Parental Disorders, Childhood Abuse, and Binge Eating in a Large Community Sample

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ABSTRACT

Objective: Genetic and nonshared environmental factors are implicated in the etiology of binge eating behaviors (BEB), with genetic risk expressed as parental psychopathology. Traumatic experiences including child abuse predict onset of BEB. It is not clear if each separately contribute to BEB, or whether parental pathology leads to abuse which in turn influences BEB.

Method: Data were obtained from the National Comorbidity Survey-Replication (N = 2960). Through structural equation modeling, we estimated unique and combined effects of mother’s and father’s internalizing (INT) and externalizing (EXT) disorders, and child abuse on BEB.

Results: Parental INT and EXT psychopathology contributed to child abuse and BEB. Abuse predicted BEB and partially mediated associations between parental psychopathology and BEB.

Discussion: Results emphasize the value of models that incorporate nature and nurture to understand risk for psychopathology in offspring, with childhood abuse being one mediator of how parental psychopathology may reflect genetic risk and influence environmental risk. © 2011 by Wiley Periodicals, Inc.

Keywords: binge eating behavior; child abuse; epidemiological

Introduction

Binge eating behaviors (BEB) are defined by the consumption of a large amount of food within a limited period of time and are associated with the subjective experience of losing control.1 Binge eating increases future risk for obesity and is associated with significant psychosocial impairment.2,3 BEB afflict ~4% of U.S. adults, occur across the eating disorder diagnoses (EDs), and are the central criterion for bulimia nervosa (BN) and binge eating disorder (BED).4 Thus, examining the effect of heritable and environmental influences on BEB may lead to a better understanding of the etiology of a wide range of EDs. This article focuses on the role of parental psychopathology and experiences of child abuse on risk for BEB using data from a large, nationally representative epidemiological study.5

Current research on the etiology of EDs supports a substantial influence of genetic factors6 with genes accounting for ~50% of the variance in EDs and disordered eating behaviors,7 including binge eating8 and binge eating disorder.9 For example, one population-based study of 8,045 Norwegian twins reported a heritability estimate of 41% for participants with binge eating without compensatory behaviors, with individual environmental factors accounting for the remaining variance.10 Similarly, liability to BED was moderately heritable with 45% of the variance due to additive genetics, 13% due to common environmental, and 42% due to unique environmental factors.9 Genetic factors that increase risk for EDs in offspring may be expressed as both eating and general psychopathology in parents.11 For example, significantly higher lifetime rates of DSM IV disorders have been shown in female relatives of women with BED when compared with female relatives of control women without BED.12 In another study, the familial coaggregation of EDs with mood disorders was found to be significant and of the same magnitude as the aggregation of mood disorders alone, suggesting that EDs and mood disorders have common familial factors.13 Further, a genetic analysis of the co-occurrence of BN and major depression (MD) in 1,033 female twin pairs found that additive genes
played an important etiological role in both BN and MD. The genetic liabilities of the two disorders were found to be correlated at 0.456. Whereas, environmental factors account for around half of the variation in liability for both BN and MD, these risk factors appear to be unrelated, suggesting that each disorder has its own set of unique environmental factors.

In addition to indicating the presence of genetic factors that increase risk for the development of EDs, parental disorders may contribute to specific environmental factors that further increase risk for developing BEB. In this instance, parents’ genetic make-up influences the environments in which they rear their children, reflecting a gene-environment correlation. While twin studies have largely supported the influence of genetic factors and non-shared environmental factors on the development of EDs in adults, a correlation between genetic and environmental factors would be reflected in estimates of genetic effects within models that treat genetic and environmental factors as independent. Thus, it is unclear whether parental disorders solely increase risk of BEB via genetic transmission of liability for developing psychopathology or whether parental disorders may also have an indirect effect on risk of BEB by influencing specific environmental factors that may further increase risk for developing BEB.

