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Family Histories of Anxiety in Overweight Men and Women with Binge Eating Disorder: A Preliminary Investigation

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Abstract

Objective—A preliminary examination of the significance of family histories of anxiety in the expression of binge eating disorder (BED) and associated functioning.

Methods—Participants were 166 overweight patients with BED assessed using diagnostic interviews. Participants were administered a structured psychiatric history interview about their first-degree relatives (parents, siblings, children) ($N=897$) to determine lifetime diagnoses of DSM-IV anxiety disorders and completed a battery of questionnaires assessing current and historical eating and weight variables and associated psychological functioning (depression).

Results—BED patients with a family history of anxiety disorder were significantly more likely than BED patients without a family history of anxiety disorder to have lifetime diagnoses of anxiety disorders and mood disorders but not substance use disorders. A family history of anxiety was not significantly associated with timing or sequencing of age at onset of anxiety disorder, binge eating, dieting, or obesity, or with variability in current levels of binge eating, eating disorder psychopathology, or psychological functioning.

Conclusions—Although replication with direct interview method is needed, our preliminary findings suggest that a family history of anxiety confers greater risk for comorbid anxiety and mood disorders but is largely unrelated to the development of binge eating, dieting, or obesity and unrelated to variability in eating disorder psychopathology or psychological functioning in overweight patients with BED.

Keywords

Family History; Anxiety; Binge Eating Disorder; Overweight

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Introduction

Binge eating disorder (BED) comprises recurrent consumption of an unusually large amount of food accompanied by a sense of loss of control with no regular inappropriate compensatory behaviors. BED affects 2% of men and 3.5% of women in the United States [1] and is associated strongly with obesity and with elevated rates of psychiatric comorbidity, particularly high rates of anxiety disorders [1, 2]. The high rate of comorbidity between BED and anxiety disorders (AD), which ranges roughly 30-60% across studies [1-5] suggests the need to explore the significance of such disorder co-occurrences. Conceptually, developmental models of binge eating broadly propose that binge eating develops in attempt to regulate negative affect such as anxiety [6-8], and research has found that AD onset significantly and strongly predicts BED onset whereas BED onset is less likely to predict AD onset [2].

Family history studies examine the aggregation of disorders and afford an economical and helpful tool for exploring the relationship between anxiety disorders and BED. To date, very few family psychiatric history studies have been performed with BED. Lilenfeld et al [9] found that individuals with BED were more likely to have family histories of anxiety, mood, and eating disorders compared to those without BED. Lilenfeld et al [9] and Yanovski et al [10] both found that individuals with BED were more likely to have a family history of substance use disorders compared to individuals without BED. Conversely, three other family history studies found no associations between BED and psychiatric disorders or obesity in first-degree relatives [11-13]. In family history studies with other eating disorders, Lilenfeld and colleagues [14] reported high rates of generalized anxiety disorder (GAD), obsessive compulsive disorder (OCD), and social phobia in first-degree relatives of individuals with anorexia nervosa (AN) and bulimia nervosa (BN). Similarly, Strober and colleagues [15] found high rates of panic disorder in the first-degree relatives of those with AN, which suggests shared familial transmission between ADs and eating disorders.

Several studies have examined familial factors associated with the development of binge eating and BED. Sachs-Ericsson and colleagues [16] found that maternal internalizing disorders, including anxiety and depressive symptoms, significantly predicted the development of binge eating in a large community sample of adults participating in the National Comorbidity Survey-Replication. Brewerton et al [17] found that a family history of emotion dysregulation and substance use disorder was associated with a younger age at binge eating onset. Similarly, Blomquist et al [18] found that a parental history of substance use disorder in individuals with BED was associated with a younger onset of binge eating and shorter lag time between first binge episode and onset of BED.

Research on the timing and sequencing of age at binge eating onset, diet onset, and BED onset suggests that there may be several pathways for the development of BED and—in contrast to the dietary restraint model of binge eating onset [19]—many individuals with BED report binge eating onset prior to diet onset [20-23]. Binge eating onset prior to diet onset has been found to be significantly associated with a younger age at BED onset, shorter lag time between first binge and BED onset [20, 22, 23], more family stressors, and greater psychopathology, including substance use disorders and number of lifetime Axis I and Axis

II disorders [23]. More recently, a younger, child-age-onset of binge eating was found to be associated with greater risk of developing an eating disorder, substance use disorder, and posttraumatic stress disorder (PTSD) compared to those with an older, adult-age-onset of binge eating [17]. Collectively, these findings highlight the importance of identifying factors associated with different developmental trajectories among individuals with BED.

