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Genetic and Environmental Factors Underlying Comorbid Bulimic Behaviours and Alcohol Use Disorders: A Moderating Role for the Dysregulated Personality Cluster?

Jennifer D. Slane^{1,2,*}, Kelly L. Klump³, Matthew McGue⁴, and G. Iacono⁴

¹VISN 4 Mental Illness Research, Education and Clinical Center, VA Pittsburgh Healthcare System, Pittsburgh, PA, USA

²University of Pittsburgh Medical Center, Department of Psychiatry, Pittsburgh, PA, USA

³Department of Psychology at Michigan State University, East Lansing, MI, USA

⁴Department of Psychology at the University of Minnesota, Minneapolis, MN, USA

Abstract

Women with bulimia nervosa (BN) frequently have co-occurring alcohol use disorders (AUDs). Studies of shared genetic transmission of these disorders have been mixed. Personality heterogeneity among individuals with BN may explain discrepant findings. Cluster analysis has characterized women with BN in groups on the basis of personality profiles. One group, the Dysregulated cluster, characterized largely by behavioural disinhibition and emotional dysregulation may be more closely linked etiologically to AUDs. This study examined whether genetic associations between BN and AUDs are the strongest among the Dysregulated cluster. Symptoms of BN and AUDs were assessed in female twins at ages 17 and 25 years from the Minnesota Twin Family Study. Personality clusters were defined using the Multidimensional Personality Questionnaire. Twin moderation models suggested small-to-moderate common genetic transmission between BN and AUDs. However, shared genetic effects did not differ by personality cluster. Findings suggest that personality clusters are unlikely to account for inconsistent findings regarding their shared aetiology.

Keywords

bulimia nervosa; alcohol use disorders; personality

Individuals with bulimia nervosa (BN) frequently have comorbid alcohol use disorders (AUDs; Dansky, Brewerton, & Kilpatrick, 2000; Holderness, Brooks-Gunn, & Warren, 1994; Wilson, 1991). Comorbidity rates in women with BN have ranged from 33% to 47% (Bulik, Sullivan, Carter, & Joyce, 1997; Mitchell, Hatsukami, Eckert, & Pyle, 1985), with an estimated median prevalence rate of 22.9% (Holderness et al., 1994). The co-occurrence of

these disorders is associated with many maladaptive problems (Dansky et al., 2000; Duncan et al., 2006; Keel, Mitchell, Miller, Davis, & Crow, 1999). For example, women with comorbid BN and AUDs have higher rates of attempted suicide, and they are more likely to have diagnoses of drug dependence and major depressive disorder than women with BN or AUDs alone (Duncan et al., 2006).

Given the deleterious effects of the co-occurrence of BN and AUDs, several researchers have used family and twin studies to determine whether there is some shared aetiology in their development. Family studies have generally indicated that these disorders are transmitted independently (Bulik, 1987; Kasset et al., 1989; Lilenfeld et al., 1997); however, higher rates of AUDs have been indicated among family members of BN probands with co-occurring alcohol problems compared with BN probands without this comorbidity (Lilenfeld et al., 1997). Yet, twin studies are necessary to confirm these findings, as family studies cannot indicate whether familial co-aggregation is due to genetic influences. Importantly, BN and AUDs, as well as symptoms of these disorders, have been found to have moderate-to-high heritabilities (Bulik, Sullivan, Wade, & Kendler, 2000; Heath et al., 1997; Prescott, Aggen, & Kendler, 1999; Reichborn-Kjennerud, Bulik, Tambs, & Harris, 2004; Sullivan, Bulik, & Kendler, 1998; Whitfield et al., 2004). However, to date, only five twin studies have examined associations between BN and AUDs to determine whether genetic and/or environmental influences underlie their comorbidity with mixed results. In the first study, 'problem drinking' and BN were found to have only a small genetic overlap (i.e. 6%) with results suggesting that they primarily load on two separate and independent genetic factors, suggesting distinct etiologies (Kendler et al., 1995). Further, findings indicated that shared environmental influences (those that are common to siblings and act to make them similar to each other) and nonshared environmental influences (environmental factors, and measurement error, that differentiate twins within a pair) were predominately disorder-specific, suggesting that there is no overlap in these factors. Mitchell et al. (2010) also found mixed evidence for shared genetic effects. Although this study observed large genetic correlations [i.e. 1.0; 95% confidence intervals (CIs): -1.0, 1.0; correlations that indicate the degree of overlap in genetic risk factors for two phenotypes] and moderate nonshared environmental correlation (.53; CIs: -.32, 1.0; correlations that indicate the degree of overlap in nonshared environmental risk factors for two phenotypes), both estimates had wide CIs that overlapped with zero, indicating that they were not statistically significant effects.

By contrast, four twin studies suggested significant genetic overlap between BN and AUDs. Baker, Mitchell, Neale, and Kendler (2010) found a moderate genetic correlation between BN and AUDs (.53; CIs: .30, .80) and a small, nonsignificant nonshared environmental correlation between these phenotypes (-.03; CIs: -.24, .18). A recent study identified a genetic correlation of .23 (CIs: .01, .44) between BN and AUDs, as well as a nonshared environmental correlation of .35 (CIs: .08, .61; Trace et al., 2013). With regard to the primary symptoms of BN (i.e. binge eating and compensatory behaviours), evidence was indicated for common genetic etiologies between binge eating and compensatory behaviours and alcohol use, with genetic correlations of .31 (CIs: .09, .53) for binge eating and .61 for compensatory behaviour (CIs: .34, 1.0; Slane, Burt, & Klump, 2012). Similarly, another study found genetic correlations of .29 (CIs: .11, .47) for binge eating and AUDs, and .32

(CIs: .19, .45) for compensatory behaviours and AUDs (Munn-Chernoff et al., 2013). Notably, in both studies examining BN symptoms, no overlap in shared or nonshared environmental factors were detected, as shared environmental factors were estimated to be close to zero, and nonshared environmental influences were all disorder-specific.

