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Parental Substance Use History of Overweight Men and Women with Binge Eating Disorder Is Associated with Distinct Developmental Trajectories and Comorbid Mood Disorder

Kerstin K. Blomquist¹, Robin M. Masheb¹, Marney A. White¹, and Carlos M. Grilo^{1,2}

¹ Department of Psychiatry, Yale University School of Medicine, New Haven, Connecticut

² Department of Psychology, Yale University, New Haven, Connecticut

Abstract

Objective—To examine the significance of parental histories of SUD in the expression of binge eating disorder (BED) and associated functioning.

Method—Participants were 127 overweight patients with BED assessed using diagnostic interviews. Participants were administered a structured psychiatric history interview about their parents ($N=250$) and completed a battery of questionnaires assessing current and historical eating and weight variables and associated psychological functioning (depression and self-esteem).

Results—BED patients with a parental history of SUD were significantly more likely to start binge eating before dieting, had a significantly earlier age at BED onset, and reported less time between binge eating onset and meeting diagnostic criteria for BED than patients without a parental history of SUD. In terms of psychiatric comorbidity, BED patients with a parental history of SUD were significantly more likely to meet criteria for a mood disorder. A parental history of SUD was not significantly associated with variability in current levels of binge eating, eating disorder psychopathology, or psychological functioning.

Discussion—Our findings suggest that a parental history of SUD is associated with certain distinct trajectories in the development of binge-eating (earlier binge onset predating dieting onset) and with elevated rates of comorbidity with mood disorders in patients with BED.

INTRODUCTION

Binge eating disorder (BED) is characterized by recurrent binge eating (i.e., consumption of unusually large amounts of food in discrete periods of time while experiencing a subjective sense of loss of control over the eating) without extreme compensatory weight-control behaviors. BED, which affects approximately 2.0% of men and 3.5% of women, is strongly associated with obesity and with elevated rates of psychiatric comorbidity [1,2,3]. Understanding the etiology and development of BED is critical for targeting at risk individuals and designing effective prevention and treatment interventions. Currently the etiology and development of BED remain largely unexplored and unclear.

Correspondence: Kerstin K. Blomquist, Ph.D., Yale University School of Medicine, P.O. Box 208098, New Haven, CT. 06520. Fax: 203-785-7855, Phone: 203-737-5537, Kerstin.Blomquist@Yale.Edu.

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Research has found that, unlike bulimia nervosa, binge eating does not typically begin following dietary restraint [4,5,6]; however, there appear to be different clinically significant pathways in the development of BED specifically related to the timing of binge eating onset and the sequencing of binge eating versus dieting onset [7]. Studies have found that individuals whose binge eating preceded dieting reported a younger age of binge eating onset [4,5,8], were more likely to meet full BED criteria [4], reported a younger age when first met BED criteria [5], and reported a younger age at overweight onset [5] than individuals whose dieting preceded binge eating. Spurrell and colleagues [8] found that individuals whose binge eating preceded dieting were also more likely to have psychiatric comorbidities, in particular a history of substance use disorders, and were more likely to be diagnosed with a personality disorder than individuals whose dieting preceded binge eating. Most recently, Reas and Grilo [7] reported that weight problems preceded binge eating and dieting behaviors in a majority of 284 treatment-seeking men and women; consistent with the previous literature, many reported binge eating prior to dieting behaviors.

Family studies of bulimia nervosa and anorexia nervosa have revealed family factors associated with the development of these disorders. Although numerous studies have explored familial transmission of psychopathology in individuals with bulimia nervosa (BN) and anorexia nervosa (AN) [e.g., 9,10,11,12,13,14], very few family history studies have been performed with BED. In a blinded family history study of overweight probands with and without BED, Hudson and colleagues [15] found that BED aggregated significantly in families independently of obesity. The three small psychiatric family history studies for individuals with BED have produced varied findings. Lee and colleagues [16] did not find elevated rates of psychiatric disorders among relatives of obese women with BED compared to those without BED. In contrast, Yanovski and colleagues [17] found higher rates of substance use disorders (SUD) in relatives of obese men and women with BED. Lilenfeld and colleagues [18] found higher rates of mood, anxiety, eating and substance use disorders in relatives of women with BED compared to those without BED. These psychiatric disorders seem to follow transmission patterns separate from BED, with the notable exception of SUD, which—unlike in BN—may share a common mechanism [18].