Given the high comorbidity between AD and BED, the frequency of AD onset prior to the onset of BED, the possible shared familial transmission between ADs and other eating disorders, and the significance of the timing and sequencing of BED onset with comorbid psychopathology, anxiety appears to be an important family history factor to examine further with regard to BED development and expression. The current study is a preliminary examination of the significance of family histories of anxiety disorder in the psychiatric comorbidities (Axis I disorder, anxiety disorders, mood disorders, substance use disorders), timing and sequencing of age at onset of psychiatric disorders, BED, and associated eating, weight, and psychological domains in a sample of overweight men and women with BED. Based on previous research indicating high co-occurrence rates between BED and AD [1,2], an AD onset predicting BED onset [2], as well as higher rates of AD in first-degree relatives of BED [9], we hypothesized that a family history of anxiety disorder would be associated with greater psychiatric comorbidities, an anxiety onset prior to BED onset, a younger age at binge eating and BED onset, greater current eating psychopathology, and worse current psychological functioning.

METHOD

Participants

Participants were 166 overweight (BMI ≥ 25) individuals who met full DSM-IV (Diagnostic and Statistical Manual for Mental Disorders) [24] research diagnostic criteria for BED who responded to media advertisements seeking men and women with concerns about binge eating and weight for research studies at a medical school. Participants were limited to these available individuals with BED who were able to provide family history data on anxiety disorders for at least one first-degree biological family member (parent, sibling, child). Participants were aged 18 to 59 years ($M=45.8$, $SD=9.0$), 76.5% ($n=127$) were female, 80.7% ($n=134$) were Caucasian, 12.0% ($n=20$) were Black/African-American, 4.2% ($n=7$) were Hispanic, and 3.0% ($n=5$) self-described as "Other." Mean BMI was 37.5 ($SD=5.9$) and ranged from 25.1 to 57.7. Educationally, 82.5% ($n=137$) reported at least some college. All participants provided informed voluntary written consent prior to study procedures, which had received full review and approval by the Yale Human Investigation Committee.

Procedures and Assessments

Assessment procedures were performed by trained and monitored doctoral-level research-clinicians. During the first evaluation session, DSM-IV Axis I psychiatric disorders, including the BED diagnosis, were determined using the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I/P) [25]. The SCID-I/P also assessed age at BED onset, age at onset of specific anxiety disorders, as well as other psychiatric disorders. In addition, research-clinicians administered the Eating Disorder Examination interview (EDE) [26] and

a Weight and Eating History interview and participants completed a battery of self-report measures. Participants' height and weight were measured at the initial assessment appointment in a standardized fashion and body mass index (BMI) was calculated from these measurements. During a second evaluation session, research-clinicians administered the family history interview to BED participants; this interview was not blinded with regard to BED status but interviewers did not have access to the participants' SCID-I/P psychiatric disorder findings.

Eating Disorder Examination (EDE) [26]—The EDE is a well-established investigator-based interview method for assessing eating disorder psychopathology [27, 28] with good reliability [29]. Except for diagnostic items, which are rated according to the appropriate duration stipulations, the EDE focuses on the previous 28 days. The EDE assesses the frequency of different forms of overeating, including *objective bulimic episodes (OBEs)*; i.e., binge eating defined as unusually large quantities of food with a subjective sense of loss of control). The EDE comprises four subscales (*restraint, eating concern, weight concern, and shape concern*) and a global total score (average of the four subscales). The items assessing eating disorder features for the subscales are rated on a 7-point forced-choice format (0 to 6), with higher scores reflecting greater severity or frequency.