One specific environmental factor that parental disorders may influence is likelihood of child abuse. In this instance, the influence of parental disorders on risk of BEB may be mediated by child abuse. Specifically, parental disorders may increase risk of child abuse which in turn influences BEB. A body of research has accumulated documenting the long-term negative sequelae of child abuse including a well-replicated association between child abuse and EDs. For example, in a population-based sample of 732 women, those who reported child abuse were twice as likely to suffer from an ED or have subclinical ED symptoms compared with women who had not reported child abuse. Further, women who reported both childhood physical and sexual abuse were four times as likely to develop an ED and three times as likely to develop ED symptoms as women who had not reported abuse. Utilizing a case-control design in women with BED, healthy control participants, and psychiatric controls, Fairburn et al. found that those with BED were more likely to report childhood sexual abuse as well as repeated and severe childhood physical abuse when compared with healthy controls. However, no significant difference was found between women with BED and psychiatric controls, suggesting that abuse history increases risk for psychopathology in general, rather than BED in particular. Thus, both parental pathology and child abuse may reflect nonspecific risk factors for psychopathology including BED. However, it remains unclear whether abuse mediates an association between parental psychopathology and BEB, representing a mechanism through which heritable factors influence risk for BEB, or whether both abuse and BEB are correlated due to the influence of parental psychopathology on both.

The aforementioned etiological explanations suggest parental disorders may influence risk of BEB in a variety of ways. Specifically, parental disorders may (1) directly increase the risk of ordered behavior in children via genetic transmission, which in turn influences BEB, or (2) have both direct and indirect effects (via abuse) on offspring’s negative outcomes. Finally, child abuse may emerge outside of the family-of-origin and represent a nonshared risk factor in the development of binge eating. In this instance, abuse may have direct effects on the development of BEB that are independent of the impact of parental psychopathology.

The current study used data from the National Comorbidity Survey-Replication (NCS-R, N = 2960) to evaluate the following models using Structural Equation Modeling (SEM): (1) father’s and mother’s externalizing and internalizing symptoms each contribute to the participant’s BEB, (2) parental psychopathology leads to CA which leads to participant’s BEB—with child abuse either fully or partially mediating the relationship between parental psychopathology and BEB, and (3) both parental psychopathology and child abuse have independent contributions to participant’s BEB.

**Method**

**Sample**

The methods for the current study, including weighting and sampling procedures for the NCS-R have been described in detail elsewhere. Briefly, the study was based on a nationally representative sample of adults 18 years and older. Data were collected from 2001 to 2003. Response rate was 73%.

All respondents (N = 9282) received the Part I psychiatric interview from professional interviewers who were supervised by the Institute for Social Research at the University of Michigan. Part II was administered to 5,692
participants from Part I who met criteria for any lifetime core disorder (including anxiety and mood disorders, impulse control disorders, drug and substance use disorders, and schizophrenia and other psychotic disorders) plus a probability subsample of other respondents. Part II included questions about additional psychiatric disorders, early childhood experiences and information about participant’s parents. A subset of participants in the Part II survey completed a section on EDs (N = 2960). Thus, current analyses are based on this subsample. The sample comprised 46.3% males; the Mean (SD) age was 44.9(17.8), and racial/ethnic composition was 74.1% white, 11.8% African American, 10.1% Hispanic, and 4.0% other. Mean (SD) family income was $59,245 (47,894).

Sampling and Weighting

Data were weighted to adjust for the over-sampling of Part I respondents with a mental disorder, differential probabilities of selection within households, systematic nonresponse, and residual socio-demographic-geographic differences between the sample and the 2000 Census.28 For the SEM analyses, sample weights, stratification, and clustering variables were controlled for in analyses using the clustering option in MPLUS,29 and, in the analysis of descriptive data, these weighting variables were controlled for using SAS.30

Respondent Recruitment and Consent

Participants received a letter in the mail. Respondents were paid $50. Interviewers obtained verbal informed consent. The human subjects committees of Harvard Medical School and University of Michigan approved the study.

Measures

Demographics. A demographic section assessed sex, age, education, family income, race and ethnicity.