Thus, two studies found that a familial history of SUD may have some significance for the nature of the development of BED [17,18]. Emerging research provides preliminary support for the hypotheses that certain hyperpalatable foods may share certain features with addictive substances [19] and that the reinforcement value from food and alcohol consumption may share some common neurobiological pathways [e.g., 20,21]. Collectively, these findings suggest the importance of examining further the potential significance of familial SUD in BED [18]. The present study explored the role of parental histories of SUD in the development of binge eating and psychiatric comorbidities. We compared overweight men and women with BED who did and did not have a parental history of SUD on developmental variables, including timing of binge onset and sequencing of binge eating versus dieting onset, as well as lifetime history of psychiatric comorbidities. We hypothesized that a parental history of SUD would be associated with earlier binge eating onset and greater psychopathology.

METHOD

Participants

Participants were 127 overweight (BMI ≥ 25) individuals who met full 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-based specialty clinic. The current sample includes only those participants who were able to provide complete family history data for at least one biological parent. Participants were

aged 18 to 59 years ($M=45.7$, $SD=9.2$), 71.7% ($n=91$) were female, 78.7% ($n=100$) were Caucasian, 13.4% ($n=17$) were Black/African-American, 4.7% ($n=6$) were Hispanic, and 3.1% ($n=4$) self-described as “Other.” Mean BMI was 37.8 ($SD=5.9$) and ranged from 25.1 to 52.7. Educationally, 81.9% ($n=104$) 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) [22]. The SCID-I/P also assessed *age at onset of BED* and other psychiatric disorders. The BED diagnosis was confirmed with the Eating Disorder Examination interview (EDE) [23]. In addition, research-clinicians administered a Weight and Eating History interview (described below) and participants completed a battery of self-report measures (listed below). Participants’ height and weight were measured at the initial assessment appointment using a medical balance beam scale, and body mass index (BMI) was calculated from these measurements. During a second evaluation session, research-clinicians administered the family history interview; this interview was not blinded with regard to BED status but interviewers did not have access to the SCID-I/P psychiatric disorder findings.

Eating Disorder Examination (EDE) [23]—The EDE is a well-established investigator-based interview method for assessing eating disorder psychopathology [24,25] with established reliability [26]. 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) which corresponds to the DSM-IV definition of binge eating. The EDE comprises four subscales: *restraint*, *eating concern*, *weight concern*, and *shape concern*. The *Restraint* subscale reflects attempts to restrict food intake to influence weight or shape; the *Eating Concern* subscale reflects the degree of concern about eating; and the *Weight Concern* and *Shape Concern* subscales measure the degree of concern about weight and shape, respectively. The items assessing eating disorder features for the four scales are rated on a 7-point forced-choice format (0 to 6), with higher scores reflecting greater severity or frequency.

Family History Interview—The family history interview was based on the Family History- Research Diagnostic Criteria interview [27] modified and updated to be consistent for DSM-IV criteria [e.g., 28]. The lifetime histories of the major psychiatric disorders were assessed in the first-degree biological relatives of the probands. A psychiatric diagnosis was determined to be absent, suspected, or present. A meta-analysis of the family history interview method revealed an average kappa reliability of 0.70 for substance use disorders [29]. Vandeleur and colleagues [30] compared family history method to direct psychiatric interviews and found that inter-informant agreement was fair to good for all SUDs, except for alcohol abuse, and that lowering the diagnostic thresholds for SUDs increased the accuracy of estimates for these disorders using the family history method. Consequently, for the purpose of this study, a parental history of SUD was considered present if the proband either suspected or reported the mother or father meeting criteria for SUD.