Overall, results are split with three studies finding evidence for common genetic factors, and two others suggesting no shared aetiology. It is unclear why results have been inconsistent. However, one hypothesis is that extant twin studies have not considered the influence of personality. Personality traits are moderately heritable (Jang, Livesley, & Vernon, 1996; Jang, Livesley, Vernon, & Jackson, 1996) and have been studied extensively in BN with findings indicating that women with BN often show a heterogeneous mix of personality and temperamental traits (Vitousek & Manke, 1994; Westen, Thompson-Brenner, & Peart, 2006). This heterogeneity may reflect etiologic differences in the development of BN that results in distinct comorbidities (e.g. AUDs) and genetic risk profiles. Studies have begun to use a personality profiling/cluster analytic approach to personality classification in women with BN in order to decrease heterogeneity and obtain more homogeneous groups that may reflect common etiologic factors.

Most cluster analytic studies of eating disorders have found three clusters of women that include a high-functioning/perfectionistic group (i.e. conscientious, perfectionistic and anxious), a constricted/overcontrolled group (i.e. passive, avoidant and emotionally constricted) and an emotionally dysregulated/undercontrolled group (i.e. emotionally intense and labile, impulsive, poor negative mood regulation and behaviourally disinhibited; Westen & Harnden-Fischer, 2001). Among these clusters, the emotionally dysregulated/undercontrolled cluster, frequently labelled the 'dysregulated' cluster (Espelage, Mazzeo, Sherman, & Thompson, 2002; Thompson-Brenner, Eddy, Satir, Boisseau, & Westen, 2008; Thompson-Brenner & Westen, 2005; Westen & Harnden-Fischer, 2001), is the most stable cluster across time (Slane, Klump, Donnellan, McGue, & Iacono, 2013), and AUDs are more common in this group compared with other clusters (Claes et al., 2006; Thompson-Brenner & Westen, 2005; Wonderlich et al., 2005). Further, the dysregulated cluster tends to consist primarily of women who exhibit bulimic behaviours (e.g. bingeing and purging) rather than women who restrict their intake only (Claes et al., 2006; Perkins, Slane, & Klump, 2013; Thompson-Brenner et al., 2008; Westen and Harnden-Fischer, 2001). The frequent co-occurrence of BN symptoms and AUDs in the dysregulated group suggests that this may be an ideal group to examine shared aetiology between the disorders. Indeed, this group could represent a unique subtype among individuals with BN that is more closely linked etiologically to AUDs.

The overall aim of the present study was to examine whether etiologic associations between BN and AUD symptoms are the strongest among individuals in the dysregulated cluster as compared with individuals from other personality clusters. Data from the Minnesota Twin Family Study (MTFS) that was previously cluster-analysed at two time points (i.e. at ages 17 and 25 years; Slane et al., 2013) were utilized in the present study. Given that personality traits tend to change from adolescence into adulthood, with a trend towards increased growth and maturity in traits (Slane et al., 2013), and BN and AUD symptoms also increase

during this developmental period, the two ages were examined to determine whether findings differ on the basis of twin age.

Methods

Participants

Participants included a sample of female twins from the MTFS. The MTFS is a population-based, longitudinal study of reared-together same-sex female and male twins and their parents (see Iacono, Carlson, Taylor, Elkins, & McGue, 1999 for study details). Briefly, public databases were utilized to obtain birth records used to identify twins born in the state of Minnesota. Over 90% of twins born between 1971 and 1985 were located.

The current study utilized data from two cohorts of MTFS female twins. The first cohort began the study when they were 11 years old, whereas the second cohort began the study at age 17 years. Twins from both cohorts were included in the study if they had data available for at least one time point (i.e. age 17 or 25 years). The age 17 cohort ($M = 17.87$; $SD = 0.74$) included 1,264 twins [750 monozygotic (MZ); 426 dizygotic (DZ)] and the age 25 cohort ($M = 25.05$; $SD = 0.69$) included 1,184 twins (690 MZ; 392 DZ). There were 88 and 102 participants without co-twin data included in analysis in the age 17 and age 25 cohorts, respectively. A total of 178 and 98 participants only had data available at age 17 and age 25 years, respectively, but not at both time points.

Zygosity determination

Zygosity was determined using three separate methods (Iacono, Malone, & McGue, 2003). First, the twins' parents completed a physical similarity questionnaire. Second, research staff evaluated the similarity of twins' physical characteristics. Third, each twin's ponderal index, cephalic index and number of fingerprint ridges were measured. Disagreements among the three methods were resolved using the highly accurate method of serological analysis (McGue, Elkins, & Iacono, 2000).

Measures

Bulimic behaviours—Bulimic behaviours were assessed using the Eating Disorders Structured Clinical Interview (EDSCI) and the Minnesota Eating Behavior Survey (MEBS; von Ranson, Klump, Iacono, & McGue, 2005).¹ The EDSCI and MEBS were administered at both time points.

Bulimia nervosa symptoms—The EDSCI is a semi-structured interview based on the Structured Clinical Interview for the Diagnostic and Statistical Manual of Mental Disorders (DSM) Axis I Disorders (SCID; Spitzer, Williams, & Gibbon, 1987). The BN symptoms that were assessed include binge eating, loss of control over binge eating, purging (e.g. vomiting and laxatives) and nonpurging (e.g. excessive exercise and fasting) behaviours,

¹The MEBS [previously known as the Minnesota Eating Disorder Inventory (M-EDI)] was adapted and reproduced by special permission of Psychological Assessment Resources, Inc., 16204 North Florida Avenue, Lutz, Florida 33549, from the Eating Disorder Inventory (collectively, EDI and EDI-2) by Garner, Olmstead, Polivy, Copyright 1983 by Psychological Assessment Resources, Inc. Further reproduction of the MEBS is prohibited without prior permission from Psychological Assessment Resources, Inc.

and the undue influence of shape and weight on self-evaluation. Because of the relatively low prevalence of BN diagnoses in the population (Hudson, Hiripi, Harrison, & Kessler, 2007) as well as the current sample [i.e. eight probable/definite diagnoses at age 17 years (0.6%) and four at age 25 years (0.3%)], the EDSCI BN symptom counts were used in analyses instead of diagnoses. Each symptom was counted as 1, and the symptom count is a sum of these scores with a range from 0 to 8. The BN symptom counts allow for the examination of clinical levels of symptomatology without having reduced power due to a small number of full diagnoses.

