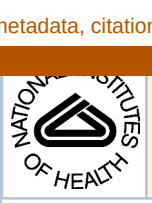



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# Shared and Unique Genetic and Environmental Influences on Binge Eating and Night Eating: A Swedish Twin Study

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## Abstract

We applied twin methodology to female and male twin pairs to further understand the nature of the relation between two behaviors associated with eating disorders—binge eating (BE) and night eating (NE) in an effort to determine the extent of overlap of genetic and environmental factors influencing liability to these behaviors. We calculated heritability estimates for males and females for each behavior and applied bivariate twin modeling to the female data to estimate the genetic and environmental correlation between these two traits. Data on BE and NE were derived from the Swedish Twin Study of Adults: Genes and Environment (STAGE) of the Swedish Twin Registry (STR; $N = 11604$). Prevalence estimates revealed sex differences with females more likely to endorse BE and males more likely to endorse NE. In males, we were only able to estimate univariate heritabilities due to small sample sizes: The heritability for BE was .74 [95% CI = (0.36, 0.93)] and for NE was .44 [95% CI = (0.24, 0.61)]. The best fitting bivariate model for females included additive genetic and unique environmental factors as well as the genetic correlation between BE and NE. Heritability estimates were 0.70 [95% CI = (0.26, 0.77)] for BE and 0.35 [95% CI = (0.17, 0.52)] for NE. The genetic correlation, 0.66 [95% CI = (0.48, 0.96)] suggests considerable overlap in the genetic factors influencing liability to BE and NE. In females, there is considerable overlap in the genetic factors that contribute to these traits, but the incomplete overlap allows for the influence of independent genetic and environmental factors as well. BE and NE in females are therefore best conceptualized as related but not identical traits.

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## Introduction

Binge eating (BE; (Stunkard, 1959) and night eating (NE; (Stunkard, Grace, & Wolff, 1955) are behaviors associated with dysregulated eating. Each represents a core behavioral feature in a proposed DSM eating disorder category [(i.e., binge eating disorder (BED) and night eating syndrome (NES);]. Each also represents a potentially modifiable behavioral feature that can be targeted in interventions for both eating disorders and weight management. We address the extent to which BE and NE represent distinct behavioral phenomena using twin methodology.

BE, first described in 1959 (Stunkard, 1959), is characterized by the consumption of a large amount of food in a short period of time coupled with a feeling of lack of control over ones' eating behavior. The lifetime prevalence of the behavior of BE is estimated to be 4.5% for adults in the United States with slightly higher prevalence among women (Hudson, Hiripi, Pope, & Kessler, 2007). Between 9% (Allison et al., 2006) and 47% (Adami, Meneghelli, & Scopinaro, 1999) of individuals seeking treatment for obesity report BE.

BE is heritable. Several twin studies using varying definitions of the behavior of BE report heritability estimates ranging from 46% to 51% (Bulik, Sullivan, & Kendler, 1998; Reichborn-Kjennerud et al., 2003; Sullivan, Bulik, & Kendler, 1998). Moreover, for the disorder of BED, family studies have reported significantly elevated relative risk (Fowler & Bulik, 1997; Javaras et al., 2008; Lee et al., 1999) and twin studies (Javaras et al., 2008; Reichborn-Kjennerud, Bulik, Tambs, & Harris, 2004) have reported heritability estimates ranging from 41% to 57 % for varying definitions of BED. Thus both the behavior of BE and the clinical syndrome of BED appear to be at least in part influenced by genetic factors.

Turning to NE, the syndrome of NES was originally defined as comprising morning anorexia, evening hyperphagia (i.e. consuming at least 25% of daily calories after the evening meal) and insomnia three or more times a week (Stunkard et al., 1955). Due to a lack of consensus regarding the definition of NES, the proposed diagnostic criteria have undergone several modifications (de Zwaan, Burgard, Schenck, & Mitchell, 2003) including the replacement of insomnia with night time awakenings with ingestions at least three times a week (Birketvedt et al., 1999). These changes are in accordance with the precision of various features in discriminating those with NE problems based on item response analyses (Allison et al., 2008).

The prevalence of NE has been estimated to be approximately 25% in community-based studies (de Zwaan, Roerig, Crosby, Karaz, & Mitchell, 2006; Rand, Macgregor, & Stunkard, 1997; Striegel-Moore, Franko, Thompson, Affenito, & Kraemer, 2006). Relative to BE, less is known about genetic and environmental contributions to the etiology NE. The only study that examined familial aggregation of NES (Lundgren, Allison, & Stunkard, 2006) reported a significantly higher risk (odds ratio = 4.9; $p < .001$) for first degree relatives of affected probands compared with first degree relatives of unaffected probands, suggesting that NES may aggregate in families.