Weight and Eating History Interview (WEH)—The WEH is a structured clinical interview, which assesses current and historical obesity-related variables of interest. Age at

binge eating onset was assessed with the following question: “At what age do you remember first binge eating on a regular basis—at least one time per week?” 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 self-report measures were given to assess related variables including: *The Questionnaire for Eating and Weight Patterns-Revised (QEWP-R)* [31], 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 *Beck Depression Inventory (BDI)* [32], a 21-item widely used and well-established inventory [33], which assesses symptoms of depression and negative affect. The *Rosenberg Self-Esteem Scale (RSES)* [34], a 10-item, widely-used, psychometrically well-established measure of global self-esteem.

Statistical Analyses

Analyses of variance (ANOVA) with continuous variables and chi-squares with dichotomous variables were employed to compare participants with versus without parental histories of SUD on weight and disordered eating developmental variables as well as psychiatric comorbidities. Analyses of covariance (ANCOVA) with continuous variables and logistic regressions with dichotomous variables were employed to assess whether findings persisted after controlling for participant history of SUD. Participants who began binge eating before dieting were categorized as bingers first and those who began dieting before binge eating were categorized as dieters first. Participants who reported the same age for binge onset and dieting onset ($n=15$, 11.9%) were not included in those specific analyses. The time between age at binge onset and age at BED onset was calculated by subtracting age at binge onset from age at BED onset.

RESULTS

Probands

BED participants ($N=127$) served as informants on the psychiatric history of their first-degree biological parents ($N=250$): 124 fathers (49.6%) and 126 mothers (50.4%). Fifty participants (39.4%) had at least one parent with a history of SUD. There were 9 participants (7.1%) for whom only their mothers met SUD criteria, 29 BED participants (22.8%) for whom only their fathers met SUD criteria, and 12 BED participants (9.4%) for whom both their mothers and fathers met SUD criteria. Participants with a parental history of SUD did not differ from participants without a parental history of SUD on age (Parental SUD: $M=45.8$ years, $SD=6.7$; No Parental SUD: $M=45.7$ years, $SD=10.6$; $F(1,125)=0.005$, $p=0.944$), gender (Parental SUD: 74.0% female; No Parental SUD: 70.1% female; $\chi^2(1,127)=0.224$, $p=0.636$), ethnicity (Parental SUD: 82.0% Caucasian, No Parental SUD: 76.6% Caucasian; $\chi^2(3,127)=1.448$, $p=0.694$), education (Parental SUD: high school or less (24.0%), some college (34.0%), college and/or graduate school (42.0%); No Parental SUD: high school or less (13.2%), some college (32.9%), college and/or graduate school (53.9%); $\chi^2(2,127)=2.916$, $p=0.233$), or current BMI (Parental SUD: $M=38.5$, $SD=5.9$; No Parental SUD: $M=37.4$, $SD=5.9$; $F(1,126)=0.953$, $p=0.331$). The same result was found after controlling for participant history of SUD in ANCOVA; participants with a parental history of SUD did not differ from participants without a parental history of SUD on current BMI ($F(1,126)=0.762$, $p=0.385$).

Comparisons of Parental History of SUD Versus No SUD

Developmental Variables—Table 1 presents the results from a series of ANOVAs and chi-square analyses comparing participants with a parental history of SUD to participants

without a parental history of SUD on disordered eating developmental variables. Participants with a parental history of SUD did not differ from participants without a parental history of SUD on age at overweight onset, age at dieting onset, or age at first binge eating onset. The same results were found after controlling for participant history of SUD in a series of ANCOVAs. After controlling for participant history of SUD, participants with a parental history of SUD did not differ from participants without a parental history of SUD on age at overweight onset ($F(1,115)=0.014$, $p=0.907$), age at dieting onset ($F(1,126)=0.359$, $p=0.550$), or age at first binge eating onset ($F(1,90)=0.053$, $p=0.818$).