Family History Interview—This family history method involves interviewing BED participants regarding the psychiatric history of their first-degree relatives based on the structured Family History-Research Diagnostic Criteria interview [30] and is modified to be consistent for DSM-IV diagnostic criteria [24]. Doctoral-level research-clinicians first assess the number, relative type (mother, father, brother, sister, son or daughter), gender, age, and life status (alive, deceased, unknown) of all first-degree relatives of BED patients. Then, research-clinicians describe the diagnostic criteria for each psychiatric disorder, and BED patients are asked if any of their biological relatives exhibited symptoms consistent with the diagnostic criteria. A psychiatric diagnosis is determined to be present, suspected, absent, or unknown for the following DSM-IV anxiety disorders: panic disorder, agoraphobia, social phobia, generalized anxiety disorder (GAD), obsessive-compulsive disorder (OCD), and posttraumatic stress disorder (PTSD). If the BED patient provides examples that clearly fit the disorder description, the disorder is deemed to be “present” for that first-degree relative. If the BED patient provides data that seemed to fit the disorder description but leaves some ambiguity, the disorder is “suspected.” A disorder is determined to be “absent” if the first-degree relative does not meet criteria for the psychiatric disorder. If the BED patient does not know the relative well enough to determine whether or not she or he meets criteria due to estrangement, death, etc., the disorder is determined to be “unknown.”

Regarding the reliability and validity of the family history method, a meta-analysis of the family history interview method revealed kappa values for anxiety disorders ranging from 0.30 to 0.50, indicating low to moderate reliability [31]. Rougemont-Bueckin et al [32] compared the family history method to direct interview method for diagnosing anxiety disorders in first-degree relatives and found that the family history method has high specificity for diagnosing any anxiety disorder (94.8%) including panic disorder, agoraphobia, social phobia, GAD and OCD (ranging 97.4% to 99.5%). This high specificity

indicates that there is a low likelihood of diagnosing false positives in relatives. However, they found that the family history method is much less likely to detect anxiety disorders than the direct interview method with sensitivity ranging from 5.9% to 18.5%. This low sensitivity indicates that there is high likelihood of false negatives or not diagnosing an anxiety disorder in relatives. The lowest sensitivity rates are for OCD (5.9%) and agoraphobia (9.5%). Davidson et al [33] also found low sensitivity rates for PTSD using the family history method compared to direct interview. Given overall low sensitivity for detecting anxiety disorders [32, 33] and findings indicating that lowering the threshold for anxiety disorders helps achieve greater accuracy [32], we determined a family history of anxiety disorder to be present if the BED participants either reported or suspected a first-degree relative meeting diagnostic criteria for at least one anxiety disorder.

Weight and Eating History Interview (WEH)—The WEH is a structured clinical interview that has been used in previous studies [e.g., 3, 18, 22] to assess the timing and sequencing of current and historical obesity- and eating-related variables of interest. *Age at dieting onset* was assessed with the following question: “At what age do you remember first going on a diet?” (dependent variable used in *age at dieting onset* analyses).

Several widely used and established self-report measures were given to assess related variables including: The Questionnaire for Eating and Weight Patterns-Revised (*QEWP-R*) [34], which assesses participants’ age first overweight, age first lost at least 10 pounds by dieting, and age at binge eating onset (dependent variable used in *age at first binge eating onset* analyses). The question assessing age at binge eating onset states: “How old were you when you first had times when you ate large amounts of food and felt that your eating was out of control?” [34]. The *Beck Depression Inventory (BDI)* [35], a 21-item measure, assesses symptoms of depression.

Statistical Analyses

Analyses of variance (ANOVA) with continuous variables and chi-squares with dichotomous variables were employed to compare participants with a family history of anxiety disorder (FAD) versus no family history of anxiety disorder (NFAD) on psychiatric comorbidities, age at onset, binge eating developmental and current clinical variables. Age at mood disorder onset was determined by the youngest age at onset of major depressive disorder and/or dysthymic disorder as determined by the SCID. Age at anxiety disorder onset was determined by the youngest age at onset of any specific anxiety disorder determined by the SCID (panic disorder, agoraphobia, social phobia, generalized anxiety disorder, obsessive-compulsive disorder, and posttraumatic stress disorder). Participants who reported an age at anxiety disorder onset prior to their BED onset were categorized as Anxiety-First whereas those whose BED onset preceded their AD onset were categorized as BED-First. Participants who reported the same age at both anxiety disorder and BED onset ($n=5$, 10.6%) were not included in these specific analyses. Similarly, participants who began binge eating (“age at first binge?”) before dieting (“age at dieting onset”) were categorized as bingers first (Binge-First) and those who began dieting before binge eating were categorized as dieters first (Diet-First). Participants who reported the same age at binge eating onset and dieting onset ($n=19$, 11.5%) were not included in these specific analyses.