Abuse. Four items related to childhood physical and sexual abuse were used to comprise the dichotomous variable “any childhood abuse.” Participants’ past history of child abuse was embedded in the Post Traumatic Stress Disorder (PTSD) section of the survey, which has been shown to have good validity and reliability.31 Additionally, an item pertaining to the frequency of experiences of physical abuse was obtained from a subsequent section on family life. The reliability and validity of these abuse items have been established.26,32 Pilot testing of the survey methods identified methods to increase reliability and self-disclosure,33 and the PTSD question on severe parental physical abuse demonstrated high concurrent validity with the family section question of frequency of abuse (99% of those endorsing the former endorsed the latter).34 The PTSD section included questions about being raped, molested, and being physically abused as well as age when the traumatic event occurred. Using the guidelines established previously in population studies of childhood abuse; childhood abuse was defined by such events occurring before the age of 15.32,33,36 For childhood sexual abuse, we included rape and molestation before the age of 15 from responses to the following questions: (1) “Were you ever raped? Someone had sexual intercourse with you or penetrated your body with a finger or an object, when you did not want to by threatening you or using some degree of force or were you were so young you did not know what was happening,” (No/Yes) and (2) “Were you ever sexually molested? You were sexually assaulted, where someone touched you inappropriately or when you did not want them to? (No/Yes)” Two questions were used to assess physical abuse. Participants were asked (1) “As a child were you ever badly beaten by your parents?” (No/Yes). In a subsequent section, participants were asked (2) “How frequently have your parents pushed, grabbed, shoved, threw something, slapped, or hit you?” Participants responding “Often” or “Sometimes” were coded “Yes” for abuse. We then computed the dichotomous variable “any child abuse” which represented the participant endorsing any of the four abuse items above.

Family History of Psychiatric Symptoms. Latent constructs were developed for (1) father’s internalizing (FIN), (2) mother’s internalizing (MIN), (3) father’s externalizing (FEIN), and (4) mother’s externalizing (MEIN) disorders using the Family History Research Diagnostic Criteria Interview.37 Participants were asked about psychiatric symptoms of each parent. For the SEM analyses a latent construct was developed separately for father’s and mother’s INT symptoms based on anxiety and depression symptom scores. Internal consistency was α = 0.67 for MIN and α = 0.83 for FINT. In the same fashion, we developed latent constructs for mother’s and father’s EXT symptoms including a scale computed for father’s ASPD and father’s alcohol or substance abuse/dependence (α = 0.69) and for mother ASPD and alcohol substance abuse/dependence (α = 0.69).

Participant’s Diagnostic Assessment. In the current sample, each participant was assessed for the presence or absence of DSM-IV disorders38 including EDs.8 The NCS-R diagnoses are based on the World Health Organi-

*A model which included all the internalizing and externalizing symptoms and diagnoses of the participants with paths to each of the latent variables was considered and the model provided a marginal fit of the data (e.g., RMSEA value was >0.06.35 Thus, a model without participant’s INT and EXT was retained. However, it should be noted that specific internalizing and externalizing disorders may have fit the model such that parental disorders and child abuse led to a specific disorder (other than an eating disorder) and that disorder in turn increased the risk of BEB.
zation Composite International Diagnostic Interview (WHO-CIDI), a structured lay-administered diagnostic interview from which DSM-IV diagnoses could be derived. It has good validity and reliability for most of the DSM-IV disorders. Percent agreement between the diagnoses derived from the survey and the psychiatrists’ clinical impression ranged from 79 to 96% with kappa ≥ 0.60. Specificities were all ≥90%. Although the NCS-R section on EDs has not been specifically examined for reliability and validity, findings based on large National and International epidemiological studies conducted with the EDs section have supported the reliability and construct validity of the measure.

Participant’s BEB. Embedded in the ED section of the NCS-R were items related to BEB. Nine items contributed to the endogenous latent variable of BEB. These are similar to the BEB items examined individually in a heritability study of BED and were the items used in epidemiological studies of EDs.

Importantly, all participants were first asked if they binged on large amounts of food on a regular basis. If the participant said “Yes,” the participant was asked the following nine questions about the behavior (No = 0/Yes = 1): (1) During binges, eat much more quickly than usual; (2) During binges, eat until uncomfortably full; (3) During binges, continue to eat even when they do not feel hungry; (4) During binges, eat alone because embar- rassed by quantity; (5) During or after binges, feel guilty/ upset/depressed; (6) Around time of binging, afraid you would gain weight; (7) Felt like self-esteem/confidence depended on weight/body shape; (8) Worry about effect of binge on your health and body; and (9) Often upset that eating out of control during and after binges. Participants who never binged were not asked the specific nine binge items and were coded ‘No = 0’, on each binge item. A scale score for binge eating behavior, ranging from 0 to 9, was derived for the descriptive purposes (α = 0.97), and these items were included in the latent BEB variable in SEM.

Covariates. Family-of-origin variables were identified as covariates to control for their influence on abuse and BEB. Early parental loss or separation (before the age of 15) and reliance on welfare for 6 months or more were included. Additional covariates used in the model included sex, education, and income, because of their potential influences on abuse, BEB, or both.

