn et al., 1997), although some evidence suggests few differences between AN and BN and between BN and BED on this characteristic (Bardone-Cone et al., 2007). BN has been additionally associated with a

personal history of childhood obesity and weight-related teasing, as well as a parental history of disordered eating such as dieting and overeating (Fairburn et al., 1997), risk factors that are also pertinent in the development of BED (Fairburn et al., 1998; Striegel-Moore et al., 2005). Personality traits such as novelty-seeking and neuroticism also appear to be relevant in the etiology of BN and BED (Cassin and von Ranson, 2005).

According to individual studies, AN and BED seemed to have few risk factors in common, while several risk factors were shared between AN and BN, and, separately, BN and BED (Fairburn et al., 1997, 1998, 1999; Striegel-Moore et al., 2005; Pike et al., 2008). These data are consistent with the diagnostic migration literature, which shows relatively frequent crossover between AN and BN, and between BN and BED (e.g., Eddy et al., 2008; Stice et al., 2013), but infrequent transition between AN and BED (Fichter and Quadflieg, 2007). These results suggest possible shared pathways between AN and BN, and BN and BED.

Relatedly, the vast majority of individuals with BN reported that dieting attempts preceded binge eating in the onset of their disorder (Bulik et al., 1997; Stice et al., 2008). This onset pattern was associated with a history of AN (Haiman and Devlin, 1999), suggesting that dieting may represent a common developmental path to both disorders. Conversely, a larger subset of individuals with BED endorsed a binge-first onset pattern (e.g., Reas and Grilo, 2007). Individuals with AN tended to report an earlier age of onset than those with BN and BED, whereas the age of onset for BN and BED was quite similar (Stice et al., 2009; Swanson et al., 2011). Taken together, these data suggest that BN may share an etiologic pathway with both AN and BED, but that AN and BED are unlikely to share a developmental path with one another. This could have important implications for targeted prevention and early identification of individuals who are likely to develop a specific eating disorder diagnosis.

Similarities and differences among the eating disorder diagnoses in terms of risk factors and onset patterns could be related to their differing behavioral profiles. For example, while AN and BN were both characterized by stringent dietary restraint, this feature seemed to be much less prominent in BED which was instead characterized by a more chaotic eating pattern (Heaner and Walsh, 2013). Similarly, both BN and BED are characterized by recurrent binge eating, whereas this behavior is prominent only in the binge eating/purging subtype of AN (APA, 2013). Indeed, although DSM-5 retains a categorical classification system in which AN, BN, and BED are represented as distinct diagnoses, some data suggest that distinguishing individuals with eating pathology according to the dimensions of low body weight and binge eating/purging behaviors has distinct clinical utility (e.g., Walsh and Sysko, 2009). It is unclear if risk factors can differentially predict the onset of eating disorder subtypes characterized by varying degrees of dietary restriction, binge eating, and purging.

The aim of this study was to conduct the first direct comparison utilizing interview-based assessments for diagnosis and assessment of both symptom onset and variable risk factors across AN, BN and BED. Based on extant data from multiple independent studies of risk factors for eating disorders, including studies that have employed the same risk factors interview protocol utilized in the current study, we conducted a retrospective case-control

study design and focused on providing direct comparisons of the most salient risk factors that have emerged to date. We hypothesized that women with AN would be more likely to have a family history of AN but less likely to have a family history of overweight and other aspects of weight and eating concerns (i.e., overeating, history of BN) and a personal history of being bullied or teased than women with BN and BED (Fairburn et al., 1997, 1998; Striegel-Moore et al., 2005). We further hypothesized that temperamental factors of perfectionism would be more significant for AN than BED but that BN would not differ from either group (Bulik et al., 2006; Bardone-Cone et al., 2007; Pike et al., 2008; Nicholls and Viner, 2009), whereas temperamental factors associated with impulsivity, as marked by conduct problems, substance abuse, and a history of early pregnancy, would be associated with greater risk for BED and BN than for AN (Cassin and von Ranson, 2005). Negative affectivity, parenting problems (including family discord, high parental demands, separations from parents, and parental absence or death), parental psychopathology, family history of dieting, environmental disruptions and deprivations (e.g., frequent moves), and sexual and physical abuse were hypothesized to be common risk factors across the eating disorders (Fairburn et al., 1997, 1998, 1999; Striegel-Moore et al., 2005; Pike et al., 2008). We also expected that individuals with BN would share similar onset patterns and symptom trajectory with both AN (i.e., dieting history; Bulik et al., 1997; Haiman and Devlin, 1999) and BED (i.e., age of onset; Stice et al., 2009), which would support a model in which developmental pathways are shared between AN and BN, and separately, BN and BED. Finally, based on evidence that the eating disorders may be best characterized by their primary behavioral features (Walsh and Sysko, 2009), we hypothesized that distinct risk factors would be associated with eating disorder subtypes distinguished by dietary restriction (i.e., AN-restricting subtype), binge eating and purging (i.e., AN-binge eating/purging subtype and BN-purging subtype), and binge eating without purging (i.e., BN-non-purging subtype and BED). Because this study utilized a case-control design, our primary aim was to identify risk factors that warrant further investigation using longitudinal research methodologies that are able to more rigorously assess prospective risk.