The time between age at first binge and age at BED onset was calculated by subtracting age at first binge from age at BED onset. Eta squared was calculated for ANOVAs.

RESULTS

First-Degree Relatives

BED participants served as informants on the psychiatric history of 897 first-degree biological relatives: 166 fathers (18.5%), 166 mothers (18.5%), 207 brothers (23.1%), 185 sisters (20.6%), 76 sons (8.5%), and 97 daughters (10.8%). Each BED participant reported on an average of 5.4 first-degree relatives. Eighty percent ($n=721$) of the first-degree relatives were alive at the time of the family history interview, 16% ($n=145$) were deceased, and the status of 3.5% ($n=31$) was unknown. Fifteen percent (15%) of first-degree relatives ($n=135$) were known and/or suspected to have at least one anxiety disorder. More specifically, 10% of the first-degree relatives were known to have at least one anxiety disorder including the following disorders: panic disorder ($n=36$), agoraphobia ($n=1$), social phobia ($n=1$), GAD ($n=34$), OCD ($n=18$), and PTSD ($n=14$). Six percent (6%) of the first-degree relatives were “suspected” to have an anxiety disorder including the following disorders: panic disorder ($n=8$), agoraphobia ($n=0$), social phobia ($n=2$), GAD ($n=28$), OCD ($n=5$), and PTSD ($n=15$). There were “unknown” or missing data for the following disorders: panic disorder (0.8%; $n=7$), agoraphobia (20%; $n=180$), social phobia (19%; $n=171$), GAD (20%; $n=179$), OCD (0.4%; $n=4$), and PTSD (0.4%; $n=4$). Eighteen BED participants were excluded due to insufficient anxiety history data for their first-degree relatives. We considered a family history of anxiety disorder to be present if the BED participants either reported or suspected at least one of their first-degree relatives to meet diagnostic criteria for at least one anxiety disorder.

BED Participants

Eighty-five participants (51.2%) had at least one first-degree relative with a history of AD. Participants with a family history of anxiety disorder (FAD) did not significantly differ from participants with no family history of anxiety disorder (NFAD) on age (FAD: 47.0 ± 8.4 years; NFAD: 44.7 ± 9.6 years; $F(1,165)=2.857$, $p=0.093$), ethnicity (FAD: 83.5% Caucasian; NFAD: 77.8% Caucasian; $\chi^2(3,166)=2.325$, $p=0.508$), education (FAD: high school or less (13.1%), some college (38.1%), college and/or graduate school (48.8%); NFAD: high school or less (20.0%), some college (27.5%), college and/or graduate school (52.5%); $\chi^2(2,164)=2.694$, $p=0.260$), or current BMI (FAD: 37.6 ± 5.9 ; NFAD: 37.5 ± 6.0 ; $F(1,165)=0.008$, $p=0.929$). Participants with FAD significantly differed from participants with NFAD on gender (FAD: 83.5% female; NFAD: 69.1% female; $\chi^2(1,165)=4.781$, $p=0.029$). Gender was not included as a covariate in order not to decrease power given the small sample size of men.

Family History of Anxiety Disorder Versus No Family History of Anxiety Disorder

Psychiatric Comorbidities—Table 1 presents the results from a series of chi-square analyses comparing participants with a family history of anxiety disorder (FAD) to participants with no family history of anxiety disorder (NFAD) on participants' psychiatric comorbidities. Participants with FAD were significantly more likely to have a lifetime

anxiety disorder and lifetime mood disorder than participants with NFAD. Participants with FAD were not significantly more likely to have an Axis I psychiatric disorder or substance use disorder than participants with NFAD.