Data Analysis

Structural equation modeling (SEM) was performed using MPlus version 5.2. Because abuse was coded as dichotomous we used the weighted least squares mean and variance adjusted estimator (WLSMV), which is better suited for handling categorical outcome variables.

The sampling and weighting procedures were accounted for by using the COMPLEX analysis option in MPlus. Descriptive statistics were calculated in SPSS, and significance levels were calculated in MPlus using the COMPLEX analysis option and also in SAS, which also allows for the proper consideration of weighting variables, stratification, and clustering of data. There were no outliers, and no variables demonstrated significant skew or kurtosis. Standard fit criteria were used to evaluate the overall model fit, with nonsignificant X² value, CFI values greater than 0.95, TLI values greater than 0.90, and RMSEA values of less than 0.06 all indicating good fit. It should be noted that in large samples, such as the current study, the X² is often significant regardless of fit and thus one needs to rely on the other fit indicators (e.g., CFI, TLI, RMSEA) to examine model fit.

Results

Descriptive statistics are provided in Table 1. Among participants, 4.6% (n = 137) endorsed at least one or more of the 9 items included in BEB. Symptomatic participants were younger than those who had no symptoms. There were no significant differences in gender, race, years of education, or household income. Childhood physical abuse and childhood sexual abuse were twice as common in participants with binge-eating compared with those who never binged (N = 2960, 47.8% vs. 23.7%; X²(1) = 28.8, p < 0.01 and 21.9% vs.10%; X²(1) = 19.5, p < 0.01, respectively). Reflecting these differences, participants with binge-eating had a higher frequency of any abuse compared with those who never binged (55.1% vs. 28.9%; X²(1) = 42.6, p < 0.01). Although women were not more likely to binge than men, among those who binged, women had a higher number of binge symptoms than men, 6.5 (2.0) in women vs. 4.4 (2.5) in men, F(1,136) = 28.8, p < .01).

Structural Equation Modeling

It is important to note that the small number of individuals with BEB precludes our examination of models by gender. Gender was included as a covariate in the model.

Measurement Model. The measurement model consisted of the five latent variables. All indicators significantly loaded onto their latent variables (p < .01). This model provided a good fit to the data.
### Table 1. Participant characteristics

<table>
<thead>
<tr>
<th>Category</th>
<th>Binge group Mean (SD) or % N = 137</th>
<th>Nonbinge group Mean (SD) or % N = 2823</th>
<th>F or χ² Value²</th>
<th>p-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>40.1 (18)</td>
<td>45.2 (14.7)</td>
<td>χ² = 10.7</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Female Gender</td>
<td>57.7%</td>
<td>42.3%</td>
<td>χ² = 0.9</td>
<td>.380</td>
</tr>
<tr>
<td>Education in years</td>
<td>13.2 (2.3)</td>
<td>13.2 (2.5)</td>
<td>χ² = 0.8</td>
<td>.372</td>
</tr>
<tr>
<td>Income</td>
<td>$63,126 (49149)</td>
<td>$59,057 (47834)</td>
<td>F = 0.94</td>
<td>.332</td>
</tr>
<tr>
<td>Physical abuse</td>
<td>47.8%</td>
<td>23.7%</td>
<td>F = 40.2</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Sexual abuse</td>
<td>21.9%</td>
<td>10%</td>
<td>χ² = 19.5</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Any Abuse</td>
<td>55.1%</td>
<td>28.8%</td>
<td>χ² = 55.1</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Child welfare</td>
<td>18.7%</td>
<td>9.3%</td>
<td>χ² = 12.8</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Early parental loss</td>
<td>25.7%</td>
<td>21.7%</td>
<td>χ² = 2.8</td>
<td>.14</td>
</tr>
<tr>
<td>Mother depression</td>
<td>7 (1.2)</td>
<td>3 (0.9)</td>
<td>F = 24.4</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Mother anxiety</td>
<td>1.1 (1.8)</td>
<td>1 (1.1)</td>
<td>F = 48.3</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Mother substance</td>
<td>.3 (0.7)</td>
<td>.1 (0.6)</td>
<td>F = 8.2</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Mother Anti-Social</td>
<td>.3 (0.8)</td>
<td>.1 (0.5)</td>
<td>F = 28.3</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Father depression</td>
<td>8 (1.5)</td>
<td>3 (0.9)</td>
<td>F = 51.0</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Father anxiety</td>
<td>5 (1.2)</td>
<td>2 (0.7)</td>
<td>F = 25.0</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Father substance</td>
<td>8 (1.2)</td>
<td>3 (0.8)</td>
<td>F = 48.3</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Father Antisocial</td>
<td>7 (1.2)</td>
<td>3 (0.8)</td>
<td>F = 36.3</td>
<td>&lt;.01</td>
</tr>
</tbody>
</table>

² df for F-values: 1,2959.