## 2. Method

### 2.1. Design and recruitment

This study compiles data from several inter-related studies utilizing the same methodologies to study eating disorder risk factors. Included in the current study are women with BN and BED, and case-controls with no psychiatric diagnosis who were recruited under the auspices of the *New England Women's Health Project* (Striegel-Moore et al., 2005); and women with AN who were recruited in the related *Columbia AN Risk Factors* study (Pike et al., 2008). Individuals with BED and BN and non-psychiatric controls were recruited from the community by telephone screening utilizing a consumer information database of 10,000 women and through print media, community referrals, and public service announcements. Individuals with AN were recruited at *Columbia University/The New York State Psychiatric Institute Center for Eating Disorders*. All individuals who contacted the Center between 1998 and 2002 and were screened positive by telephone for current AN were invited to participate in the current study. Half of the AN participants in this study contacted the Center in pursuit of treatment; half the AN sample contacted the Center to inquire about

general information regarding eating disorders or in response to announcements of research studies. All of these individuals were offered the opportunity to pursue treatment upon completion of this study; approximately half opted to do so. The current study reports, for the first time, on participants with BN, and, also for the first time, compares the three eating disorder groups directly to one other and to a control group with no psychiatric diagnosis.

In all cases, after completing a telephone-screening interview, eligible individuals were invited to participate in the study, which included face-to-face diagnostic and risk factor interviews, and several self-report instruments. Weight and height were measured by trained research assistants. The Wesleyan and Columbia University institutional review boards approved this study and all participants provided written informed consent.

## 2.2. Participants

Participants were 297 women with a current DSM-IV (APA, 2000) eating disorder as primary diagnosis (BED=160; BN=66; AN=71) and 323 women with no current psychiatric diagnosis (NC). Exclusion criteria were physical conditions known to influence eating or weight, current pregnancy, or a psychotic disorder. Inclusion criteria for the NC group were absence of past or current clinically significant eating disorder symptoms and absence of a current psychiatric disorder.

Groups differed on current age, body mass index (BMI; $kg/m^2$), and racial distribution ($p<0.001$; see Table 1): As expected, the AN group was younger than the BED and NC groups, and had a lower BMI than all other study groups, while the BMI in the BED group was greater than that in all other study groups. The AN group was less racially diverse than the other study groups. There were no group differences on education ($p>0.05$).

The "index age" was determined in individuals with eating disorders using items from the Eating Disorder Examination (EDE) interview measuring behavioral symptoms of eating disorders (Fairburn and Cooper, 1993). Index age referred to the age at which the participant first experienced a period of sustained dieting (i.e. intake<1200 kcal/day for at least three consecutive months), binge eating (i.e. objective bulimic episodes, on average, at least once a week for at least three consecutive months), or purging (i.e. purging at least once a week for at least three consecutive months). Groups did not differ on index age ($p>0.05$; see Table 1). Assessment of risk factors focused on the period before the index age. For participants who did not develop a sustained pattern of dieting, binge eating, or purging until after age 18, index age was capped at age 18 in order to ensure that all participants were interviewed about a similar developmental timeframe.

## 2.3. Assessment

**2.3.1. Diagnostic assessment**—Current and lifetime psychiatric disorders were assessed using the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID; First et al., 1997), and eating disorder diagnoses and psychopathology were assessed using the diagnostic version of the EDE (Fairburn and Cooper, 1993), a semi-structured investigator-based eating disorder interview. Both interviews have good psychometric properties (Lobbestael et al., 2011; Berg et al., 2012). The EDE was used to ascertain the age of onset

of one's eating disturbance (including the index age in individuals with an eating disorder, using EDE items as described above), as well as the temporal order of dieting versus binge eating in the onset of the eating disturbance.