Timing and Sequencing of BED and Psychiatric Comorbidities—The majority of the BED participants (69%) reported an earlier age at anxiety disorder onset than BED onset with a significantly younger mean age at anxiety disorder onset ($M=21.3\pm13.9$ years) than at BED onset ($M=26.1\pm13.1$ years; $t(1,46)=3.276$, $p<0.002$). However, participants with a FAD did not differ from those with NFAD on age at AD onset (FAD: $M=22.6\pm13.8$ years vs NFAD: $M=18.9\pm14.1$ years; $F(1,45)=0.711$, $p=0.404$, $\eta^2=0.016$). Nor were participants with a FAD significantly more likely to develop an AD before the development of BED than those with NFAD (FAD: 32.1% BED-First, 67.9% Anxiety-First; NFAD: 28.6% BED-First, 71.4% Anxiety-First; $X^2=0.056$, $p=0.813$, $\phi=0.036$). Furthermore, BED participants with a FAD did not differ from those with NFAD on age at mood disorder onset (FAD: $M=29.8\pm14.4$ years vs NFAD: $M=28.7\pm11.6$ years; $F(1,70)=0.111$, $p=0.740$, $\eta^2=0.002$).

Developmental Variables—Table 2 presents the results from a series of ANOVAs and a chi-square analysis comparing participants with FAD to participants with NFAD on disordered eating developmental variables. Participants with FAD did not differ from participants with NFAD on age at overweight onset, age at dieting onset, age at first binge eating onset, or age at BED onset. Participants with a FAD did not differ from participants with NFAD on time lag between age at first binge eating onset and age at BED onset. Participants with a FAD did not differ on sequencing of binge eating onset and dieting onset from those with NFAD. Approximately one-third of the sample (FAD: 33.3%; NFAD: 37.3%) reported binge eating before dieting. Participants with a FAD were not significantly more likely to binge eat before dieting or diet before binge eating than participants with NFAD.

Current Clinical Variables—Participants with a FAD did not differ from participants with NFAD on mean frequency of weekly binge episodes during the 6 months prior to seeking treatment, global eating psychopathology, or depression scores (see Table 2).

DISCUSSION

This study is a preliminary investigation of the significance of family histories of anxiety disorder in a sample of overweight men and women with BED. Our findings indicate that a participant-reported family history of anxiety disorder (FAD) is associated with elevated rates of lifetime anxiety and mood disorders in patients with BED but is largely unrelated to rates of lifetime substance use disorder or to the timing and sequencing of age at onset of psychiatric disorders, BED, binge eating, dieting, or obesity, and unrelated to variability in current eating disorder psychopathology or depressive symptoms.

Consistent with frequent co-occurrences between anxiety disorders and BED [1-5] as well as between anxiety disorders and obesity [36], approximately one third of our sample had a history of anxiety disorder. As hypothesized, a family history of anxiety disorder was associated with higher rates of comorbid anxiety and mood disorders in BED participants.

These findings are similar to Lilenfeld et al [9] who found that anxiety disorders were more prevalent in the first-degree relatives of women with BED compared to women without BED. However, our findings contrast with Lee et al [13] who found no family anxiety history differences between obese women with BED and obese women without BED. The difference between our findings and Lee et al's [13] might be partially due to methodological differences, in that Lee et al [13] used both the family history method and direct interviews to determine the psychiatric diagnoses of first-degree relatives, whereas Lilenfeld et al [9] only used the family history method—analogous to our method.

Our investigation of the significance of family history of anxiety *within* overweight patients with BED is preliminary in nature and precludes examination of shared and non-shared familial transmission more directly. We did not, like previous studies [9-13] include a non-BED comparison group, because our focus was on exploring the potential significance of family history the development and presentation of BED. We note that the few available studies have reported somewhat mixed findings about the nature and extent of shared transmission between BED (and other eating disorders) with anxiety disorders [9, 14, 15], with, for example, some studies suggesting independent transmission [e.g., 14] and other studies, including a methodologically rigorous twin study, suggesting shared familial transmission [e.g., 37]. Our study, which focused on associations *within* BED found expected associations between family history of anxiety disorders and risk for anxiety disorders in BED but no associations with the timing and sequencing of age at onset of BED, binge eating, dieting, or obesity, or with variability in current eating-disorder psychopathology.