'Best estimates' of lifetime BN symptoms were assessed at age 17 years. That is, both the twin's mother and the twin herself reported on all BN symptoms, and best estimates coded a symptom as present if either the mother or twin indicated that it was present. Symptoms at age 25 years were based on twin report only (as mothers did not report on symptoms at this time point) and include symptoms present over the earlier 3–4 years. Importantly, reliability of BN diagnoses (based on symptom counts) was good, with a kappa coefficient of .72 for the mother and twin combined report at age 17 years and .71 for the adult self-report. These kappa coefficients were similar to those for the combined (parent and twin) reports of other disorders assessed in the MTFs, such as major depression and social phobia (.71 and .80, respectively).

Minnesota Eating Behavior Survey—The MEBS was also used to assess bulimic behaviours. This 30-item true/false self-report questionnaire assesses overall levels of eating pathology (total score) as well as specific disordered eating symptoms including body dissatisfaction (i.e. dissatisfaction with one's size or shape), binge eating (i.e. thoughts about overeating or tendency to binge eat), compensatory behaviours (i.e. the use of compensatory behaviours, such as self-induced vomiting, for weight loss) and weight preoccupation (i.e. preoccupation with dieting, thinness and weight). Scores range from 0 to 30 for the total score, 0 to 6 for body dissatisfaction, 0 to 7 for binge eating, 0 to 6 for compensatory behaviours and 0 to 8 for weight preoccupation. Examining these continuous measures of bulimic behaviours allowed for a more fine-tuned analysis of BN and component symptoms. Importantly, the MEBS has shown good psychometric properties (von Ranson et al., 2005). Alpha reliabilities for the present sample were .69 or greater for all scales at both ages with the exception of compensatory behaviour, which was .57 at age 17 years and .56 at age 25 years (Table 1). Thus, the results with compensatory behaviour should be interpreted with caution.

AUD symptoms—Alcohol abuse and dependence symptoms (i.e. AUD symptoms) were assessed at both ages using the Substance Abuse Module (SAM) from the Composite International Diagnostic Interview (Robins, Babor, & Cottler, 1987). These include symptoms such as recurrent alcohol-related legal problems, tolerance and withdrawal. The SAM is a well-established, semi-structured interview measure of these symptoms that has been used in field trials for the development of the DSM (Cottler et al., 1995; Spitzer et al., 1987) and has been shown to be valid for use in adolescents (Crowley, Mikulich, Ehlers, Whitmore, & MacDonald, 2001). The SAM shows excellent inter-rater reliability (Cottler, Robins, & Helzer, 1989), with an average kappa reliability of 0.92 for individual alcohol

abuse and dependence symptoms. For the purposes of this study, an AUD composite score (i.e. a continuous variable composed of a sum of all alcohol abuse and alcohol dependence symptoms) was used in analyses with a score range from 0 to 11.

Notably, only DSM-III-R data were available for both cohorts at age 17 years. However, at age 25 years, both DSM-III-R and DSM-IV symptoms were assessed. Analyses were conducted with DSM-III-R data at age 17 years and DSM-IV data at age 25 years. Importantly, DSM-III-R and DSM-IV alcohol abuse and dependence criteria are very similar, as the transition between these manuals was ‘a much more conservative process’ than previous years (Hasin, 2003, p. 8). Changes predominately included either combining symptoms into a single criterion or separating symptoms into two criteria (Hasin, 2003).

Procedures for diagnostic interviews—The DSM AUD and BN symptoms were assessed by trained bachelor’s and master’s level clinicians. Each symptom assigned was discussed in a clinical case conference with at least two advanced clinical psychology doctoral students. Discrepancies that emerged during the case conference were clarified by listening to interview audiotapes or re-contacting study participants. Symptoms were counted as present if they qualified for clinical significance in both frequency and severity.

Personality clusters—The present study utilized clusters that were previously identified using the facet scales of the Multidimensional Personality Questionnaire assessed longitudinally at two time points (i.e. ages 17 and 25 years; Slane et al., 2013). Data were from the MTFs, and four clusters were identified at age 17 years, whereas three were identified at age 25 years. These clusters included the Resilient, Sensation Seeking and Dysregulated clusters at both time points, and an additional Inhibited cluster at age 17 years. Notably, a twin’s cluster membership could differ between ages 17 and 25 years, that is, cluster membership was not fixed to be equal across the two time points.

As stated previously, the Dysregulated cluster is characterized as interpersonally aggressive, emotionally labile and impulsive. The Resilient cluster tends to be cautious, have high moral standards and value social closeness. The Sensation Seeking cluster is characterized as decisive, persuasive, with a preference for novel, risky activities. Lastly, women in the Inhibited cluster tend to opt for safe activities over risky ones, prefer being alone and not being the centre of attention, and are not very imaginative.

Statistical analyses

Data preparation and descriptive statistics—Prior to analyses, log transformations ($\log_{10} X + 1$) were performed for the Binge Eating scale, Compensatory Behaviours scale, DSM BN symptoms and Alcohol Composite (i.e. AUD symptoms) to account for positive skew.

Phenotypic associations—In order to examine initial phenotypic associations, cross-sectional Pearson correlations were calculated within cluster at each age between bulimic behaviours and AUD symptoms. Note that moderation by the Dysregulated cluster was the primary focus of the present study, as this cluster has increased levels of bulimic behaviours and AUD symptoms compared with the other clusters. Given this focus and the fact that the

present study did not have specific hypotheses about the remaining clusters, the age 17 Resilient, Sensation Seeking and Inhibited clusters, and the age 25 Resilient and Sensation seeking clusters were collapsed at each time point into one cluster labelled 'Other clusters' as a comparison group for the Dysregulated cluster.

Genetic and environmental associations—Bivariate models (i.e. models used to examine the co-aggregation of two phenotypes or traits in twins) were initially examined to determine whether there is genetic overlap between BN and AUD symptoms at each time point. Models at each time point (ages 17 and 25 years) included all twins, regardless of cluster membership.