**2.3.2. Risk factor assessment—**Exposure to putative risk factors was measured using the Oxford Risk Factor Interview, which has good interrater reliability (Fairburn et al., 1997). Assessment of risk factors focused on the period before the index age, thereby ensuring that the risk factor preceded the onset of clinically significant eating disturbances. Exposure to a risk factor was rated on a 5-point scale ranging from 0=*no exposure* to 4=*high severity, long duration,* or *high frequency of exposure*. To reduce the likelihood of false positives, data were recoded into 0=*no definite exposure* (initially coded 0, 1, or 2) versus 1=*definite exposure* (initially coded 3 or 4). Additionally, the Parental Bonding Instrument (Parker et al., 1979), a self-report questionnaire assessing participants' experiences with both parents up to index age, was administered. Measures of parenting problems, including overprotection and low care, were extracted from the PBI. The PBI has good psychometric properties (Ravitz et al., 2010).

Consistent with Fairburn et al. (1997), seven *a priori* risk domains and multiple items within these domains were examined (see Table 2). For six of the *a priori* risk domains, 22 risk factor composite scales were constructed using factor analytic procedures (Fairburn et al., 1997). The seventh risk domain of Childhood Abuse, as described in a separate report (Striegel-Moore et al., 2002), contains two items, Sexual Abuse and Physical Abuse.

## 2.4. Data analysis

Statistical analysis of history and onset data included univariate $F$ tests or $\chi^2$ tests and post-hoc comparisons in case of significance (two-tailed $t$ tests). Analysis of risk factors was based on: a multivariate generalized linear model analysis of risk factor domains by case status and univariate analyses of risk factor domains followed by univariate analyses for individual risk factor items in case of significance. For interpretation of significant $F$ ratios, a series of post-hoc comparisons was conducted (two-tailed $t$ tests) to compare exposure to individual risk factors in the BED, BN, and AN groups versus the NC group and between single eating disorder groups. Partial $\eta^2$ describing the proportion of total variability attributable to a factor was displayed for estimation of effect sizes (partial η2: small 0.01, medium 0.06, large 0.14; Cohen, 1988).

Stepwise discriminant function analysis was used to identify the combination of the most sensitive risk factors in predicting the development of specific diagnoses and behavioral subtypes [restricting subtype (AN-R) versus binge-purge subtype (AN-BP and BN-P) versus binge eating without purging subtype (BN-NP and BED)]. For each function, the significance of the relationship between the diagnostic group and risk factor items was determined with the $\chi^2$ statistic. Effect size was evaluated according to Cohen (1988) using the squared canonical correlation ($R_c^2$: small 0.02, medium 0.15, large 0.35). Significance level for all statistical analyses was set at a two-tailed α<0.05, and for all post-hoc analyses at two-tailed α<0.01.

## 3. Results

### 3.1. Exposure to risk factor domains and risk factors

Table 2 summarizes the results on risk factor domains and individual risk factors by group. Following a significant multivariate test on the risk factor domains [$F(21, 1826)=20.01$, $p<0.001$, $\eta^2=0.19$], univariate analyses on all risk factor domains revealed significant group differences ($p<0.001$). Further, univariate analyses on individual risk factors revealed significant group differences on 1) all individual risk factors from the domains of Subject's Mental Health, Subject's Physical Health, Sexual and Physical Abuse, Other Environmental Experiences, Parental Psychopathology, and Quality of Parenting ($p$ 0.022); and 2) on most risk factors from the domains of Family Weight and Eating Concerns ($p$ 0.003), except for Maternal Overweight, Paternal Overweight, and Parental Absence or Death ($p>0.05$). Large effect sizes were documented for Negative Affectivity, Family Discord, and Family Overeating (partial $\eta^2>0.15$).

In post-hoc tests comparing the exposure to individual risk factors in all eating disorder groups versus the NC group, more BED, BN, and AN individuals reported Negative Affectivity, Perfectionism, Family Dieting, Family Overeating, Maternal Problem Parenting, Family Discord, Paternal Problem Parenting, High Parental Demands, Parental Mood, Parental Substance Disorder, and Physical Abuse ($p<0.01$). Mostly in line with our hypotheses, Conduct Problems, Severe Childhood Obesity, Bullied and Teased, Family History of Bulimia Nervosa, Separations from Parent, and Sexual Abuse were risk factors for both BED and BN ($p<0.01$), but not AN ($p>0.01$), when compared to NC. Contrary to expectations, Substance Abuse, Pregnancy History, Disruptions and Deprivations, Parental Absence or Death increased the risk for a development of BED ($p<0.01$), but not AN or BN ($p>0.01$), when compared to NC. As predicted, Family History of AN increased the risk for a development of AN ($p<0.01$); however, it did not increase risk for BED or BN ($p>0.01$).