Future research should consider including a non-overweight BED sample, in addition to a non-BED comparison group in order to clarify whether our findings are more a reflection of BED or weight status. Unlike other studies with BED, which sampled no men [9, 13] and unlike other eating disorders, which have lower rates of men than in women, relative to BED, our study is unique with the inclusion of men with BED. However, the inclusion of men could have led to lower sensitivity to anxiety disorders in the family history method [32] and resulted in inaccurately lower rates of men with reported family histories of anxiety disorder. Indeed, in our study, there were fewer men with a family history of anxiety. Although it is unlikely that the lower sensitivity to anxiety disorders in men completely changed the nature of our findings, given the relatively small number of men in the overall sample, this highlights the need to employ direct interviewing of first-degree relatives in future studies. Our findings may also differ from those of other eating disorders due to not employing direct interviews for the assessment of the psychiatric histories of first-degree relatives.

Similar to research with other eating disorders in which anxiety disorder onset frequently preceded eating disorder onset [38-40], a majority of our BED participants reported an earlier age at anxiety disorder onset than BED onset. However, in contrast with our hypothesis, the timing and sequencing of age at anxiety disorder and BED onset did not significantly differ between those with a FAD compared to those with NFAD. Nor was a family history of anxiety disorder significantly associated with current levels of binge eating, eating psychopathology, or depression symptoms. These findings are consistent, in

part, with Arikian and colleagues [41], who found that parental anxiety was not associated with either a good or poor long-term prognosis for individuals with BN. However, our findings that age at BED onset was not associated with a FAD contrast with Brewerton et al's study [17] that found that family histories of emotion dysregulation were associated with child-age binge eating onset [17]. However, our sample was somewhat different from Brewerton et al's for several reasons including the exclusion of men, the lack of a formal eating disorder diagnosis, the possibility of having an eating disorder other than BED, and their relatives' emotional problems not being limited to anxiety disorders. Therefore, although there is research to support an association between a family history of affect dysregulation and the development of eating pathology, it may be less clear with regard to the development of BED.

Developmental models of binge eating broadly propose that binge eating is an attempt to regulate negative affect (e.g., escape model [6]; interpersonal model [7]; and transdiagnostic model [8]). Although our investigation was not designed to test these models, our findings might be viewed generally supportive in that BED participants reported a younger age at AD onset than at BED onset (independent of a family history of anxiety) perhaps suggesting anxiety and difficulties coping with affect might have led to binge eating. However, the lack of significant associations between a family history of anxiety disorders and the timing or development of binge eating, dieting, obesity, as well as the levels of current eating psychopathology, suggests that BED may be genetically and etiologically different from other eating disorders (anorexia nervosa and bulimia nervosa) that have different patterns of family transmission. In the interest of generating further research, it should be noted that obese individuals with BED might share familial transmission with substance use disorders [e.g., 9, 18, 42].

Strengths, Limitations, and Future Directions

Our study builds upon existing family history studies with BED by using a much larger BED participant sample size and the inclusion of men with BED. Other strengths include carefully characterized participants with BED, a large number and variety of first-degree biological relatives, doctoral-level research-clinicians who were trained in the administration of the diagnostic and family history interviews and the use of reliable, validated self-report measures.

Limitations include the use of the family history method to assess for DSM-IV anxiety disorders in first-degree relatives given its low to moderate reliability for the diagnosis of anxiety disorders [31, 32], rather than direct diagnostic interviewing of the relatives. Although direct interviewing is the gold standard for assessing the psychiatric histories of proband relatives, the family history method was selected for convenience, relative inexpensiveness, and for the variety of first-degree relatives that could be assessed [43, 44]. The family history method is unlikely to lead to false positives of anxiety disorders [32], and we attempted to account for the likelihood of false negatives by lowering the threshold for an anxiety disorder diagnosis, including both known and suspected anxiety disorders [32]. In our sample 15% of the first-degree relatives had a known or suspected anxiety disorder. This rate of lifetime anxiety disorders in first-degree relatives of BED patients is greater than the