In order to examine the influence of a moderator variable (i.e. cluster membership; Dysregulated vs Other clusters) on genetic and environmental influences on *associations* between BN and AUD symptoms, a gene–environment interaction model (i.e. G×E model; a model testing whether genetic and/or environment influences on a trait/disorder differ by levels of a moderator) was examined (Purcell, 2002). Typically, G×E models are used to examine the effects of a moderator (e.g. stressful life events) on genetic and environmental influences on one trait or disorder (e.g. depression). However, a modification of the G×E in the presence of r_{GE} model (i.e. a model that controls for genetic and/or environmental associations between the moderator and the primary phenotype, where r_{GE} is a measure of the degree of this association) was used in the present study to examine the extent to which a moderator influences the degree of genetic and/or environmental associations between two phenotypes (i.e. BN and AUD). In the current study, this modified model was used to determine whether there are differences in the extent to which genetic (and environmental) factors influence the covariation between BN and AUD across levels of the moderator (i.e. Dysregulated vs Other clusters).

The altered G×E in the presence of r_{GE} moderator model, used in the present study, is shown in Figure 1. In this figure, there are three estimates of genetic influences. First, there is an estimate of the genetic effects specific to AUD symptoms (i.e. specific to the first phenotype in the model; a_{AUD}). Second, there are genetic factors unique to BN symptoms (i.e. specific to the second phenotype in the model, after accounting for the overlap with AUD symptoms; a_{BUL}). Third, there are genetic influences common to both phenotypes (a_C ; i.e. overlap in genetic effects influencing BN and AUD symptoms). It is the genetic (and environmental) influences on this covariance path and their potential moderation by personality cluster that is of key interest in the present study. Shared and nonshared environmental influences are also estimated, but are not included in Figure 1.

Moderation effects (i.e. β_C and β_{BUL}) are also estimated in the model. The model (Purcell, 2002) includes the influence of these β coefficients on the cross-path and the unique path to the second phenotype (in this case, bulimic behaviours; see Figure 1). The first β coefficient (β_C) represents differences (either positive or negative) in genetic or environmental influences on *the covariance between BN and AUD symptoms* on the basis of the effect of the moderator (i.e. cluster membership). The β_C moderator coefficient is of particular interest in the present study as it addresses the primary aim indicating whether genetic influences on the covariance between bulimic behaviours and AUD symptoms are the

strongest in the Dysregulated cluster compared with the Other clusters. Specifically, a positive and significant estimate indicates increased influence of genetic effects in the Dysregulated cluster compared with the Other clusters. By contrast, a nonsignificant estimate indicates that there is no change in genetic effects by cluster and a negative, and significant estimate indicates that the genetic factors are lower in the Dysregulated cluster compared with the Other clusters.

The second β coefficient (β_{BUL}) indicates whether there are changes in genetic effects that are unique to bulimic behaviours on the basis of changes in cluster membership. It may be that there are no changes in genetic effects on the *covariance* between bulimic behaviours and AUD symptoms with different levels of cluster membership, but that there is a moderator effect on genetic influences specific to bulimic behaviours. For example, if this coefficient is significant and positive, it would indicate that genetic factors on bulimic behaviours are greater in the Dysregulated cluster compared with the Other clusters. Thus, this would suggest that women in the Dysregulated cluster have an increased genetic risk for developing bulimic behaviours. As specified in this study, these models do not provide unique estimates of genetic factors (or changes in these effects) on AUD symptoms. Reverse models, with the phenotypes entered in the opposite order, could be examined to determine the unique impact of the moderator on AUD symptoms; however, given that the present study does not have hypotheses about the influence of cluster membership on unique effects on AUD symptoms, these reverse models were not examined.

Two groups of models were examined in the present study: (i) cross-sectional models with age 17 cluster membership, age 17 bulimic behaviours and age 17 AUD symptoms, and (i) cross-sectional models with age 25 cluster membership, age 25 bulimic behaviours and age 25 AUD symptoms. Each of the modified G×E in the presence of r_{GE} moderator models (Purcell, 2002) were fit to the raw data using full-information maximum-likelihood in Mx (Neale, 1997). Initially, a full ACE model with main and moderation effects was fit to the data. Next, a full AE submodel (with main and moderation effects) of the ACE model was examined to determine if a reduced model provides a better fit to the data given that BN and AUD symptoms are influenced primarily by additive genetic (A; the effect of individual genes summed over loci) and nonshared environmental (E; environmental factors, and measurement error, that differentiate twins within a pair) factors in late adolescence and adulthood (e.g. Bulik, Sullivan, & Kendler, 1998; Heath et al., 1997). Additional submodels (dropping the moderation coefficients) were not examined, as the remaining sources of variance would have absorbed the dropped effects and CIs for other estimates in the model would be artificially narrowed (Sullivan & Eaves, 2002). Although dropping the shared environmental effects (C; environmental influences that are common to both twins and act to make them similar to each other) can also have this influence, the AE model with full moderation was examined in the present study because of consistent extant research indicating little to no influence of these effects on BN or AUDs in adulthood.

The fit of all models was examined using a likelihood-ratio chi-square goodness of fit (χ^2) and Akaike's information criterion ($AIC = \chi^2 - 2df$; Akaike, 1987). The likelihood-ratio chi-square goodness of fit (χ^2) examines differences in an index of fit (minus twice the log likelihood; $-2\ln L$) and degrees of freedom between the AE submodel and the full ACE

model. If the chi-square goodness of fit is nonsignificant, then the most parsimonious (i.e. the model containing fewer estimated parameters, and hence more degrees of freedom) model is preferred. In addition to this test, the AIC is utilized to compare model fit, with the lowest AICs indicating better fit.