BE and NE have been conceptualized as independent behaviors (Adami et al., 1999; Allison, Grilo, Masheb, & Stunkard, 2005), but have also been posited to be alternate manifestations of the same underlying trait of dysregulated eating (Napolitano, Head, Babyak, & Blumenthal, 2001; Striegel-Moore, Franko, May et al., 2006). Two distinctions support the independence of the behaviors. First, whereas BE is associated with the consumption of a large amount of food, NE is marked by a temporal shift in the consumption of food. Second, BE is associated with loss of control which is not a component of NE as currently conceptualized. Yet commonalities between the behaviors also exist. Both BE and NE are associated with overweight and obesity among clinical and nonclinical samples (Bulik, Sullivan, & Kendler, 2003; de Zwaan et al., 2006; Hudson et al., 2007; O'Reardon, Peshek, & Allison, 2005; Tholin

et al., 2009). For example, Tholin and colleagues (Tholin et al., 2009) report that risk for BE is approximately 3.5 times greater in those with NE compared to those without NE behaviors in the Swedish twins who are the subject of this report. In addition, although the circadian shift in eating patterns is less extreme in BE, individuals with BE do report a preponderance of caloric consumption later in the day (Napolitano et al., 2001) suggesting some circadian effects. Contemporary twin methodology can be applied to address this question and to determine the extent to which the observed patterns of co-occurrence of BE and NE can be accounted for by overlap of genetic and environmental influences.

The Swedish Twin Study of Adults: Genes and Environment (STAGE) of the Swedish Twin Registry (Lichtenstein et al., 2006) included a comprehensive assessment of eating disorders as well as two questions referring to NE behaviors. These data afforded us the opportunity to explore the extent to which genetic and environmental factors influencing BE and NE overlap.

## Method

### Participants

The STAGE sample includes all Swedish twins born 1959–1985 (Lichtenstein et al., 2006), and is part of the Swedish Twin Registry (STR; http://www.mep.ki.se/twinreg/index_en.html) consisting of more than 170,000 individuals in approximately 85,000 twin pairs. “Using web-based questionnaires (or computer assisted telephone interviews for those who preferred this manner of response), data for the STAGE study were collected in 2005 on health measures, physiological/biological measures, sociodemographic and socioeconomic measures, life habits/behaviors, and many of the common complex diseases including information on eating disorders and NE. The total response rate was 59.6% of the eligible 43,000 individuals (43.1% web-based questionnaire; 16.5% telephone interview). For the purpose of this study, we included all individuals from same-sex twin pairs: 11,604 participants ($n_{females}$ = 7,001; 60%; $n_{males}$ = 4,603; 40%). One hundred twins were recontacted to assess test-retest reliability as well as to compare data collection method (computer-based vs. telephone interview). The Kappa value for the eating disorder subsection of the questionnaire was good (.76) suggesting adequate agreement between methods of data collection (Lichtenstein et al., 2006).

The STAGE study was approved by the Regional Ethics Committee at the Karolinska Institutet. This study was also approved by the Biomedical Institutional Review Board at the University of North Carolina at Chapel Hill. All participants provided informed consent, by responding to the questionnaire or verbally over the telephone before participation. A detailed description of study design is described elsewhere (Lichtenstein et al., 2006).

### Zygosity

Determination of zygosity was based on responses to one of the following questions: (1) “During childhood, were you and your twin partner as like as ‘two peas in a pod’ or not more alike than siblings in general?” and (2) “How often did strangers have difficulty in distinguishing between you and your twin partner when you were children?” Twin pairs who responded *alike as two peas in a pod* for Q1 and *almost always* or *often* for Q2 were classified MZ. If both twins responded *not alike* for Q1 and either *seldom*, *almost never*, or *never* for Q2, they were classified DZ. All other twins were classified as ‘not determined.’ We validated this algorithm with a panel of 47 SNPs in a random sample of 198 twin pairs. Ninety five percent (*N* = 188) were correctly classified. Of the misclassified twin pairs 8 were MZ and 2 were DZ. This zygosity algorithm has also previously been validated with similar results (Lichtenstein et al., 2002).

## Measures

**Binge eating—**Broadly defined BE was assessed using an expanded, on-line Structured Clinical Interview for DSM-IV (SCID)-based instrument. A dichotomous variable for lifetime history of BE was created. Men and women with present or past symptoms of BE were identified through the following two questions: "Have you ever had binges when you ate what most people would regard as an unusually large amount of food in a short period of time?" with response alternatives *yes*, *no* and *don't know/refuse*; and "When you were having eating binges, did you feel that your eating was out of control?" with response alternatives *not at all*, *slightly*, *moderately*, *very much*, *extremely*, and *don't know/don't wish to answer*. Those who answered *yes* to the first question and *slightly*, *very much* or *extremely* to the second question were defined as reporting BE.

**Night eating—**The two questions included in the STAGE questionnaire on NE were: (1) "How often do you get up at night to eat?" with response options *never*, *once or twice*, *weekly*, *nightly*, and *don't know/don't wish to answer*, and (2) "What proportion of your daily food intake takes place after the evening meal?" with response options *0%*, *1–24%*, *25–49%*, *50–74%*, *75–100%*, and *don't know/don't wish to answer*. A dichotomous variable was created for NE. Consistent with our previous work (Tholin et al., 2009), participants were classified as experiencing NE if they endorsed having awakenings with food intake during the night at least once a week and/or 25% or more of daily food intake after the evening meal.