4% reported by Lee et al [13], but it is less than the 28% reported by Lilienfeld et al [9] and less than the 28.8% of lifetime prevalence of anxiety disorders reported by Kessler et al [45]. Lee et al [13] assessed five DSM-IV anxiety disorder diagnoses and used the family history method as well as direct interviews. Lilienfeld et al assessed seven DSM-IV anxiety disorder diagnoses and used only the family history method. Kessler et al [45] assessed eight DSM-IV anxiety disorder diagnoses as part of the National Comorbidity Survey and used direct interviews. Our study assessed six DSM-IV anxiety disorder diagnoses, which, along with using a less sensitive diagnostic method, may partially explain our lower rates of anxiety disorders in the first-degree relatives of BED patients relative to Kessler et al [45].

To further explore the sensitivity of the family history method in this sample, we examined whether only using “present” anxiety disorders (i.e., excluding “suspected” anxiety disorders in first-degree relatives) to determine FAD would make a difference in our findings. We found that this stricter diagnostic threshold reduced the number of BED participants with FAD from 51% to 35% ($n=58$), and that a family history of anxiety was still significantly associated with higher rates of anxiety disorder in BED participants ($p=0.002$) but no longer significantly associated with higher rates of mood disorder ($p=0.093$). These findings suggest that by lowering the threshold to include “suspected” anxiety disorders in first-degree relatives, we effectively increased the sensitivity of the family history method as intended. However, the difference in findings regarding mood disorder highlights the need for replication with direct interview method to ensure that we have accurately categorized BED individuals with a FAD.

Although we found significant differences in the gender composition of BED participants with versus without a family history of anxiety, we chose to not co-vary by gender in our analyses so as to not limit our power as well as due to the preliminary nature of our investigation. It is possible that the gender composition difference may have led to more false negatives in the NFAD group relative to the FAD group due to women being more likely to diagnose their relatives with an anxiety disorder than men [32]. Therefore, future research should replicate this investigation using direct interview method and with a larger sample of men in order to more definitively examine gender differences in families history of anxiety and the development of BED.

Since two family history studies [32, 46] reported that individuals provided more reliable diagnostic data regarding anxiety disorders for their parents versus for their siblings or children, we explored whether a different pattern of results would emerge when using only parents (versus all first-degree relatives) to determine a family history of anxiety disorder. The number of BED participants who had a parental history of anxiety disorder was 31% ($n=51$), and, unlike with a family history of anxiety disorder, a parental history of anxiety disorder was not significantly associated with elevated rates of anxiety disorders ($p=0.070$) or mood disorders ($p=0.418$) in BED participants. These disparate findings may be due to differences in the sensitivity and specificity of the family history method for parental and sibling reports or they may reflect actual differences in the transmission of anxiety disorders among overweight BED participants. Future research is needed to clarify these differences.

Although multiple informants would have increased the validity and reliability of the anxiety disorder diagnoses [32], we were limited to a single-informant design. In addition, the family history method relies on participants' ability to recall their relatives' internalized cognitions and emotions and, therefore, may be biased and particularly poorly suited to the assessment of internalizing disorders (e.g., anxiety disorders) versus externalizing disorders (e.g., substance use disorder, conduct disorder, etc.) where there are observable overt behaviors. That is, the family history method may provide more accurate data when used to determine a family history of substance use disorder (e.g., [18]) and less accurate when used to determine a family history of anxiety disorders, which may partially explain inconsistencies among previous studies using this method (e.g., [9, 13]). However, the family history method affords less ascertainment bias than direct interviews, in which relatives able to be interviewed may have less psychopathology than those relatives unable or unwilling to take part [43]. Future research concerning family histories of anxiety should include direct interview of first-degree relatives for a more valid and reliable assessment of psychiatric history. When direct interviews are not feasible, future research involving the indirect psychiatric assessment of first-degree relatives might involve the development and employment of a more psychometrically sound interview method, and, at a minimum, multiple informants for the psychiatric assessment of first-degree relatives to improve reliability of the family history method. Future research should also assess for a family history of anxiety using the DSM-5 classification of anxiety disorders (e.g., excluding OCD and PTSD).