Prior to conducting model fit analyses, some data preparation was necessary. First, all variables (including previously log transformed variables) were standardized (i.e. z-transformed) prior to twin analysis. This transformation has been conducted in previous research (Burt & Klump, 2009; Klump, Perkins, Burt, McGue, & Iacono, 2007) as it aids in interpretation of moderation effects. Second, the clusters were coded dichotomously as 'Other clusters' (coded 0) and Dysregulated cluster (coded 1) in all of the models.²

In all tables, unstandardized parameter estimates from the models are reported because standardized estimates can be misleading with regard to changes in genetic and environmental effects across levels of the moderator. For example, if the moderator causes an increase in genetic effects at different levels of the moderator, but the environmental effects stay constant, standardization of parameter estimates would make it appear as though environmental effects are decreasing as genetic effects are increasing because the standardization requires that the estimates are proportional (i.e. A, C and E estimates add up to 1.0). Thus, the unstandardized estimates allow for a straightforward indication of absolute changes in the genetic and environmental effects with changes in the moderator. However, in order to allow for easier comparison to previously published twin studies of these phenotypes, some standardized values and corresponding CIs were included in the results. These are calculated by squaring the unstandardized estimates (and unstandardized CIs).

Results

Descriptive statistics

Means and standard deviations for the bulimic behaviours and AUD symptoms within the Dysregulated cluster and Other clusters at both time points are presented in Table 1. Importantly, at both time points, there was sufficient variability in the score ranges as well as the percent of participants scoring above the clinical cut-offs for eating disorders (i.e. mean scores among individuals with eating disorders) on the MEBS scale (age 17 years: 5–28%; age 25 years: 5–35%; von Ranson et al., 2005).

Mean differences

As shown in Table 1, means of bulimic behaviours, BN symptoms and AUD symptoms were significantly different between the Dysregulated and Other clusters. In each case, the Dysregulated cluster had higher levels of symptoms compared with the Other clusters. The only exceptions to this general pattern were for compensatory behaviours at age 17 years and BN symptoms at age 25 years. In the first case, although compensatory behaviour scores were higher in the Dysregulated group, they were not significantly greater than the scores

²Twin moderation models were also examined with all clusters included and ordered by mean levels of AUD symptoms and bulimic behaviors at each time point, from the lowest levels (coded 0) to the highest levels (coded 3 at the age of 17 years and 2 at the age of 25 years; data not shown). Results were nearly identical for these models as those reported herein (data not shown).

for the Other clusters. In the latter case, BN symptoms were lower among the Dysregulated cluster as compared with the Other clusters. This finding was somewhat surprising given higher levels of bulimic symptoms in this group; however, it may be due to the low rates of symptoms and diagnoses in general at this age.

Phenotypic associations

Pearson correlations³ were calculated to examine phenotypic associations between bulimic behaviours and AUD symptoms within the Dysregulated cluster versus the Other clusters (Table 2) at both time points. At age 17 years, nearly all of the bulimic behaviours (with the exception of weight preoccupation) were significantly and positively associated with AUD symptoms in the Dysregulated cluster ($r_s = .16-.29$). Surprisingly, nearly all of the bulimic behaviours also were significantly and positively associated with AUD symptoms in the Other clusters, although correlations were comparatively smaller ($r_s = .12-.15$).

In contrast to age 17 correlations, at age 25 years, the only significant correlation in the Dysregulated cluster was between DSM BN symptoms and AUD symptoms ($r = .15$). In the Other clusters, small associations were indicated between each of the bulimic behaviours and AUD symptoms ($r_s = .08-.15$), with the exceptions of body dissatisfaction and weight preoccupation. Hypotheses that there would be a stronger association between bulimic behaviours and AUD symptoms in the Dysregulated cluster compared with the Other clusters were not confirmed; however, this may be due, in part, to smaller sample sizes in the age 25 Dysregulated group as compared with the Other clusters group at age 25 years. For example, some correlations in the Dysregulated group are of the same magnitude as those in the Other cluster group (e.g. binge eating $r = .11$), but the correlations are not significant.

Genetic and environmental associations

Bivariate models—Bivariate models indicated genetic overlap between most bulimic behaviours and AUD symptoms at age 17 years, but only for two associations at age 25 years (data not shown). Genetic correlations at age 17 years ranged from .24 to .44 for all MEBS variables examined; however, the genetic correlation between DSM BN symptoms and AUD symptoms was not significant. In contrast, at age 25 years, the only significant genetic correlations were between MEBS total score and AUD symptoms (.20; CIs: .03, .36) and DSM BN symptoms and AUD symptoms (.31; CIs: .09, .54). The nonshared environmental correlations were not significant for any of the BN behaviours examined at either time point.

Moderator models—The Pearson correlations described previously were also used to inform the twin analyses. Specifically, twin analyses were only conducted if there were significant associations between bulimic behaviours and AUD symptoms among either the Dysregulated or Other clusters. Thus, significant associations were examined between AUD symptoms and MEBS total score, body dissatisfaction, binge eating, compensatory

³Because of the nonindependence of twin data, phenotypic correlations were re-calculated after randomly selecting one twin from each pair to ensure that correlations were not inflated by twin similarity. Importantly, the correlations did not significantly differ (all $p_s > .19$) from those calculated using the twin pairs (data not shown).

behaviours and DSM BN symptoms at age 17 years, and AUD symptoms and MEBS total score, binge eating, compensatory behaviours and DSM BN symptoms at age 25 years. Associations between AUD symptoms and Weight Preoccupation at both time points and Body Dissatisfaction at age 25 years were not examined because Pearson correlations were not significant for either cluster group at these time points. Note that clusters were coded dichotomously as 'Other clusters' (coded 0) and Dysregulated cluster (coded 1) in all of the models.

Fit statistics from the moderator models are included in Table 3. Initially, a full ACE $G \times E$ in the presence of r_{GE} moderator model was examined to determine whether cluster membership moderates the genetic and environmental influences underlying the association between bulimic behaviours (e.g. binge eating) and AUD symptoms. Subsequently, an AE moderator submodel was examined (with C estimates constrained to zero). In all cases, the AE model resulted in a superior fit compared with the ACE fully unconstrained model as indicated by lower AICs and a non-significant change in chi-square.