## Statistical Analyses

**Rationale—**Univariate and bivariate twin modeling are methodologies used in genetic epidemiology research to parse out genetic and environmental factors influencing liability to a latent phenotype. Because MZ twins share 100% of their genes and DZ twins share on average 50%, when certain assumptions are met (Kendler, 1993), higher concordance among MZ than DZ twins suggests that genetic factors influence liability to expression of the phenotype.

The classic twin study assesses the contribution of (1) additive genetic effects (i.e., heritability, $a^2$); (2) shared (or common) environmental effects ($c^2$); and (3) unique environmental effects ($e^2$) to the liability to a phenotype. The $e^2$ estimate also includes measurement error. The sum of $a^2 + c^2 + e^2 = 1$ (total variance). Additive genetic effects (A) represent the cumulative impact of several genes of small to moderate effect. In general additive genetic effects are indicated when the intrapair correlation for monozygotic twins ($r_{mz}$) is greater than intrapair correlation for dizygotic twins ($r_{dz}$). Common or shared environmental effects (C) result from etiological influences both members of a twin pair are exposed to regardless of zygosity (e.g., childhood SES), contribute equally to $r_{mz}$ and $r_{dz}$, and increase twin similarity. Unique (or individual specific) environmental effects (E) are events occurring to one twin but not the other (e.g., one twin suffers an injury requiring surgery and the other does not) and serve to decrease twin similarity. Determination of the ACE model reveals what proportion of variance in liability to a disorder is due to genetic variance what proportion is due to shared (or common) environment.

**Current study—**The design of our current study was constrained by the low prevalence of BE and NE in males. For females, the prevalence of BE and NE was sufficiently large to enable us to fit a series of bivariate structural equation models using Cholesky's decomposition using Mx (Neale, Boker, Xie, & Maes, 1999) to measure $a^2$, $c^2$, and $e^2$ estimates for each variable (i.e., BE and NE) and to estimate correlations corresponding to the extent of overlap between $a^2$, $c^2$, and $e^2$ for the BE and $a^2$, $c^2$, and $e^2$ for the NE ($r_a$, $r_c$, and $r_e$, respectively). A graphical depiction of the full bivariate twin model for BE and NE is presented in Figure 1. The full model and seven nested models were estimated. Model selection was based on a statistical test for the difference in $\chi^2$ values between the nested model and the full model (Neale & Cardon, 1992) where degrees of freedom (df) is equal to the difference between the df of the nested

model and the full model. A non-significant result suggests that there is no difference, or decrement, in model fit between the two models thus making the parsimonious model (i.e., fewer parameters estimated) the preferred model. Model selection was also based on Akaike's Information Criteria (AIC; (Akaike, 1987), a measure of the goodness of fit when comparing nested models. The lowest AIC value among all models is suggestive of the best fitting model with regard to precision and complexity.

For males, due primarily to the low prevalence of BE and the relatively rare concordance and cross-concordance of BE and NE in male twin pairs, we were unable to obtain stable parameter estimates in more complex bivariate or sex-limitation models. We were therefore constrained to fitting univariate ACE models to the BE and NE data separately.

For all models, the raw ordinal data option was used, which allows both complete and incomplete (one twin missing information) twin pairs to be used. Parameter estimates and 95% confidence intervals were obtained from the models.

## Results

### Sample Characteristics for Twin Models

The final sample for twin modeling included 1964 MZ female pairs, 1388 DZ female pairs, 1290 MZ male pairs and 921 DZ male pairs. There were 171 unpaired MZ females, 126 unpaired DZ females, 104 unpaired MZ males and 77 unpaired DZ males. For the bivariate models, 1808 MZ and 1276 DZ female pairs had complete data. The participants ranged in age from 20 to 47 years. The mean age of the female respondents was 33.5 years (*SD* = 7.7; range 20 – 47) and for males the mean age was 32.9 (*SD* = 7.6; range 20 – 47). Mean lifetime highest and lowest body mass index (BMI) values are presented in Table 1 by sex, comparing those with and without BE as well as those with and without NE.

BE was reported in 5.7% (*n* = 395) of the females and 0.7% (*n* = 32) of males. NE was reported in 3.4% (*n* = 227) of females and 4.3% (*n* = 192) of males. Forty-four women with BE (11.1%) also endorsed NE, and 20% of those with NE also endorsed BE. Eight men endorsed both BE and NE. No significant differences between MZ and DZ twins emerged for the prevalence of either BE (females: $\chi^2 = 0.70$, $p = .41$; males: $\chi^2 = 0.01$, $p = 0.92$) or NE (females: $\chi^2 = 0.16$, $p = 0.69$; males: $\chi^2 = 0.24$, $p = 0.63$).