Post-hoc tests determining differential exposure to individual risk factors among the eating disorder groups provided some indication of risk factors specific to the development of a certain eating disorder: as hypothesized, Conduct Problems, Substance Abuse, Severe Childhood Obesity, and Family Overeating were endorsed by more BED than by AN individuals, while Perfectionism was endorsed by more AN than BED individuals (all $p<$. 01). As expected, there were no differences in exposure to individual risk factors between the BED versus BN groups and between the BN versus AN groups ($p>0.01$).

### 3.2. Prediction of diagnostic status from risk factors

As expected, the discriminant function analyses to predict the development of BED versus BN identified Perfectionism as a significant predictor (–0.60), indicating greater risk for BN. The effect size was small [$R_c^2=0.03$; $\chi^2(1, n=226)=3.92$; $p=0.048$]. The development of BED versus AN could be predicted with a moderate to large effect size [$R_c^2=0.18$; $\chi^2(5, n=217)=47.09$; $p<0.001$]. As hypothesized, risk factors that loaded on the function were Perfectionism (–0.46), indicating greater risk for AN, and Substance Abuse (0.18), Bullied and Teased, Family Overeating (both 0.17), and Severe Childhood Obesity (0.14), all indicating greater risk for BED. The development of BN versus AN could be predicted

significantly with a small to moderate effect size [$R_c^2$=0.07; $\chi^2$(2, $n$=123)=10.47; $p$=0.005]. Among the risk factors that loaded on the function were Perfectionism (−0.40), indicating greater risk for AN, and Bullied and Teased (0.37), indicating greater risk for BN.

### 3.3. Prediction of behavior subtype from risk factors

In an additional step, discriminant function analysis served to determine whether the discriminative power of risk factors was increased in the prediction of behavioral subtypes (i.e., restricting versus binge eating and purging versus binge eating without purging; Walsh and Sysko, 2009) compared to the prediction of diagnoses. Based on the EDE, 80.3% of BN participants met diagnostic criteria for current purging type BN (BN-P; 53/66), while 19.6% met criteria for the non-purging type BN (BN-NP; 13/66). Among AN participants, 57.7% met criteria for current restricting-type AN (AN- R; 41/71), while 42.3% met criteria for binge eating/purging-type AN (AN-BP; 30/71).

Contrary to expectation, the discriminant function analysis to predict the binge eating without purging subtype (BN-NP and BED) versus the binge-purge subtype (AN-BP and BN-P) was not significant ($p$>0.05). However, as expected, the analysis to predict the development of binge eating without purging subtype (BN-NP and BED) versus the restricting subtype (AN-R) yielded a similar effect size as the analysis to predict the development of BED versus AN [$R_c^2$=0.21; $\chi^2$(5, $n$=204)=50.88; $p$<0.001]. In contrast, the development of the binge-purge subtype (AN-BP and BN-P) versus the restricting subtype (AN-R) could be predicted with an effect size that was twice as large as for the prediction of BN versus AN [$R_c^2$=0.14; $\chi^2$(3, $n$=110)=19.22; $p$<0.001]. The risk factors that loaded on the function included Perfectionism (−0.40), indicating greater risk for the restricting subtype, and Family Overeating (0.26) and Sexual Abuse (0.17), indicating greater risk for the binge-purge subtype.

### 3.4. Onset of current eating disorder and migration across eating disorders

While the eating disorder groups did not differ on age of onset of clinically significant eating pathology ($p$>.05), groups differed significantly on age of onset of the current full-syndrome eating disorder as expected, with a later onset for BED and BN compared to AN [$F$(2, 201)=3.42, $p$<0.037; in years, BED: 20.43±8.34; BN: 20.44±6.37; AN: 17.70±4.09; post-hoc tests $p$>.01]. Regarding history of eating pathology, 11.9% (19/160) of the BED group reported a history of BN, at a mean age of 21.68±6.13 years, which was later than the onset of BED, and 0.6% (1/160) reported a history of AN, at an age of 12 years, thus, preceding the onset of BED. Prior to onset of BN, 27.3% (18/66) reported a history of BED, at an average age of 16.11±7.06 years, and 16.7% (11/66) reported a history of AN, at an average age of 17.64±2.83 years. In the AN group, 2.8% (2/71) showed a history of BED at age 22.50±4.95, thus, after the onset of AN, and 19.4% (14/71) a history of BN at age 17.79±3.29 (which corresponds to the average onset age of AN).