(χ² = 44.4, df = 14, p < .001, CFI = 0.99, TLI = 0.99, and RMSE = 0.032). Thus, the measurement model suggests that all latent variables contained appropriate indicators.

#### Structural Model

The SEM model (see Fig. 1) provided a good fit to the data (χ² = 141.4, df = 19, p < .01 CFI = 0.99, TLI = 0.995, RMSE = 0.047). Table 2 describes the relationships among the variables in the model. Among the covariates, welfare status as a child (β = 0.065, p < .01), higher current income (β = 0.653, p < .01), and female gender (β = -.087, p = 0.03), were related to BEB.

Two parental disorders predicted any abuse. Specifically, MINT (β = 0.081, p = 0.010) and FEXT (β = 0.245, p < .01) predicted any abuse. In turn, any abuse predicted participant’s BEB (β = 0.152, p < .01) even after controlling for the main effects of parental pathology on BEB, patient characteristics, and family-of-origin variables. Among the parental disorders, FEXT had a significant relationship with participant’s BEB (β = 0.057, p = 0.048) and MINT had a significant relationship with participant’s BEB (β = 0.101, p = 0.011). Other parental disorders were not significantly related to participant’s BEB.

To test whether abuse mediated associations between parental pathology and participant’s BEB, we used the Model Indirect function in MPlus to generate indirect effect indices for the original source variables. In this model, we found that abuse mediated the relationship between parental pathology and participant’s BEB, though the relationships were relatively weak. Specifically, abuse partially mediated the relationship between Father’s EXT and participant’s BEB (β = 0.037, p < .01); abuse also partially mediated the relationship between Mother’s INT and participant’s BEB (β = 0.012, p = 0.043).

#### Discussion

The current study supports significant associations between participant’s BEB and both parental psychopathology and child abuse. Specifically, as illustrated in Figure 1, father’s externalizing (FEXT) and mother’s internalizing (MINT) symptoms demonstrated direct effects on participant’s BEB. We also found parental pathology to predict abuse which, in turn, predicted participant’s BEB. Finally, we found abuse partially mediated associations between FINT and MINT symptoms and participant’s BEB. Our data and theoretical model are consistent with the idea that parental psychopathology can have direct effects on participant’s BEB as well as indirect effects on BEB through abuse. The results support evidence that the environments to which parents expose their children are important for understanding parental transmission of psychopathology to offspring, with abuse being one important environmental mediator.

#### Parental Psychopathology and Abuse

We found parent’s pathology to increase the risk for child abuse. This is consistent with the literature showing that parents with pathology are more likely to resort to physical punishment than nondisordered parents.25 Specifically, we found MINT disorders to have a direct relationship to abuse. For example, De Bellis et al.47 found that abusive mothers had a greater incidence of anxiety disorders
when compared with nonabusing mothers. Moreover, studies have shown that children of depressed mothers are at a greater risk for abuse.48

Second, we found FEXT symptoms to have a direct relationship to child abuse. By definition, several of the EXT disorders include criteria related to abuse or assault. For example, both intermittent explosive disorder and ASPD are defined, in part, by assaults against others.38 Moreover, intermittent explosive disorder and ASPD are highly comorbid with substance use disorders,49 which in turn increases the risk for parental abuse. Parental substance use disorders are associated with a more than twofold increase in children’s risk of exposure to both physical and sexual abuse.50

**Mother’s INT Disorders on BEB.** MINT disorders also had a direct affect on participant’s BEB. Forbush et al.51 found that EDs best fit a model in which EDs were included in the INT syndrome. Consistent with this conceptualization, EDs display significant comorbidity with mood and anxiety disorders.12,13,52,53 The influence of MINT disorders on participant’s BEB also supports a conceptualization in which EDs represent an underlying INT spectrum disorders.