Tetrachoric correlation coefficients along with 95% confidence intervals (CI) for BE and NE for all zygosity groups are presented in Table 2. Within-trait tetrachoric correlations were strong for BE and moderate for NE for MZ twin pairs for both females and males indicating genetic liability to the risk for *each of the traits*. The within twin cross-trait tetrachoric correlations, a measure of association between BE and NE within individuals, suggest that liability to BE and NE was moderately correlated for females. For males, the within twin cross-trait correlation was weak for MZ twins and strong for DZ twins. We expect the within twin cross-trait correlations to be equal for MZ and DZ twin pairs thus this discrepancy could be due to small sample size and should be interpreted with caution. Overall these findings suggest shared liability to both BE and NE; however, the source of this correlation (i.e., genetic, environmental) cannot be determined from these coefficients. Across twin pairs, correlations for BE and NE were moderate for both female and male MZ pairs compared with DZ pairs providing evidence for genetic effects on the liability to BE and NE.

### Twin Modeling

**Females—**Model fit results for the females are presented in Table 3a and parameter estimates are presented in Table 3b. The full model (i.e., model 1) and seven nested submodels with varying parameters (models 2–8) were examined. Inspection of the $\chi^2$ difference tests suggest

that models 7 and 8 could be rejected as fitting significantly more poorly than the full model. Of the remaining models, model 6 (i.e., AE-AE, $r_a$) in which additive genetic and unique sources of variation were estimated for both behaviors (i.e., C pathways were constrained to zero) represents the best fitting model as indicated by the lowest AIC value and the non-significant $\chi^2$ difference test. In this model, additive genetic factors explained approximately 70% (95% CI = 0.60, 0.77) of the variance in BE and 35% (95% CI = 0.17, 0.52) of the variance for NE. The genetic correlation, $r_a$ = 0.66 (95% CI = 0.48, 0.96), indicates that there is considerable overlap in the genetic factors that contribute to liability to BE and NE.

**Males—**The results for the univariate ACE models for BE and NE are presented in Tables 4 and 5, respectively. While the full ACE models fit the data well, C could be removed from both BE and NE ACE models without degrading the model. The AE model fit best for both BE and NE, with 74% (95% CI = 0.36, 0.93) of the observed variance for BE and 44% (95% CI = 0.24, 0.61) of the observed variance for NE attributable to genetic factors, suggesting substantially higher heritability of BE compared to NE. The proportion of variance attributable to unique environmental effects was 26% (95% CI = 0.07, 0.64) for BE and 56% for NE (95% CI = 0.39, 0.76).

## Discussion

We applied twin methodology to further understand the nature of the relation between two behaviors associated with eating disorders—BE and NE—using data from the STR (Lichtenstein et al., 2006). To the best of our knowledge this is the first univariate twin analysis of NE and the first bivariate twin analysis of BE and NE. Four main findings emerged from our analyses: (1) females were more likely to endorse BE behaviors while males were more likely to endorse NE behaviors; (2) a substantial contribution of additive genetic effects to liability in BE was found for both females and males, but a minimal contribution from common (shared) environmental factors; (3) moderate heritability for NE was found for both males and females; and (4) the genetic correlation between BE and NE in females indicated a substantial, but not complete, overlap in genetic etiological factors.

### Prevalence of Binge Eating and Night Eating

The prevalence of BE in females was slightly higher in our sample (5.8%) compared with U.S. national averages (4.9%), but was lower for males in our sample (0.8%) compared with US averages [(4%) ((Hudson et al., 2007)]. When using a more comparable Scandinavian sample based in Norway, our prevalence for BE in females was slightly higher (5.8% vs. 5.2%) for females and lower for males [(0.8% vs. 3.8%) (Reichborn-Kjennerud et al., 2004)]. Definitional issues could explain this discrepancy, including variations across studies with BE frequency (e.g., once per week vs. twice per week) as well as differences in categorization of BE (Hudson et al., 2007); Reichborn-Kjennerud et al., 2004). The observed lower prevalence for males in our sample may reflect cultural differences in BE or stigma associated with the endorsement of items related to BE such as having a sense of being ‘out of control’ when eating.

The prevalence of NE as defined in our sample (awakenings with food intake during the night at least once a week and/or 25% or more of daily food intake after the evening meal) was higher in males (4.6%) than females [(3.3%) (Tholin et al., 2009)]. Little has been published on the prevalence of specific NE behaviors, thus making cross-sample comparison difficult. However, Striegel-Moore and colleagues report the prevalence of NE behaviors (defined as eating 50% or more of daily calories after 7pm, or any eating after 11pm) to be approximately 25% in a community sample of $N$ = 8250 participants 15 – 39 years of age (Striegel-Moore, Franko, Thompson et al., 2006); however, their definition of NE is quite discrepant from ours which could account for the observed differences in reported prevalence.

It is worth noting that the BMI values observed in the current study may be lower than expected. Sweden has much lower base rates of obesity compared with the U.S. (Ogden et al., 2006, Neovius et al., 2006) which could explain the lower BMI values for this sample compared with a U.S. sample. Moreover, in the study we are focusing on the component behaviors of BE and NE (not the proposed syndromes of BED and NES). It is possible that the behaviors are less strongly associated with BMI than are the complete syndromes.