Regarding patterns of symptom onset, more individuals in the BED and BN groups reported binge eating first or binge eating only than individuals in the AN group [BED: 96/159 (60.4%); BN: 43/66 (65.2%); AN: 19/68 (27.9%)], while fewer individuals with BED and BN than with AN revealed dieting first or dieting only [BED: 60/159 (37.3%); BN: 18/66

(27.3%); AN: 43/68 (63.2%); $\chi^2$(4, *n*=293)=20.03, *p*<0.001, post-hoc tests *p*<.01]. A small number of individuals within all eating disorder groups reported onset of binge eating and dieting at the same time [BED: 3/159 (1.9%); BN: 5/66 (7.6%); AN: 6/68 (8.8%)].

## 4. Discussion

This direct interview-based comparison of risk factors and symptom onset patterns in individuals with AN, BN, and BED confirmed that women with eating disorders are exposed to many risk factors prior to onset of illness compared to women with no current psychopathology. Because this study represents a merger of data sets to permit direct comparisons across eating disorders, this study enabled us to test empirically what appeared to be differences in risk factor patterns across eating disorders. Consistent with our hypotheses, AN and BED were distinct from each other on multiple risk factors, with AN reporting greater exposure to perfectionism and BED reporting greater exposure to conduct problems, substance abuse, severe childhood obesity, and family overeating, although the groups did not differ in terms of family history of dieting or personal history of pregnancy. BN and BED did not differ with respect to perfectionism as anticipated, and indeed, all risk factors for BN were shared by individuals with either AN or BED. Discriminant function analysis supported these findings, showing that risk factors more strongly predicted the development of AN versus BED than either AN versus BN or BN versus BED; similarly, risk factors had the most sensitivity in distinguishing between behavioral subtypes characterized by restricting versus binge eating without purging. Taken together, results suggest that AN and BED are distinct in terms of risk profiles, whereas BN takes an intermediate position. Our data are consistent with data from individual risk factors studies showing that BN shares risk factors with both AN and BED, but that the latter two do not share many risk factors with one another (Fairburn et al., 1997, 1998; Cassin and von Ranson, 2005; Striegel-Moore et al., 2005; Pike et al., 2008; Nicholls and Viner, 2009; Walsh and Sysko, 2009). The current findings substantially add to this literature by directly comparing risk factors across all three eating disorder diagnoses.

Overall, many of our results were consistent with the extant literature on risk factors for eating disorders, but others were not. Of note, contrary to expectation, parental overweight did not differ between eating disorder groups and the psychiatric control group, nor among the specific eating disorder diagnoses, which may reflect that separate genetic profiles confer risk for eating disorder psychopathology versus weight regulation (Jonassaint et al., 2011). This is consistent with our finding that family history of AN and BN, while most significant among participants with similar diagnoses themselves, did not distinguish among the eating disorder diagnoses. Similarly, while risk factors conceptually reflecting temperamental impulsivity (e.g., conduct problems, substance abuse, pregnancy history) tended to be elevated in BED as expected, these factors often did not differentiate BN from AN, which may suggest that there is heterogeneity within the BN and AN populations in terms of this trait.

Generally, categorizing the eating disorder groups according to behavioral subtypes of restriction, binge eating and purging yielded a similar pattern of findings as categorizing the groups according to DSM-IV diagnoses. The one exception was that the prediction of the

development of the binge-purge subtype versus the restricting subtype enhanced explanation of variance compared to the prediction of BN versus AN. Critical risk factors were Perfectionism, indicating greater risk for the restricting subtype as supported by previous studies (Bulik et al., 2006; Pike et al., 2008; Nicholls and Viner, 2009), and Family Overeating and Sexual Abuse indicating greater risk for the binge-purge subtype. This latter finding is novel in that family overeating has been shown to be a risk factor shared by BN and BED (Fairburn et al., 1997, 1998; Striegel-Moore et al., 2005), while sexual abuse is a general risk factor for all psychiatric disorders (Fairburn et al., 1997, 1998, 1999; Striegel-Moore et al., 2005; Pike et al., 2008). Consistent with studies reporting limited clinical utility in distinguishing purging and non-purging subtypes of BN (van Hoeken et al., 2009) as compared to AN (Peat et al., 2009), and in light of the high degree of crossover between the disorders (Eddy et al., 2008; Stice et al., 2013), our findings support the removal of the subtyping scheme for BN but not for AN in DSM-5 (APA, 2013).