Table 4 includes the unstandardized estimates of genetic and nonshared environmental variance that are unique to AUD symptoms (a_{AUD} and e_{AUD}), common to both bulimic behaviours and AUD symptoms (a_C and e_C), and unique to bulimic behaviours (a_{BUL} and e_{BUL} ; after accounting for the overlap with AUD symptoms) for the AE models. In addition, the moderation effects (i.e. βa_C , βa_{BUL} , βe_C and βe_{BUL}) are included in the table, which represent changes in the common and unique genetic and nonshared environmental variance on the covariance between bulimic behaviours and AUD symptoms and the variance in bulimic behaviours with different levels of the moderator (i.e. cluster membership). It is important to note that CIs for some parameter estimates overlapped with zero (Table 4); thus, the point estimates for these parameters are nonsignificant and not meaningful. As stated previously, submodels were not fit in these cases, as the remaining sources of variance would have absorbed the dropped effects and CIs for other estimates in the model would be artificially narrowed (Sullivan & Eaves, 2002). Instead, we kept the nonsignificant parameters in the model, and indicated significant parameter estimates via bolded text in the tables.

Age 17 models: Both bulimic behaviours and AUD symptoms were heritable in all models. Among the bulimic behaviours examined, heritability estimates were higher for body dissatisfaction and MEBS total score [i.e. 55% (95% CIs: 40%, 72%) and 50% (CIs: 35%, 67%), respectively] compared with binge eating, compensatory behaviour and DSM BN symptoms [i.e. 31% (CIs: 16%, 48%), 8% (CIs: 0, 25%) and 36% (CIs: 19%, 52%)]. In all models, the heritability estimate for AUD symptoms was 56% (CIs: 45%, 67%). The remaining variance for each of these phenotypes was due to nonshared environmental effects.

With the exception of associations with DSM BN symptoms, all of the moderator models indicated that there was small-to-moderate common genetic overlap ($a_C = .17-.27$), but no common nonshared environmental overlap ($e_C = -.03$ to $.06$) between the continuous measures of bulimic behaviours (i.e. MEBS total score, body dissatisfaction, weight preoccupation, binge eating and compensatory behaviours) and AUD symptoms. Although

these estimates provide information about the overlap in genetic effects underlying the association between bulimic behaviours and AUD symptoms, the a_C estimates are not directly comparable to the genetic correlations used in three of the extant studies examining these associations (Baker et al., 2010; Mitchell et al., 2010; Munn-Chernoff et al., 2013; Slane et al., 2012). Therefore, genetic correlations computed from the bivariate models allow for comparisons with previous research. As stated earlier, age 17 cross-sectional genetic correlations ranged from .24 to .44, indicating that between 6 and 19% of the variance in the association between bulimic behaviours and AUD symptoms is accounted for by common genetic factors.

Despite this genetic overlap, the estimates of interest [i.e. common genetic (β_{aC}) and common nonshared environmental (β_{eC}) moderation effects] were all small and nonsignificant (i.e. CIs overlapped with zero), suggesting that the magnitude of the genetic and nonshared environmental effects does not differ by cluster membership. Taken together, these findings strongly suggest that the factors that underlie the association between bulimic behaviours and AUD symptoms do not differ across the Dysregulated versus Other clusters.

In general, unique genetic (β_{aBUL}) and nonshared environmental (β_{eBUL}) moderation effects on bulimic behaviours alone also were not significant, with a few exceptions. The unique genetic moderation effect was significant for compensatory behaviours ($\beta_{aBUL} = .63$), suggesting that genetic influences on this phenotype are the strongest in the Dysregulated cluster. In order to interpret this level of increase, the genetic effects were standardized to show differences in heritability across the clusters. These standardized estimates indicate that the genetic influences on compensatory behaviours increase from 8% to 48% moving from the Other clusters to the Dysregulated cluster. Further, the unique nonshared environmental moderation coefficient was significant for MEBS total score and DSM BN symptoms, suggesting that there are small-to-moderate changes ($\beta_{eBUL} = .28$ and $.57$, respectively) in nonshared environmental influences on these phenotypes with levels of the moderator. Standardized estimates indicated that the nonshared environmental effects on these bulimic behaviours increase in magnitude across the Other clusters to the Dysregulated cluster from 40% to 48% for MEBS total score and from 50% to 82% for DSM BN symptoms. The influence of the change in magnitude of these unique effects is not clear; however, findings provide initial evidence that personality traits may influence the expression of these phenotypes.

Age 25 models: At age 25 years, heritability estimates for bulimic behaviours were somewhat larger than those obtained at age 17 years. Specifically, estimates were 55% (CIs: 42%, 71%) for MEBS total score, 38% (CIs: 26%, 52%) for binge eating, 45% (CIs: 31%, 58%) for compensatory behaviour and 28% (CIs: 20%, 41%) for DSM BN symptoms. Heritability estimates for AUD symptoms was lower at age 25 years (35%; CIs: 24%, 46%) as compared with age 17 years (i.e. 56%). As before, the remaining variance for all phenotypes was accounted for by nonshared environmental effects.

In contrast to age 17 findings, there were no significant estimates of common genetic factors underlying bulimic behaviours and AUD symptoms at age 25 years. Age 25 cross-sectional a_C estimates ranged from .05 to .14; however, these were also all non-significant, suggesting

that none of the variance in the association between bulimic behaviours and AUD symptoms is due to common genetic effects. However, common nonshared environmental factors were indicated for associations between binge eating and AUD symptoms ($e_C = .20$).

Similar to the age 17 findings, the common genetic (β_{aC}) and common nonshared environmental (β_{eC}) moderation estimates of interest were all small and nonsignificant, indicating that personality cluster membership does not moderate common genetic or environmental associations between these phenotypes cross-sectionally at age 25 years. And most of the unique genetic and nonshared environmental moderation effects on bulimic behaviours were not significant, although as before, there were some exceptions. There were no significant unique genetic moderation coefficients for bulimic behaviours at age 25 years. However, there were significant unique nonshared environmental moderation effects on weight preoccupation ($\beta_{eBUL} = .19$), compensatory behaviour ($\beta_{eBUL} = .40$) and DSM BN symptoms ($\beta_{eBUL} = .16$), suggesting increases in the unique nonshared environmental variance on these phenotypes across cluster. This suggests that the unique nonshared environmental effects on these bulimic behaviours increase in magnitude across the Other clusters to the Dysregulated cluster from 42% to 46% for weight preoccupation, from 40% to 56% for compensatory behaviour and from 64% to 67% for DSM BN symptoms.