**FEXT Disorders on BEB.** FEXT disorders had a direct affect on participant’s BEB. This is consistent with the findings that there is a high comorbidity between EDs and substance use disorders.54–57 Researchers have suggested that the significant association between bulimic symptoms and substance use disorders leads to the possibility that there is a shared nonspecific risk factor for EXT problems and bulimic pathology.58,59 Our finding of FEXT relationship to BEB is consistent with the possibility that there is a shared risk factor for EXT problems and EDs.

Together, our results suggest that disorders specifically characterized by binge eating may be best

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**TABLE 2. Model results**

<table>
<thead>
<tr>
<th>N = 2960</th>
<th>Estimate</th>
<th>Error</th>
<th>Standard Estimate</th>
<th>Standard Error</th>
<th>p-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>BEB regressed on: Father’s EXT</td>
<td>.127</td>
<td>.064</td>
<td>.057</td>
<td>.057</td>
<td>.048</td>
</tr>
<tr>
<td>Mother’s EXT</td>
<td>−.059</td>
<td>.116</td>
<td>−.017</td>
<td>−.017</td>
<td>.608</td>
</tr>
<tr>
<td>Father’s INT</td>
<td>.058</td>
<td>.055</td>
<td>.037</td>
<td>.037</td>
<td>.288</td>
</tr>
<tr>
<td>Mother’s INT</td>
<td>.185</td>
<td>.073</td>
<td>.101</td>
<td>.101</td>
<td>.011</td>
</tr>
<tr>
<td>BEB regressed on: Any Abuse</td>
<td>.194</td>
<td>.037</td>
<td>.152</td>
<td>.152</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Parental Loss</td>
<td>.006</td>
<td>.076</td>
<td>.002</td>
<td>.005</td>
<td>.939</td>
</tr>
<tr>
<td>Age</td>
<td>−.014</td>
<td>.019</td>
<td>−.027</td>
<td>−.011</td>
<td>.456</td>
</tr>
<tr>
<td>Education</td>
<td>.001</td>
<td>.001</td>
<td>.653</td>
<td>.001</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Income</td>
<td>−.222</td>
<td>.013</td>
<td>−.087</td>
<td>−.174</td>
<td>.031</td>
</tr>
<tr>
<td>Gender</td>
<td>.283</td>
<td>.110</td>
<td>.065</td>
<td>.221</td>
<td>.190</td>
</tr>
<tr>
<td>Welfare Abuse regressed on: Father’s EXT</td>
<td>.425</td>
<td>.074</td>
<td>.245</td>
<td>.245</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Mother’s EXT</td>
<td>.155</td>
<td>.096</td>
<td>.058</td>
<td>.058</td>
<td>.106</td>
</tr>
<tr>
<td>Father’s INT</td>
<td>.078</td>
<td>.043</td>
<td>.063</td>
<td>.063</td>
<td>.071</td>
</tr>
<tr>
<td>Mother’s INT</td>
<td>.116</td>
<td>.045</td>
<td>.081</td>
<td>.081</td>
<td>.010</td>
</tr>
</tbody>
</table>

BEB = Binge eating behavior, EXT = Externalizing, INT = Internalizing.
modeled as combining liability from the internalizing and externalizing spectrum in hierarchical models of mental disorders.60

**Covariates.** We found women, having a higher income and being on welfare as a child to be related to participant’s BEB. It is not surprising that women were related to participant’s BEB as higher rates of EDs in women is well documented.4 Our findings were also consistent with studies that have found higher social economic status (SES) to be associated with BEB.61,62 Within this large, nationally representative sample, this may reflect that binge episodes require access to large quantities of food, which may not be available to those with the lowest SES. A history of welfare support may reflect an early rearing environment in which food was sometimes scarce, which could increase the risk for psychopathology to be expressed as binge eating. Studies have shown that food deprivation prospectively predicts increased risk of binge eating63,64 particularly if access to food is not limited later in life.

There is literature suggesting that adults who were abused and neglected as children compared to nonabused adults experience more stressful life events, react more strongly to stressful events, and have continued victimization.65–67 In individuals with bingeing, elevated perceived stress, and increased incidence of life stressors have been demonstrated to precede the onset of binge eating.68,69 High stress reactivity (often seen in individuals who were abused) affects BEB as well.70 Thus, individuals with BEB may be born with genetic liability to develop psychopathology and the experience of abuse, combined with a childhood in which food was scarce, may increase risk for expressing that genetic liability as binge eating—particularly if access to food is available in adulthood.