As predicted, and consistent with the existing literature, BN was similar to both AN and BED in terms of developmental pathways (i.e., age of onset, trajectory of eating disturbance, patterns of symptom onset; Bulik et al., 1997; Haiman and Devlin, 1999; Stice et al., 2009). As previously reported, BN and BED tended to report a later age of onset than AN (Stice et al., 2009; Swanson et al., 2011), although the onset of clinically significant eating pathology was similar across groups, perhaps indicating that AN has a more aggressive onset reflected in the shorter lag time between the onset of disturbed eating and meeting full criteria for the disorder. As earlier studies have shown (Fichter and Quadflieg, 2007; Eddy et al., 2008; Stice et al., 2013), diagnostic crossover between AN and BN and between BN and BED, was more common than crossover between AN and BED, although notably, only a minority of individuals report crossover from one eating disorder to another. Approximately two-thirds of the women with AN reported that their eating disturbance began with dieting whereas approximately two-thirds of the women with BN and BED reported that their eating disturbance began with binge eating. These findings do not support the commonly held assumption that extreme dieting precipitates binge eating in the majority of BN individuals (Stice et al., 2011), suggesting that the relationship between dieting and binge eating is more complex and varied than often assumed. For example, dieting may have a more important role in the maintenance of BN than its onset for most individuals with the disorder. However, it is also possible that individuals with BN and BED under-reported their dieting attempts because dieting is not cognitively dissonant for them and for these individuals the experience of distress and disorder does not occur until the onset of binge eating. The modest rates of migration from AN to BN and the shared presence of dieting in the onset of both disorders suggest that dieting may not be a specific marker for the development of either disorder but rather a shared risk. Indeed, further exploration of factors associated with the development of AN versus BN is needed. Overall, results suggest that BN is developmentally similar to both AN and BED, while the latter two disorders are quite distinct.

In terms of strengths and limitations, we evaluated a wide range of putative risk factors across a large sample of individuals with eating pathology and compared them to non-psychiatric controls. We employed a careful and established interview protocol using specific definitions of risk exposure and eating-related symptomatology. It is, however,

important to consider that the differing methods of recruiting AN (i.e., from a sample of individuals contacting an eating disorder research and treatment center) versus BN, BED, and control participants (i.e., through a consumer information database and community advertisements) may influence the generalizability of our findings, although it should be noted that only half of the AN sample sought treatment. In half of the cases, individuals with AN contacted the center to gather general information about eating disorders or in response to announcements regarding research studies. In addition, despite assessing a wide range of risk factors, other potentially important factors were not included (e.g., perinatal risk factors), and mechanisms of risk cannot be articulated based on our data collection methods (e.g., exposure to family overweight could have potency as a risk factor on genetic, biological, social and environmental levels). In addition, this study utilized retrospective assessment and therefore is subject to recall biases, especially since participants had current eating disorders which could have affected their reporting of risk exposure. Prospective studies are needed to confirm that the correlates identified in the current study are truly risk factors that precede the onset of eating disorders (Kraemer et al., 1997). It is important to view our data as suggestive of risk factors for eating disorders, and it is our hope that our findings will be used to inform the selection of risk factors to be assessed in future genetic or prospective risk factor studies (Jacobi et al., 2004).

Results highlight some important directions for future prevention and early intervention research. Although existing prevention programs have had moderate success in preventing eating disorders (Shaw et al., 2009), a substantial number of participants nevertheless go on to develop eating disorders. Tailoring prevention programs to diagnosis-specific risk profiles, as identified in this study, could enhance the effects of existing programs by making them more personally relevant given one's individual vulnerability and personality characteristics. Furthermore, an enhanced understanding of risk profiles and onset patterns in the eating disorders could facilitate the early identification of individuals who are already exhibiting disordered eating symptoms and are likely to develop a specific eating disorder diagnosis, thereby helping to match participants with early intervention programs known to be efficacious for their specific pathology.