## Twin Models

**Heritability of binge eating—**Our findings support prior studies suggesting a substantial contribution of genetic factors to the liability of BE (Bulik & Reichborn-Kjennerud, 2003; Bulik et al., 2003; Reichborn-Kjennerud et al., 2004; Sullivan et al., 1998). The observed point estimates for females are somewhat higher than some [(e.g., $a^2 = 0.41$; 95% CI: 0.31, 0.50; (Reichborn-Kjennerud et al., 2004); ($a^2 = 0.49$; 95% CI: 0.38, 0.61; (Bulik et al., 2003)], but not all reported heritability estimates for binge-eating phenotypes (e.g., $a^2 = 0.82$; 95% CI: 0.68, 0.97; (Bulik et al., 1998). One possible explanation for our higher heritability estimate compared with most previous studies is that the on-line SCID-based instrument (and associated highly structured telephone option) used to assess BE may yield more precise measurement compared to the traditional interview format. Although interviews have been thought to be the gold standard for assessing sensitive information such as psychopathology, data suggest that the anonymity afforded by on-line assessment may confer advantages when assessing sensitive information (Tourangeau & Smith, 1998).

With the exception of self-induced vomiting, which is highly heritable (Sullivan, Bulik, & Kendler, 1998), our results suggest that BE may be one of the more heritable eating disorder-related traits. For example, heritability estimates for body dissatisfaction, a characteristic feature of eating disorders, range from 0.52 (Rutherford, McGuffin, Katz, & Murray, 1993) to 0.59 (Keski-Rahkonen et al., 2005) and for drive for thinness from 0.44 (Rutherford et al., 1993) to 0.51 (Keski-Rahkonen et al., 2005). Although high heritability in no way guarantees mapability, our results support continued focus on BE phenotypes in genetic studies of eating disorders.

**Heritability of night eating—**No studies examining the heritability on NE have been conducted and only one study has examined familial aggregation of NES (Lundgren et al., 2006), thus we do not have findings with which to compare our results. In our study, heritability estimates for NE were moderate for females and males and lower than that observed with BE. Notwithstanding, ours are the first results to document that genetic, not shared environmental influences, explain familial aggregation for NE.

The NE findings should be interpreted with caution given the wide confidence intervals (i.e., lower precision) around the heritability estimates for both sexes. Although the reporting of these first estimates is important given how little is known about the familial transmission of NE, considerably more work must be done in refining the definition of NE and NES before firm conclusions can be drawn.

## Genetic and Environmental Correlations between Binge Eating and Night Eating

Our results speak directly to the debate regarding the extent to which BE and NE represent independent behaviors. The observed genetic correlation of 0.66 reveals considerable overlap in genetic factors that influence liability to BE and NE. Although the twin approach cannot implicate specific genes, it does suggest considerable sharing of genetic factors influencing liability to these two behaviors. However, because the genetic correlation was not unity, genetic factors unique to each trait are also operative. This pattern of results would argue against

conceptualizing BE and NE as either completely independent or completely distinct behavioral phenomena and suggests that, on the genetic level, their etiologies are partially overlapping.

## Limitations

Potential limitations to our study must be considered. First, as our sample represents Swedish adults, results cannot be generalized to other ancestry or age groups. Second, because of the low prevalence of BE we were not able to estimate a bivariate model for males or a sex-limitation model. Third, although twin studies suggest the involvement of genetic factors in etiology, causal conclusions or information regarding specific genes and/or specific unique environmental influences are not possible. Fourth, although the AE model was the most parsimonious for both males and females, model selection based on AIC values can be misleading (Sullivan & Eaves, 2002). Fifth, we were not able to assess the equal environment assumption (EEA; (Kendler, 1993) – whether the greater similarity in MZ twins is due to greater environmental or genetic similarity. However, in general the EEA has not been found to be violated in twin studies of eating disorders (Bulik et al., 1998; Klump, McGue, & Iacono, 2000; Reichborn-Kjennerud et al., 2004; Reichborn-Kjennerud et al., 2003; Sullivan et al., 1998). Sixth, as the diagnostic definitions of BE and NE and the parent syndromes of BED and NES continue to be in flux, our results must be contained to understanding the behaviors as explicitly defined in this study. Finally, self-report measures may lead to inaccurate or biased reporting. In the current sample, questionnaires were administered via computers. The impact of computer-based self-report on reporting of these symptoms is unknown. Although social desirability bias, a phenomenon in which respondents try to present themselves in the most favorable light during face-to-face interviewing particularly for sensitive items, may be decreased with computer-based self-report assessments (Tourangeau & Smith, 1998), it is not known whether differential effects in online reporting exist for males and females. It is however important to keep in mind the prohibitively heavy resource allocation required for interview methods particularly for samples as large as the STR thus making computer-based methods an attractive alternative

## Conclusion

BE and NE are both forms of dysregulated eating. Our findings extend our general understanding of the nature and placement of the boundaries between various expressions of disordered eating. Our findings do not support the hypothesis that BE and NE are alternate expressions of the same underlying trait. Although highly correlated, independent genetic and environmental factors also play a role in liability to these behaviors. Clinically, the relatively frequent co-occurrence of these behaviors and the substantial genetic correlation highlights the importance of consistently screening for both behaviors in individuals presenting with dysregulated eating.