Participants with a parental history of SUD were significantly more likely to binge eat before dieting than participants without a parental history of SUD ($\chi^2(1,111)=5.428$, $p=0.020$). Sixty-two percent of the participants with a parental history of SUD began binge eating prior to dieting. In contrast, 39.1% of the participants without a parental history of SUD began binge eating prior to dieting. The same results were found after controlling for participant history of SUD in a logistic regression (Odds Ratio=2.624, 95% CI=1.182–5.825, $p=0.018$), indicating that participants with a parental history of SUD were more than twice as likely to begin binge eating prior to dieting. There were 15 participants who reported the same age onset for binge eating and dieting, 14.3% ($n=7$) with a parental history of SUD and 10.4% ($n=8$) without a parental history of SUD.

Participants with a parental history of SUD were significantly more likely to have an earlier age at BED onset than those without a parental history of SUD ($F(1,123)=7.156$, $p=0.008$). The mean age at BED onset for participants with a parental history of SUD was 22.4 years ($SD=10.7$), whereas the mean age at BED onset for participants without a parental history of SUD was 28.6 years ($SD=13.9$). That is, on average, participants with a parental history of SUD met criteria for BED 6.2 years earlier than participants without a parental history of SUD. Furthermore, participants with a parental history of SUD reported a shorter time between the age at which they first began binge eating and the age when they met diagnostic criteria for BED compared to participants without a parental history of SUD ($F(1,121)=8.561$, $p=0.004$). On average, participants with a parental history of SUD met diagnostic criteria for BED 0.8 years ($SD=7.1$) after they first began binge eating, whereas participants without a parental history of SUD met diagnostic criteria on average 6.6 years ($SD=10.4$) after they first began binge eating. There was a much steeper progression to a clinical diagnosis of BED in those with a parental history of SUD. The same results were found after controlling for participant history of SUD in two ANCOVAs. Participants with a parental history of SUD were significantly more likely to have an earlier age at BED onset ($F(1,123)=7.097$, $p=0.009$) and to report a shorter time between the age at which they first began binge eating and age at BED onset ($F(1,89)=8.109$, $p=0.005$) than participants without a parental history of SUD. If a Bonferroni correction for multiple comparisons ($p \leq 0.008$) is applied to Table 1 analyses, statistically significant findings would hold as follows. Participants with a parental history of SUD have a significantly earlier onset of BED and a significantly faster progression from the onset of binge eating until the onset of diagnostic BED than participants without a parental history of SUD.

Clinical Variables—Table 2 presents the results from a series of ANOVAs comparing participants with a parental history of SUD to participants without a parental history of SUD on current clinical variables. Participants with a parental history of SUD did not differ from participants without a parental history of SUD on mean frequency of weekly binge episodes during the 6 months prior to seeking treatment, restraint, eating concern, shape concern, weight concern, global EDE, self-esteem or depression scores. The same results were found after controlling for participant SUD history in a series of ANCOVAs.

Psychiatric Comorbidities—Table 3 presents the results from a series of chi-square analyses comparing participants with a parental history of SUD to participants without a parental history of SUD on participants' psychiatric comorbidities. Participants with a parental history of SUD were significantly more likely to have a lifetime diagnosis of a mood disorder ($\chi^2(1,127)=3.83, p=0.050$) than participants without a parental history of SUD. If a Bonferroni correction ($p \leq 0.01$) is applied, this specific finding is no longer significant. There were no other differences between those with versus without a parental history of SUD on psychiatric comorbidities. In a series of logistic regressions controlling for participant history of SUD, the same results persisted for Axis I disorders and anxiety disorders. However, when controlling for participant history of SUD, a parental history of SUD no longer significantly predicted lifetime mood disorders (Odds Ratio=0.523, 95% CI=0.250–1.097, $p=0.086$).