## Acknowledgments

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## Table 1

Sociodemographic characteristics.

| | BED n=160 | BN n=66 | AN n=71 | NC n=323 | Univariate tests | | | Post-hoc tests (p<.01) |
|---|---|---|---|---|---|---|---|---|
| | M±SD | M±SD | M±SD | M±SD | F | df | p | |
| Index Age, years | 15.79±7.10 | 15.14±6.23 | 15.42±3.64 | 15.33±6.22 | 0.26 | 3, 616 | 0.858 | |
| Current Age, years | 31.03±5.75 | 28.73±6.25 | 26.45±6.50 | 29.34±5.69 | 10.22 | 3, 613 | <0.001 | BED, NC>AN |
| Body Mass Index, kg/m$^2$ | 34.49±9.51 | 26.76±7.79 | 14.87±1.82 | 25.51±6.33 | 114.79 | 3, 556 | <0.001 | BED>BN, NC, AN; NC>AN |
| | n (%) | n (%) | n (%) | n (%) | $\chi^2$ | df | p | |
| Race | | | | | 47.47 | 6 | <0.001 | BED, BN, NC vs. AN |
| Asian | 0 (0) | 0 (0) | 3 (4.7) | 1 (0.3) | | | | |
| Black | 60 (37.5) | 14 (21.2) | 1 (1.6) | 87 (27.2) | | | | |
| White | 100 (62.5) | 52 (78.8) | 60 (93.8) | 232 (72.5) | | | | |
| Education | | | | | 2.81 | 6 | 0.832 | |
| High School or Less | 30 (18.8) | 9 (13.6) | 13 (18.6) | 51 (15.9) | | | | |
| Some College | 79 (49.4) | 33 (50.0) | 31 (44.3) | 147 (45.8) | | | | |
| College Grad or Higher | 51 (31.9) | 24 (36.4) | 26 (37.1) | 123 (38.3) | | | | |

*Notes.* BED=binge eating disorder; BN=bulimia nervosa; AN=anorexia nervosa; NC=non-psychiatric control group.

**Table 2**

Univariate analyses for risk factors.

| Composite Risk Factor Scales[†] (n of variables per scale) | BED M±SD | BN M±SD | AN M±SD | NC M±SD | Univariate tests[‡] F | p | $\eta^2$ | Post-hoc comparisons (p<.01) |
|---|---|---|---|---|---|---|---|---|
| SUBJECT'S MENTAL HEALTH | 0.54±1.44 | 0.66±1.27 | 0.70±1.11 | -0.56±0.66 | 65.53 | <0.001 | 0.24 | BED,BN,AN>NC |
| Conduct Problems (2) | 0.58±2.54 | 0.63±2.49 | -0.10±1.73 | -0.39±0.99 | 13.48 | <0.001 | 0.06 | BED,BN>NC; BED>AN |
| Negative Affectivity (5) | 1.07±3.36 | 1.32±3.49 | 2.01±3.57 | -1.24±1.44 | 53.42 | <0.001 | 0.21 | BED,BN,AN>NC |
| Substance Abuse (2) | 0.43±2.40± | 0.12±1.85 | -0.28±0.79 | -0.18±1.25 | 5.70 | 0.001 | 0.03 | BED>NC,AN |
| Perfectionism (2) | 0.0±1.58 | 0.56±1.84 | 1.19±1.81 | -0.41±1.29 | 26.33 | <0.001 | 0.11 | BED,BN,AN>NC; AN>BED |
| SUBJECT'S PHYSICAL HEALTH | 0.67±2.17 | 0.59±2.26 | -0.25±1.27 | -0.40±1.00 | 20.43 | <0.001 | 0.09 | BED,BN>NC; BED,BN>AN |
| Pregnancy History (3) | 0.48±3.27 | 0.49±3.15 | -0.36±1.81 | -0.26±1.87 | 4.61 | 0.003 | 0.02 | BED>NC |
| Severe Childhood Obesity (3) | 0.87±3.08 | 0.68±3.33 | -0.14±1.86 | -0.54±0.57 | 19.63 | <0.001 | 0.09 | BED,BN>NC; BED>AN |
| OTHER ENVIRONMENTAL EXPERIENCES | 0.49±1.64 | 0.51±1.76 | 0.11±1.45 | -0.37±1.39 | 14.19 | <0.001 | 0.07 | BED,BN>NC |
| Bullied and Teased (3) | 0.44±2.16 | 0.80±2.20 | -0.06±2.13 | -0.37±1.95 | 9.27 | <0.001 | 0.04 | BED,BN>NC |
| Disruptions and Deprivation (3) | 0.53±2.32 | 0.21±2.35 | 0.27±1.79 | -0.36±1.84 | 7.80 | <0.001 | 0.04 | BED>NC |
| FAMILY WEIGHT AND EATING CONCERNS | 0.43±1.36 | 0.42±1.28 | 0.27±1.27 | -0.36±0.85 | 23.65 | <0.001 | 0.10 | BED,BN,AN>NC |
| Family Dieting (4) | 0.91±3.45 | 0.50±3.30 | 0.66±3.44 | -0.70±2.47 | 12.81 | <0.001 | 0.06 | BED,BN,AN>NC |
| Maternal Overweight (2) | 0.17±2.10 | 0.16±1.92 | -0.13±1.71 | -0.09±1.73 | 0.97 | 0.407 | 0.01 | |
| Family History of AN (3) | 0.11±2.64 | 0.43±3.51 | 0.76±3.64 | -0.31±1.72 | 4.62 | 0.003 | 0.02 | AN>NC |
| Paternal Overweight (2) | 0.03±1.85 | 0.30±2.14 | -0.00±1.75 | -0.07±1.72 | 0.80 | 0.496 | 0.00 | |
| Family History of BN (3) | 0.35±2.93 | 0.54±3.65 | 0.34±2.88 | -0.36±1.31 | 5.43 | 0.001 | 0.03 | BED,BN>NC |
| Family Overeating (2) | 1.01±2.38 | 0.58±2.21 | 0.02±1.73 | -0.62±0.95 | 36.95 | <0.001 | 0.15 | BED,BN,AN>NC; BED>AN |
| QUALITY OF PARENTING | 0.69±1.20 | 0.78±1.35 | 0.61±1.33 | -0.63±1.02 | 69.03 | <0.001 | 0.25 | BED,BN,AN>NC |
| Maternal Problem Parenting (3) | 1.01±2.24 | 0.98±2.28 | 0.78±2.16 | -0.83±2.36 | 31.06 | <0.001 | 0.13 | BED,BN,AN>NC |
| Family Discord (4) | 1.03±2.73 | 1.40±2.68 | 1.46±2.98 | -1.11±2.01 | 48.78 | <0.001 | 0.19 | BED,BN,AN>NC |
| Paternal Problem Parenting (3) | 0.81±2.36 | 0.95±2.41 | 0.52±2.26 | -0.68±2.26 | 21.16 | <0.001 | 0.09 | BED,BN,AN>NC |
| Separations from Parent (2) | 0.33±2.17 | 0.38±2.28 | 0.22±2.28 | -0.29±1.46 | 5.61 | 0.002 | 0.03 | BED,BN>NC |