Considerable debate remains regarding the appropriateness of diagnostic status for disorders such as BED and NES. Our study represents a first step in understanding the genetic architecture of the core behaviors associated with these proposed syndromes as well as the relation between those core behaviors. Additional studies of genetic and environmental influences on NE are required to replicate this initial finding as well as multivariate analyses with other core eating disorders features. Finally, additional approaches such as marginal maximum likelihood modeling (Neale et al., 2006) could facilitate identification of promising endophenotypes or liability indices, which, in turn, could promote the refinement of diagnostic criteria to more closely reflect underlying biological mechanisms (Bulik et al., 2007).

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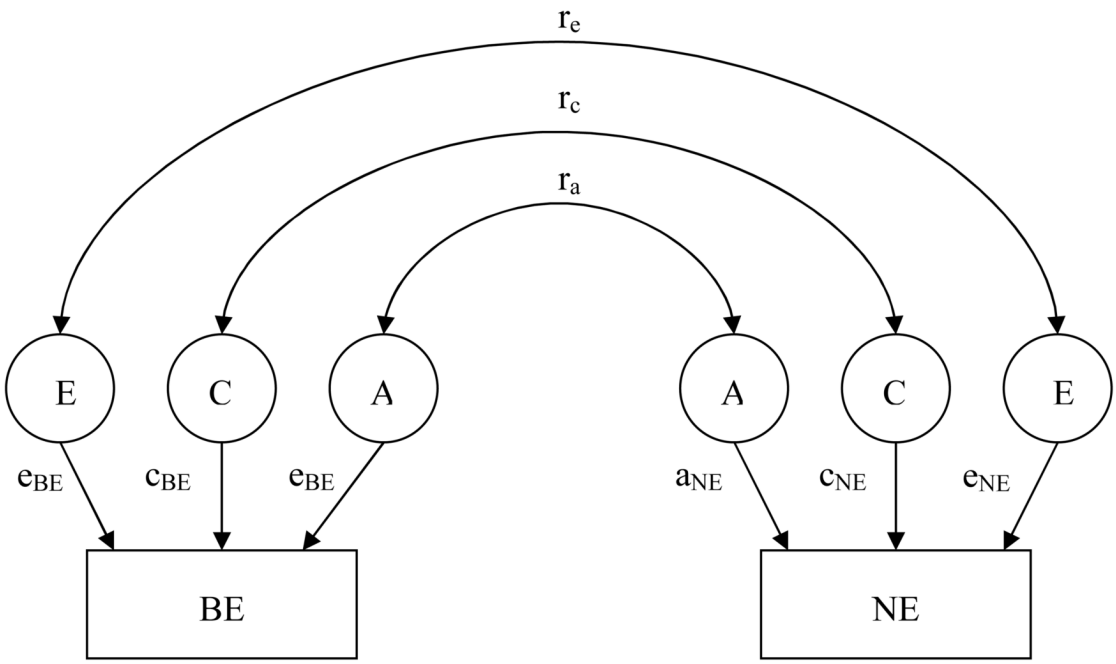


**Figure 1.**
Graphical depiction of full bivariate twin model for lifetime history of binge eating and lifetime history of night eating.

## Table 1

Mean (*SD*) highest and lowest lifetime BMI values by sex for binge eating and night eating.

| | | **No binge eating** | **Binge eating** | ***t* – value (*p* – value)** |
|---|---|---|---|---|
| Females | Highest | 24.7 (4.4) | 26.5 (5.9) | −5.87 (<.001) |
| | Lowest | 19.8 (2.6) | 19.5 (3.7) | 1.83 (.07) |
| Males | Highest | 25.8 (3.6) | 31.6 (7.7) | −4.29 (<.001) |
| | Lowest | 21.5 (2.4) | 23.3 (3.9) | −2.47 (.020) |
| | | No night eating | Night eating | *t* – value (*p* – value) |
| Females | Highest | 24.7 (4.4) | 26.0 (6.0) | −3.01 (.003) |
| | Lowest | 19.8 (2.6) | 19.3 (3.3) | 2.24 (.026) |
| Males | Highest | 25.8 (3.6) | 27.3 (4.8) | −4.30 (<.001) |
| | Lowest | 21.5 (2.4) | 21.9 (2.8) | −1.89 (.061) |

*Note*. *SD* = standard deviation. BMI = body mass index.