Discussion

The present study is the first to examine whether genetic factors underlying the association between bulimic behaviours and AUD symptoms are the strongest in the Dysregulated personality cluster. Twin model findings indicated that although common genetic factors underlie some associations between bulimic behaviours and AUD symptoms, these common genetic effects did not differ between the Dysregulated and Other clusters. Therefore, personality heterogeneity may not be what is accounting for discrepancies in twin studies examining factors underlying associations between BN and AUDs.

Several of the twin models at age 17 years indicated that common genetic effects underlie associations between bulimic behaviours and AUD symptoms (see a_C parameter estimates in Table 4). This supports findings from four studies (Baker et al., 2010; Munn-Chernoff et al., 2013; Slane et al., 2012; Trace et al., 2013), suggesting that there are common genetic influences underlying this covariance. Estimates across studies have been fairly similar in magnitude. That is, previous studies indicated significant genetic correlations between bulimic behaviours and AUD symptoms ranging from .23 to .61 (Baker et al., 2010; Munn-Chernoff et al., 2013; Slane et al., 2012; Trace et al., 2013). This suggests that between 5% and 37% of the variance in the association between bulimic behaviours and AUD symptoms is accounted for by common genetic factors. In the present study, genetic correlations were within the range of those identified previously. It is unclear why findings indicated genetic overlap between bulimic behaviours and AUD symptoms at age 17 years, but not at age 25 years. Of note, the present study found a lower heritability (and higher nonshared environmental effect) of AUD symptoms at age 25 years as compared with age 17 years. This suggests that nonshared environmental factors are more influential on AUD symptoms in young adulthood, and this may have attenuated the degree of overlap in and genetic influences on bulimic behaviours and AUD symptoms at age 25 years. Alternatively, there

was a decrease in some bulimic behaviours (i.e. binge eating and compensatory behaviours) at age 25 years, which may have resulted in decreased variability in these core BN behaviours in the sample and attenuated phenotypic and genetic associations. Future research is needed to explore these possibilities and replicate lower heritability in young adult samples.

In general, common nonshared environmental effects were not significant, corroborating most previous research (Baker et al., 2010; Kendler et al., 1995; Mitchell et al., 2010; Munn-Chernoff et al., 2013; Slane et al., 2012). These findings suggest that there is little-to-no overlap in the nonshared environmental effects that influence the association between bulimic behaviours and AUD symptoms. The only exception was a significant common nonshared environmental effect underlying associations between age 25 binge eating with age 25 AUD symptoms. This suggests that for this association, there are nonshared environmental effects (i.e. factors that act to make twins within a pair different) that similarly influence the association between binge eating and AUD symptoms. It is interesting that the bulimic behaviour that was significant was a symptom that maps on most closely to BN (i.e. binge eating). Yet, common nonshared environmental effects were not indicated for associations between compensatory behaviour and AUD symptoms or DSM BN symptoms and AUD symptoms; however, there may not have been enough variability of these symptoms in the sample.

The result of primary interest in the present study did not follow the main study hypothesis. That is, although several models indicated common genetic influences underlying the association between bulimic behaviours and AUD symptoms, these factors were not the strongest for the Dysregulated cluster compared with the Other clusters. In fact, findings indicated no change in the magnitude of shared genetic effects with changes in cluster membership. Results suggest that even though personality heterogeneity does exist among women with BN, there may not be different etiologic effects underlying the association between these phenotypes, based on personality. This was true for DSM BN symptoms as well as the continuous measures of bulimic behaviours examined.

Despite the novelty of the present findings, some limitations must be noted. First, although the present study provided information regarding associations among both continuous measures of bulimic behaviours and DSM BN symptom counts, the sample size was small for moderation analyses, and there may have not been enough variability in DSM BN symptom counts to find significant associations with this phenotype. Indeed, only about 6.5% of the sample at both time points had one or more BN symptom. Importantly, however, although findings with DSM BN symptoms were less consistent across analyses, there were some significant results involving these symptoms. Further, despite this low variability, the examination of BN symptom counts and continuous measures of bulimic behaviours was important as it allowed for the determination of whether associations with BN are driven by particular symptoms (e.g. binge eating) or symptoms of the full syndrome. Importantly, the variability in the bulimic behaviours examined using the MEBS was on par with those of previous studies (e.g. Slane et al., 2012).

Second, the present study used AUD symptom counts, rather than a continuous measure of AUDs. It may be that more significant associations would have been identified had a continuous measure been used as variability would be increased and a wider range of problems would be present in the sample. Indeed, although two of the previous studies that indicated that common genetic factors underlie the association between BN and AUDs also used a measure of AUD diagnoses (Baker et al., 2010; Trace et al., 2013), the third study finding a shared aetiology between these disorders used a continuous measure of alcohol use (Slane et al., 2012). Thus, the present study may have been limited in its ability to detect shared genetic transmission between BN and AUDs (as well as significant cluster moderation) given that only a measure of diagnostic symptoms of AUDs was available.

This study was the first to examine whether cluster membership influences the common genetic effects on the covariance between BN and AUDs. Additional research is needed to determine whether findings in the present study are replicable. Given the deleterious effects of the comorbidity between BN and AUDs (Dansky et al., 2000; Duncan et al., 2006; Keel et al., 1999), future studies should continue to determine what factors might be influencing the co-occurrence of these disorders. Future longitudinal studies examining the development of BN should include measures of both AUD and personality traits, as this could aid in identifying prospective risk factors.

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Figure 1.

G×E in the Presence of r_{GE} Model. a_{AUD} = genetic effects that are specific to the first phenotype (i.e. unique to AUD); a_C = genetic effects that are common between the phenotypes; a_{BUL} = genetic effects that are unique to the second phenotype (i.e. bulimic behaviours); C = coefficient representing moderation between the common path and the moderator; BUL = coefficient representing moderation between the unique path and the moderator; cluster = moderator value [coded as Other clusters (0) and Dysregulated (1)]. The unstandardized common genetic influence on the association between bulimic behaviours and AUD symptoms is indicated by the equation $a_C + C * Cluster$. The shared and nonshared environmental components are not shown.