Lifetime Substance Disorder—Participants with a parental history of SUD did not differ from participants without a parental history of SUD on rates of comorbid SUD ($\chi^2(1,127)=1.700, p=0.192$). Additionally, two ANOVAs and a chi-square analysis were employed to test for differences between participants with versus without comorbid SUD. Participants with SUD did not differ from participants without SUD on sequencing of binge versus diet onset ($\chi^2(1,111)=0.514, p=0.473$), age of BED onset ($F(1,122)=0.036, p=0.850$), or time lag between age of binge onset and age of BED onset ($F(1,121)=0.088, p=0.768$). However, participants with SUD were significantly more likely than participants without SUD to have a lifetime diagnosis of mood disorder ($\chi^2(1,127)=6.123, p=0.013$).

Lifetime Mood Disorder—To determine whether the comorbid mood diagnosis was accounting for the differences in sequencing and timing of BED onset, two ANOVAs and a chi-square analysis were performed. Participants with a lifetime mood disorder did not differ from participants without a lifetime mood disorder on sequencing of binge versus dieting first ($\chi^2(1,111)=0.266, p=0.606$), age of BED onset ($F(1,122)=0.217, p=0.643$), or time lag between age at binge eating onset and age at BED onset ($F(1,121)=1.322, p=0.253$).

Current Mood Disorder—Participants with a parental history of SUD did not differ from participants without a parental history of SUD on *current* mood disorder ($\chi^2(1,127)=0.101, p=0.751$). Furthermore, a chi-square analysis and two ANOVAs were employed to test for differences between participants with a current mood disorder and participants without a current mood disorder. Participants with a current mood disorder did not differ from participants without a current mood disorder on sequencing of binge versus diet onset ($\chi^2(1,111)=0.219, p=0.640$), age of BED onset ($F(1,122)=1.732, p=0.191$), or time lag between age of binge onset and age of BED onset ($F(1,121)=1.895, p=0.171$).

DISCUSSION

This study examined the significance of parental histories of SUD in the developmental trajectory of binge eating onset and presence of comorbid psychopathology in a sample of overweight men and women seeking treatment for binge eating disorder and weight loss. Major findings were that participants with a parental history of SUD were more likely to binge eat first before dieting, report a younger age at BED onset, more rapidly progress from binge eating to full diagnostic criteria for BED, and have a lifetime mood disorder than participants without a parental history of SUD.

Developmental Trajectory Differences

Participants with a parental history of SUD reported meeting diagnostic criteria for BED at a mean age of 22.4 years old, on average 6.2 years younger than the mean age of BED onset

(28.6 years) for those without a parental history of SUD. Participants with a parental history of SUD rapidly developed binge eating disorder within a year of first beginning to binge eat whereas those without a parental history of SUD developed BED more slowly—nearly 7 years after first beginning to binge eat. In addition, the majority (61.9%) of individuals with a parental history of SUD reported binge eating first before dieting. However, participants with a parental history of SUD did not differ from those without a parental history of SUD on age at overweight onset, age at first binge eating onset, or age at dieting onset.

Previous research on the timing and sequencing of binge eating onset found that individuals who reported binge eating first also reported a younger age at binge onset [4,5,7,8]. Interestingly, our findings indicate that participants with a parental history of SUD reported a similar binge-first, younger age BED developmental trajectory. These findings might suggest that one factor that may place individuals at risk for a younger and more rapid BED development is some form of a shared vulnerability with SUD. Perhaps a parental history of SUD is a marker for a biological vulnerability to either food or substance addiction, placing an individual at risk for an earlier onset of excessive food consumption and binge eating [19,35]. Alternatively, it is possible that a parental history of SUD may confer different environmental vulnerabilities that increase risk for binge eating, such as more chaotic or critical familial interactions. For example, research has highlighted the potential significance of childhood emotional abuse/neglect [36] and teasing about shape/weight [37] in BED.