**Table 2**

Tetrachoric correlation coefficients between binge eating and night eating.

| | Within-Trait | | | | | | Cross-Trait | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Binge Eating | | | Night Eating | | | Within-Twin | | | Cross-Twin | | |
| **Zyogsity** | ***N*** | ***r*** | **95% CI** | ***N*** | ***r*** | **95% CI** | ***N*** | ***r*** | **95% CI** | ***N*** | ***r*** | **95% CI** |
| MZ females | 3796 | 0.69 | 0.59, 0.79 | 3668 | 0.33 | 0.13, 0.53 | 3925 | 0.35 | 0.23, 0.47 | 3730 | 0.25 | 0.13, 0.37 |
| DZ females | 2698 | 0.36 | 0.18, 0.54 | 2582 | 0.17 | −0.10, 0.44 | 2783 | 0.34 | 0.20, 0.48 | 2639 | 0.09 | −0.09, 0.27 |
| MZ males | 2514 | 0.74 | 0.47, 1.00 | 2426 | 0.46 | 0.28, 0.64 | 2632 | 0.19 | −0.10, 0.48 | 2468 | 0.24 | −0.05, 0.53 |
| DZ males | 1772 | 0.50 | −0.07, 1.00 | 1712 | 0.07 | −0.24, 0.38 | 1842 | 0.60 | 0.22, 0.82 | 1740 | 0.03 | −0.44, 0.50 |

*Note*. CI = confidence interval. MZ = monozygotic. DZ = dizygotic.

## Table 3

**Table 3a. Model fit statistics for the binge eating and night eating bivariate models for females.**

| Model Number. | Model | −2LL | df | $\chi^2$ diff (df) | AIC |
|---|---|---|---|---|---|
| 1 | ACE-ACE $r_a\ r_c\ r_e$ | 4800.22 | 13636 | --- | −22471.78 |
| 2 | ACE-ACE $r_c\ r_e$ | 4803.48 | 13637 | 3.26 (1) | −22470.52 |
| 3 | ACE-ACE $r_a\ r_e$ | 4800.26 | 13637 | 0.04 (1) | −22473.74 |
| 4 | ACE-AE $r_a\ r_e$ | 4800.26 | 13638 | 0.04 (2) | −22475.74 |
| 5 | AE-AE $r_a\ r_e$ | 4800.42 | 13639 | 0.20 (3) | −22477.58 |
| 6 | AE-AE $r_a$ (best fit) | 4802.27 | 13640 | 2.05 (4) | −22477.73 |
| 7 | CE-CE $r_c\ r_e$ | 4813.86 | 13639 | 13.64 (3) | −22464.14 |
| 8 | E-E $r_e$ | 4958.27 | 13642 | 158.05 (6) | −22325.73 |

**Table 3b. Parameter estimates (95% confidence interval) from the binge eating and night eating bivariate models for females.**

| | | Binge Eating | | | Night Eating | | | Correlations | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Model Number | Model | $a^2$ | $c^2$ | $e^2$ | $a^2$ | $c^2$ | $e^2$ | $r_a$ | $r_c$ | $r_e$ |
| 1 | ACE-ACE, $r_a\ r_c\ r_e$ | 0.63 (0.26, 0.77) | 0.06 (0.00, 0.39) | 0.31 (0.23, 0.41) | 0.33 (0.00, 0.52) | 0.02 (0.00, 0.38) | 0.65 (0.45, 0.84) | 0.69 (−1.00, 1.00) | −1.00 (−1.00, 1.00) | 0.16 (−0.09, 0.40) |
| 2 | ACE-ACE $r_c\ r_e$ | 0.48 (0.19, 0.64) | 0.19 (0.06, 0.45) | 0.33 (0.25, 0.42) | 0.05 (0.00, 0.33) | 0.26 (0.07, 0.44) | 0.70 (0.52, 0.86) | --- | 1.00 (0.52, 1.00) | 0.26 (0.06, 0.46) |
| 3 | ACE-ACE $r_a\ r_e$ | 0.62 (0.26, 0.77) | 0.07 (0.00, 0.39) | 0.31 (0.23, 0.41) | 0.35 (0.05, 0.52) | 0.00 (0.00, 0.33) | 0.65 (0.48, 0.85) | 0.60 (0.35, 1.00) | --- | 0.16 (−0.08, 0.39) |
| 4 | ACE-AE $r_a\ r_e$ | 0.62 (0.26, 0.77) | 0.07 (0.00, 0.39) | 0.31 (0.23, 0.41) | 0.35 (0.16, 0.52) | --- | 0.65 (0.48, 0.84) | 0.60 (0.35, 1.00) | --- | 0.16 (−0.08, 0.39) |
| 5 | AE-AE $r_a\ r_e$ | 0.69 (0.60, 0.77) | --- | 0.31 (0.23, 0.40) | 0.35 (0.16, 0.52) | --- | 0.65 (0.48, 0.84) | 0.57 (0.34, 0.87) | | 0.17 (−0.07, 0.40) |
| 6 | AE-AE $r_a$ (best fit) | 0.69 (0.60, 0.77) | --- | 0.31 (0.23, 0.40) | 0.35 (0.17, 0.52) | --- | 0.65 (0.48, 0.83) | 0.66 (0.48, 0.96) | --- | --- |