The study has several strengths. First, it was based on a large representative population-based sample, as opposed to a clinical sample. Second, we examined the role of parental pathology in the relationship of abuse to participant’s BEB. It is important for abuse studies to determine whether the association between abuse and negative outcomes is simply the result of gene-environment correlations.71 Many studies have not included measures of parental pathology, and this study showed parental disorders may have both direct and indirect effects on participant’s BEB. Finally, we used SEM to specify a well-fitting model. Among the strengths of SEM is the ability to construct latent variables that are not measured directly, but are estimated in the model from several observed variables each of which is thought to ‘tap into’ the latent variable. This allows the structural relations between latent variables to be more accurately estimated.

Despite its strengths, there are some limitations to consider when interpreting results. First, the small number of participants with bingeing behavior did not allow for a comparison of separate SEM models for men and women. Men and women may have different patterns of parental transmission in relation to abuse.26,72 Second, the small number of participant’s with BEB also may have led to an unstable model. However, this is often the methodological limitation in psychiatric epidemiology when one is predicting a low base rate behavior. Third, while significant relationships were identified, some of the significant paths showed a relatively weak association.

Importantly, there are many relevant clinical variables and theoretical issues that were not addressed in the model that might better explain the associations found among the model presented. In this regard, few studies have examined possible mediating and moderating mechanisms, between childhood abuse and negative mental health outcomes.73 For example, while not all children disclose abuse to the nonoffending parent74,75 among those who do, the majority of parents are supportive, however, a substantial number are not.76 In general, disclosure to supportive parents is associated with lower rates of distress in the abused child,77 whereas delayed disclosure of abuse is associated with higher rates of PTSD.78 Thus, self disclosure may be a moderator of the abuse-BEB relationship. Moreover, there are related variables to consider that were not assessed in the current study, specifically the child’s experience of self-disclosure may impact psychiatric distress73 and parental psychopathology may influence parental support related to the abuse.79

Additionally, it is likely that parental pathology and childhood abuse led to other psychiatric disorders, such as PTSD, and these disorders may account in part for the relationship between abuse and BEB. For example, in one study the relationship between trauma and eating disorder symptoms was significantly reduced when posttraumatic stress disorder symptoms were included in the analyses.80 BEB may represent a disordered attempt to regulate other DSM-IV psychiatric symptoms. Thus PTSD may be a mediator of the abuse-BEB relationship. The influence of PTSD as well as other missing unmeasured third variables may have been of clinical and theoretical interest. Such missing variables should be kept in mind when interpreting the results.
Further, the assessment of parental pathology did not include parental EDs and was taken from participant’s report, which may be biased by the presence of psychopathology in the child. In studies that directly assessed EDs in parents, significant associations have been found between parental eating pathology and EDs in their offspring. If we had a measure of parental ED, we might have expected to find a stronger direct link between parental pathology and BEB.

One other limitation is our lack of inclusion of emotional abuse in the study. Emotional abuse has been found to predict psychiatric disorders even when controlling for the other types of abuse. Indeed emotional abuse compared with sexual and physical abuse has been found to be associated with a broader range of eating disordered symptoms. Unfortunately the NCS-R did not include questions on emotional/verbal abuse. This limitation should be kept in mind.

Importantly, the section of the NCS-R on EDs has not been directly evaluated for its validity and reliability. However, findings from previous large scale epidemiological studies which included this ED module have supported the validity of the instrument. In addition, Cronbach alpha of the scale comprising the 9 binge eating items used to form our BEB latent construct was 0.97, supporting the reliability of this assessment.

Theoretical and Clinical Implications

The current study makes unique contributions to the literature. First, the study’s results have implications for the development of intervention programs to lower child abuse behaviors in parents with INT and EXT disorders and other known risk factors for abuse (e.g., family conflict, low SES, parental loss, and parental divorce). Active outreach programs providing parent training programs for high-risk parents may eventually lower the pernicious effects on the individual and society that child abuse incurs. Although preliminary, the current results support the importance of models that include variables reflecting both nature and nurture to understand binge-eating behavior, a behavior that underlies most EDs.

References

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