Although the current investigation could not directly test for shared versus independent transmission of psychiatric disorders due to the absence of a non-BED comparison group, the distinct developmental trajectory differences between those with versus without a parental history of SUD provide some support for the hypothesis of a shared familial transmission between BED and SUD [18]. Lilenfeld and colleagues [18] found that the presence of SUD in the female relatives of BED participants was associated with the participants having BED, regardless of whether or not the BED participants themselves had a history of SUD. We found that a parental history of SUD was associated with a distinct BED developmental trajectory and that this younger and more rapid BED onset persisted regardless of the individual's personal history of SUD. These findings, however, contrast with those from family studies with bulimia nervosa (BN), which did not find evidence for a shared familial transmission with SUD [10,12]. For example, Kaye and colleagues [10] found that the presence of SUD in the relatives of BN participants was associated with the BN participants having a history of SUD, not with the presence of BN.

Clinical Variables

Furthermore, our findings suggest that although a parental history of SUD may be associated with a younger and more rapid BED development, a parental history of SUD is not associated with greater clinical severity of BED, self-esteem or depression scores. The absence of significant differences on these clinical variables could reflect restricted ranges due to the sample's composition of mainly obese (BMI $M=37.8$, $SD=5.9$) treatment-seeking individuals who met full diagnostic criteria for BED.

Psychiatric Comorbidities

A majority (58%) of participants with a parental history of SUD had a lifetime mood disorder compared to 40% of participants without a parental history of SUD. Spurrell and colleagues' study [8] investigating the timing and sequencing of BED development found that individuals who binged first before dieting and had a younger age at binge eating onset similarly reported significantly more lifetime psychopathology and more family members with psychological problems than individuals who dieted first before binge eating. Although our results indicate that psychiatric comorbidity was limited to lifetime mood disorders and

that this specific finding did not hold after controlling for participant history of SUD, our findings reveal an analogous pattern—individuals with a parental history of SUD were more likely to binge first before dieting, report a younger age at BED onset and were significantly more likely to report a lifetime mood disorder. Our findings suggest that perhaps a shared biological vulnerability with SUD may not only place individuals at risk for a younger and more rapid BED development but also for lifetime mood disorder.

There are several strengths and limitations to our study. Strengths include employing widely used and established diagnostic interviews administered by trained doctoral-level clinicians and a sample comprised of both men and women whereas previous family studies with eating disorders have focused primarily on women. We note, however, that we relied on family history method for determining parental SUD rather than direct interviews of family members, which represents the best-established method. The family history method has demonstrated acceptable convergence with direct interviews, particularly for SUD [29,30]. Additional limitations include a cross-sectional design and that the historical eating and weight variables were retrospectively assessed by self-report. Retrospective recall may be inaccurate and biased and future research should assess these variables in prospective longitudinal studies. In order to explore potential recall or reporting biases, we assessed two of the developmental historical variables at two different times using two methods (a self-report questionnaire (QEWPR) and a structured clinical interview (WEH)) and found that the Pearson bivariate correlations were $r=0.74$, $p\leq 0.0001$ for age at binge onset and $r=0.79$, $p\leq 0.0001$ for age at diet onset. These findings suggest that participants were at least relatively consistent (“reliable”) in their reporting of ages at binge eating onset. Of course, although patients were relatively reliable this does not necessarily indicate accuracy. We note, however, that research with individuals with BED has found that these patients are accurate self-reporters of weight and height [38,39] and that any bias associated with self-reported height and weight was not associated with disordered eating or psychological variables [38]. Nonetheless, prospective studies are needed to assess risk factors and directly examine developmental trajectories of binge onset. We did not employ non-BED, non-obese, or non-treatment-seeking comparison groups in the current investigation. Although it would have been valuable to compare parental histories of SUD for both BED and non-BED individuals, examining developmental trajectory differences between probands with versus without parental histories of SUD does provide specific insight into potential etiological differences in this heterogeneous BED population. Future research should investigate additional biological relatives and not just parental histories. Finally, previous family studies with BED have found contradictory findings [e.g., 16,18]. The current investigation suggests that parental histories of SUD could account for some of the variability in these previous family studies.