Other limitations include retrospective recall of BED developmental variables and cross-sectional study design. Future research might prospectively examine the development of obesity and eating psychopathology in individuals with versus without a family history of anxiety. Our sample consisted of available BED participants and did not include non-BED, non-overweight, non-treatment-seeking comparison groups, which precludes certain potentially helpful comparisons as well as generalizability to individuals with BED who are not overweight or obese. Studies should include these non-BED, non-overweight, and non-treatment-seeking comparison groups. Our study, while likely underpowered for detecting small differences in rates of co-occurring psychiatric disorders, was adequately powered (at .80) to detect differences in approximately medium effect sizes, equivalent to *Cohen's w* of .22 or a two-tailed chi-square of 3.84 or larger, using G*Power [47] in a post-hoc analysis. Cohen's *w* is similar to a phi coefficient, and indicates a small effect size at .10 and a medium effect size at .30 [48]. This provides important context for the interpretation of our overall findings—that a family history of anxiety appears to be associated with higher rates of anxiety and mood disorders but not with substance use disorders in BED patients or with BED-related developmental or clinical variables. Nonetheless, collectively—when considered along with the other methodological limitations of the study—we emphasize the preliminary nature of our findings.

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Table 1

Chi-square analyses comparing BED participants with versus without a family history of anxiety disorder on Axis I lifetime psychiatric comorbidities (N=162^a)

BED Participants' Disorders	Family Anxiety (N = 83)	No Family Anxiety (N = 79)	X^2	p	ϕ
	n (%)	n (%)			
Axis I psychiatric disorder	61 (73.5)	47 (59.5)	3.570	0.059	-0.148
Anxiety disorders	32 (38.6)	16 (20.3)	6.502	0.011	-0.200
Panic disorder	21 (25.3)	6 (7.6)			
Agoraphobia	0 (0.0)	0 (0.0)			
Social phobia	9 (10.8)	7 (8.9)			
Generalized anxiety disorder	4 (4.8)	3 (3.8)			
Obsessive-compulsive disorder	4 (4.8)	0 (0.0)			
Posttraumatic stress disorder	7 (8.4)	2 (2.5)			
Mood disorders	46 (55.4)	29 (36.7)	5.701	0.017	-0.188
Major Depressive Disorder	44 (53.0)	26 (32.9)			
Dysthymia	3 (3.6)	3 (3.8)			
Substance use disorders	18 (21.7)	12 (15.2)	1.132	0.287	-0.084

Note. BED = binge eating disorder.

^aFour participants did not complete the SCID.

Table 2

Analyses of variance (ANOVAs) comparing BED participants with versus without a family history of anxiety disorder on eating psychopathology developmental variables and current clinical variables.

	Family Anxiety		No Family Anxiety				
	<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>	F	<i>p</i>	η^2
Age at overweight onset	76	16.7 (9.5)	76	14.9 (8.4)	1.455	0.230	0.010
Age at dieting onset	85	19.9 (9.8)	81	18.9 (8.1)	0.580	0.448	0.004
Age at first binge eating onset	64	21.5 (11.8)	64	23.2 (11.1)	0.668	0.415	0.005
Age at BED onset	80	27.4 (14.1)	79	24.8 (12.0)	1.524	0.219	0.010
First Binge to BED Onset Lag ^a	62	5.3 (10.0)	62	2.9 (10.4)	1.749	0.189	0.014
OBEs (weekly mean/past 6 months)	84	3.5 (0.8)	81	3.6 (0.8)	0.447	0.504	0.003
EDE Global Score	84	2.7 (0.8)	81	2.7 (1.0)	0.061	0.805	0.000
Beck Depression Inventory	85	16.5 (7.9)	81	15.3 (8.1)	0.980	0.324	0.006

	<i>n (%)</i>		<i>n (%)</i>	<i>X</i> ²	<i>p</i>	ϕ	
Binge-First vs Diet-First	78	Binge-first: 26 (33.3)	67	Binge-first: 25 (37.3)	0.250	0.617	−0.042
		Diet-first: 52 (66.7)		Diet-first: 42 (62.7)			

Note. BED = Binge Eating Disorder. OBE = objective bulimic episode; EDE = Eating Disorder Examination.

^aTime (in years) from first binge episode to BED onset, assessed by SCID.