| Composite Risk Factor Scales[†] (n of variables per scale) | BED M±SD | BN M±SD | AN M±SD | NC M±SD | Univariate tests[‡] | | | Post-hoc comparisons (p<.01) |
|---|---|---|---|---|---|---|---|---|
| | | | | | F | p | η² | |
| Parental Absence or Death (2) | 0.25±1.53 | −0.05±1.49 | 0.13±1.34 | −0.14±1.25 | 3.22 | 0.022 | 0.02 | BED>NC |
| High Parental Demands (3) | 0.73±2.26 | 1.05±2.20 | 0.56±2.09 | −0.70±1.50 | 32.30 | <0.001 | 0.14 | BED,BN,AN>NC |
| PARENTAL PSYCHOPATHOLOGY | | | | | | | | |
| Parental Mood and Substance Disorder (3) | 0.75±2.42 | 0.57±2.23 | 0.49±2.29 | −0.58±1.52 | 20.76 | <0.001 | 0.09 | BED,BN,AN>NC |
| SEXUAL AND PHYSICAL ABUSE | 1.02±2.56 | 0.92±2.75 | 0.22±2.31 | −0.73±1.64 | 29.65 | <0.001 | 0.13 | BED,BN,AN>NC; BED>AN |
| Sexual Abuse Before Index Age | 1.03±3.93 | 1.18±4.04 | 0.20±3.01 | −0.78±2.12 | 16.99 | <0.001 | 0.08 | BED,BN>NC |
| Physical Abuse Before Index Age | 1.01±3.00 | 0.66±2.92 | 0.24±2.65 | −0.69±2.20 | 17.78 | <0.001 | 0.08 | BED,BN,AN>NC |

*Notes*. Group means represent the average sum of the standardized scores for the variables included in the factor. They can be interpreted as deviations from the mean. BED=binge eating disorder; BN=bulimia nervosa; AN=anorexia nervosa; NC=non-psychiatric control group.

[†] All variables reflect exposure before the participant's index age.

[‡] Univariate analyses with df=3, 616.