With these strengths and weaknesses as context, we offer the following conclusions. A parental history of SUD appears to be associated with a distinct developmental trajectory of BED characterized by a younger and more rapid development of BED not preceded by dieting and accompanied by a greater likelihood of a lifetime mood disorder. The association between parental histories of SUD and timing and sequencing of BED development suggests a shared familial transmission with SUD and a common biological vulnerability between SUD and BED. These findings regarding age and developmental trajectory among BED patients are perhaps noteworthy in light of previous treatment outcome findings that a younger age at binge onset (≤ 16 years of age) was associated with extremely poor prognosis from either group cognitive-behavioral therapy or group interpersonal psychotherapy for BED [40] and predicted relapse six months following dialectical behavior therapy for BED [41]. Another study, however, found that an earlier age of BED did not predict or moderate treatment outcomes [42]. Our findings that individuals with a parental history of SUD were more likely to have this younger and more rapid BED

development suggest the need for focused research on potential genetic and varied environmental influences [43] underlying biopsychosocial aspects of BED and obesity. Future research should also consider the prognostic significance of familial history and developmental trajectories in BED patients which could potentially inform treatment prescription [44].

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Table 1
Developmental variables of BED participants with versus without a parental history of SUD

	Parental SUD (N = 50)		No Parental SUD (N = 77)		F	p
	n	M (SD)	n	M (SD)		
Age at overweight onset	47	16.0 (8.7)	69	15.3 (9.0)	0.157	0.692
Age at dieting onset	50	19.1 (9.3)	77	20.1 (9.0)	0.339	0.562
Age at first binge eating onset	36	22.7 (11.4)	55	22.1 (11.1)	0.063	0.803
Age at BED onset	48	22.4 (10.7)	76	28.6 (13.9)	7.156	0.008***
First Binge to BED Onset Lag ^a	35	0.8 (7.1)	55	6.6 (10.4)	8.561	0.004**
Binge First vs. Diet First	n (%)		n (%)		X ²	p
	42	Binge: 26(61.9) Diet: 16(38.1)	69	Binge: 27 (39.1) Diet: 42 (60.9)		

Note. BED = binge eating disorder; SUD = substance use disorder.

*
p ≤ 0.05,
**
p ≤ 0.01.

^aTime (in years) from first binge episode to BED onset.

Table 2

Current clinical variables of BED participants with versus without a parental history of SUD

	Parental SUD (<i>N</i> = 50)	No Parental SUD (<i>N</i> = 77)		
	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	F	<i>p</i>
OBEs (weekly mean/past 6 months)	3.5 (0.7)	3.5 (0.7)	0.087	0.768
EDE Restraint	1.6 (1.3)	1.7 (1.3)	0.434	0.511
EDE Eating Concern	2.1 (1.4)	1.8 (1.2)	2.290	0.133
EDE Shape Concern	3.5 (1.1)	3.5 (1.1)	0.196	0.659
EDE Weight Concern	3.0 (0.8)	3.0 (0.9)	0.068	0.795
EDE Global Score	2.6 (0.9)	2.5 (0.8)	0.281	0.597
Rosenberg Self-Esteem	28.4 (5.3)	29.6 (6.0)	1.235	0.269
Beck Depression Inventory	15.9 (7.6)	15.0 (8.0)	0.362	0.549

Note. BED = binge eating disorder; SUD = substance use disorder; OBE = objective bulimic episode; EDE = Eating Disorder Examination.

Table 3

Axis I lifetime psychiatric comorbidities of BED participants with versus without a parental history of SUD

Probands' Disorders	Parental SUD (N = 50)	No Parental SUD (N = 77)	χ^2	<i>p</i>
	<i>n</i> (%)	<i>n</i> (%)		
Axis I psychiatric disorder	37 (74.0)	48 (62.3)	1.863	0.172
Mood disorders	29 (58.0)	31 (40.3)	3.828	0.050*
Anxiety disorders	20 (40.0)	28 (36.4)	0.171	0.680
Substance use disorders	14 (28.0)	14 (18.2)	1.700	0.192

Note. BED = binge eating disorder; SUD = substance use disorder.

*
 $p \leq 0.05$.