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| Table 3b. Parameter estimates (95% confidence interval) from the binge eating and night eating bivariate models for females. | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | Binge Eating | | | Night Eating | | | Correlations | | |
| **Model Number** | **Model** | **$a^2$** | **$c^2$** | **$e^2$** | **$a^2$** | **$c^2$** | **$e^2$** | **$r_a$** | **$r_c$** | **$r_e$** |
| 7 | CE-CE $r_c$ $r_e$ | --- | 0.59 (0.50, 0.66) | 0.41 (0.34, 0.50) | --- | 0.28 (0.12, 0.43) | 0.71 (0.57, 0.88) | --- | 0.56 (0.32, 0.88) | 0.23 (0.04, 0.41) |
| 8 | E-E $r_e$ | --- | | 1.00 | --- | --- | 1.00 | --- | --- | 0.35 (0.26, 0.43) |

*Note*. For each model, the first set of parameters (ACE) refers to binge eating and the second set to night eating. AIC = Akiake's Information Criterion (Akiake, 1987); −2LL = −2 log likelihood; df = degrees of freedom; ACE = additive genetic, shared (or common) environment, and unique environmental effects model; AE = additive genetic and unique environmental effects model; CE = shared (or common) and unique environmental effects model; E = unique environmental effects model; $r_a$ = genetic correlation between binge eating and night eating; $r_c$ = shared environmental correlation between binge eating and night eating; $r_e$ = unique environmental correlation between binge eating and night eating.

*Note*. For each model, the first set of parameters (ACE) refers to binge eating and the second set to night eating. AIC = Akiake's Information Criterion (Akiake, 1987); −2LL = −2 loglikelihood; df = degrees of freedom; ACE = additive genetic, shared (or common) environment, and unique environmental effects model; AE = additive genetic and unique environmental effects model; CE = shared (or common) and unique environmental effects model; E = unique environmental effects model; $r_a$ = genetic correlation between binge eating and night eating; $r_c$ = shared environmental correlation between binge eating and night eating; $r_e$ = unique environmental correlation between binge eating and night eating; $a^2$ = additive genetic variance; $c^2$ = shared (or common) environmental variance; $e^2$ = unique environmental variance.

**Table 4**

Results from the binge eating univariate models for males.

| | Model Fit Statistics | | | | Parameter Estimates (95% confidence interval) | | |
|---|---|---|---|---|---|---|---|
| **Model** | **−2LL** | **df** | **$\chi^2$ diff (df)** | **AIC** | **$a^2$** | **$c^2$** | **$e^2$** |
| ACE | 368.69 | 4528 | --- | −8687 | 0.74 (0.00, 0.93) | 0.00 (0.00, 0.80) | 0.26 (0.07, 0.64) |
| AE (best fit) | 368.69 | 4529 | 0.00 (1) | −8689 | 0.74 (0.36, 0.93) | --- | 0.26 (0.07, 0.64) |
| CE | 370.57 | 4529 | 1.88 (1) | −8687 | --- | 0.63 (0.26, 0.85) | 0.37 (0.15, 0.74) |
| E | 380.76 | 4530 | 12.07 (2) | −8679 | --- | --- | 1.00 |

*Note*. AIC = Akiake's Information Criterion (Akiake, 1987); −2LL = −2 loglikelihood; df = degrees of freedom; ACE = additive genetic, shared (or common) environment, and unique environmental effects model; AE = additive genetic and unique environmental effects model; CE = shared (or common) and unique environmental effects model; E = unique environmental effects model; $a^2$ = additive genetic variance; $c^2$ = shared (or common) environmental variance; $e^2$ = unique environmental variance

**Table 5**

Results from the night eating univariate models for males.

| | Model Fit Statistics | | | | Parameter Estimates (95% confidence interval) | | |
|---|---|---|---|---|---|---|---|
| **Model** | **−2LL** | **df** | **$\chi^2$ diff (df)** | **AIC** | **$a^2$** | **$c^2$** | **$e^2$** |
| ACE | 1564.00 | 4440 | --- | −7316 | 0.44 (0.00, 0.61) | 0.00 (0.00, 0.35) | 0.56 (0.39, 0.76) |
| AE (best fit) | 1564.00 | 4441 | 0.00 (1) | −7318 | 0.44 (0.24, 0.61) | --- | 0.56 (0.39, 0.76) |
| CE | 1567.91 | 4441 | 3.90 (1) | −7314 | --- | 0.33 (0.16, 0.49) | 0.67 (0.51, 0.84) |
| E | 1581.95 | 4442 | 17.95 (2) | −7302 | --- | --- | 1.00 |

*Note*. AIC = Akiake's Information Criterion (Akiake, 1987); −2LL = −2 loglikelihood; df = degrees of freedom; ACE = additive genetic, shared (or common) environment, and unique environmental effects model; AE = additive genetic and unique environmental effects model; CE = shared (or common) and unique environmental effects model; E = unique environmental effects model; $a^2$ = additive genetic variance; $c^2$ = shared (or common) environmental variance; $e^2$ = unique environmental variance.