Table 1

Means and standard deviations of bulimic behaviours, DSM BN symptoms and AUD symptoms by cluster at ages 17 and 25 years

	Age 17 years					Age 25 years				
	Dysregulated		Other clusters			Dysregulated		Other clusters		
	Mean (SD)		Mean (SD)	t	p	Mean (SD)		Mean (SD)	t	p
MEBS total score	7.93 (5.85)		6.00 (5.43)	3.10	.002	8.63 (5.81)		5.95 (5.16)	6.83	<.001
Body dissatisfaction	2.47 (2.18)		1.99 (2.09)	2.02	.04	3.24 (2.27)		2.19 (2.11)	6.78	<.001
Weight preoccupation	2.88 (2.42)		2.34 (2.31)	2.06	.04	3.18 (2.38)		2.33 (2.18)	5.27	<.001
Binge eating	1.66 (1.61)		0.91 (1.28)	4.28	<.001	1.34 (1.68)		0.83 (1.25)	4.70	<.001
Compensatory behaviour	0.33 (0.68)		0.25 (0.68)	1.05	.29	0.30 (0.70)		0.20 (0.61)	2.07	.04
BN symptoms	0.27 (0.89)		0.12 (0.60)	2.67	.008	0.13 (0.66)		0.17 (0.86)	-0.64	.52
Alcohol composite	0.95 (2.08)		0.31 (1.04)	4.85	<.001	0.98 (1.77)		0.55 (1.31)	3.78	<.001

Note: Raw scores for binge eating, compensatory behaviours, bulimia nervosa symptoms and alcohol composite shown for descriptive purposes.

MEBS = Minnesota Eating Behavior Survey; BN = bulimia nervosa; DSM = Diagnostic and Statistical Manual of Mental Disorders; AUD = alcohol use disorder.

Table 2

Phenotypic correlations within the Dysregulated cluster and Other clusters at age 17 and 25 years

	Age 17 AUD symptoms		Age 25 AUD symptoms	
	Dysregulated (<i>n</i> = 92–262)	Other clusters (<i>n</i> = 509–968)	Dysregulated (<i>n</i> = 313–432)	Other clusters (<i>n</i> = 577–742)
Bulimic behaviours				
MEBS total score	.25★	.14★★	.06	.09★
Body dissatisfaction	.24★	.12★★	–.01	.05
Weight preoccupation	.12	.08	.03	.07
Binge eating	.21★	.15★★	.11	.15★★
Compensatory behaviour	.29★★	.13★★	.09	.11★★
DSM BN symptoms	.16★	.05	.15★★	.08★

Note: Sample sizes vary because of missing data. Correlations between Dysregulated and Other Clusters did not differ significantly at either time point.

AUD = Alcohol Use Disorder; MEBS = Minnesota Eating Behavior Survey; DSM BN = Diagnostic and Statistical Manual Bulimia Nervosa.

★
p < .05;

★★
p < .01.

Table 3

Fit Statistics for GxE in the presence of r_{GE} models of the association between bulimic behaviours and alcohol use disorder symptoms by cluster membership

Model	-2lnL	df	χ^2 (df)	p	AIC
Cross-sectional models at age 17 years					
MEBS total score					
ACE moderation	4589.41	1690	—	—	1209.41
AE moderation	4589.41	1695	0.00 (5)	.99	1199.41
Body dissatisfaction					
ACE moderation	4593.37	1690	—	—	1213.37
AE moderation	4593.37	1695	0.00 (5)	.99	1203.37
Binge eating					
ACE moderation	4620.183	1690	—	—	1240.18
AE moderation	4624.710	1695	4.53 (5)	.48	1234.71
Compensatory behaviour					
ACE moderation	4640.210	1690	—	—	1260.21
AE moderation	4641.635	1695	1.43 (5)	.92	1251.64
DSM BN symptoms					
ACE moderation	6293.616	2277	—	—	1739.62
AE moderation	6293.616	2282	0.00 (5)	.99	1729.62
Cross-sectional models at age 25 years					
MEBS total score					
ACE moderation	5115.360	1878	—	—	1359.36
AE moderation	5117.823	1883	2.46 (5)	.78	1351.82
Binge eating					
ACE moderation	5160.696	1878	—	—	1404.70
AE moderation	5161.047	1883	0.35 (5)	.99	1395.05
Compensatory behaviour					
ACE moderation	5179.732	1878	—	—	1423.73
AE moderation	5184.600	1883	4.87 (5)	.43	1418.60
DSM BN symptoms					

Model	-2lnL	df	χ^2 (df)	p	AIC
ACE moderation	5950.401	2129	—	—	1692.40
AE moderation	5950.404	2134	0.00 (5)	.99	1682.40

Note: The best-fitting model is indicated by bold type.

DSM BN = Diagnostic and Statistical Manual bulimia nervosa; MEBS = Minnesota Eating Behavior Survey. -2lnL = -2 times log likelihood of data; df = degrees of freedom; AIC = Akaike's information criterion; χ^2 = compares the ACE fully unconstrained model and AE submodel.

Table 4

Unstandardized parameter estimates from the best-fitting G×E in the presence of r_{GE} moderator models

Model	a_{AUD}	a_C	a_{BUL}	β_{aC}	β_{aBUL}	e_{AUD}	e_C	e_{BUL}	β_{eC}	β_{eBUL}
Cross-sectional models at age 17 years										
MEBS total score										
AE	.75 (.67, .82)	.27 (.10, .43)	.71 (.59, .82)	-.01 (-.36, .34)	-.26 (-.66, .15)	.66 (.61, .71)	-.03 (-.14, .08)	.63 (.56, .72)	.18 (-.13, .49)	.28 (.03, .50)
Body dissatisfaction										
AE	.75 (.68, .82)	.24 (.08, .41)	.74 (.63, .85)	-.08 (-.41, .24)	-.18 (-.59, .18)	.66 (.61, .71)	-.03 (-.14, .08)	.62 (.55, .70)	.22 (-.07, .50)	.23 (-.01, .46)
Binge eating										
AE	.74 (.67, .82)	.25 (.09, .41)	.56 (.40, .69)	.16 (-.24, .54)	-.13 (-.96, .59)	.66 (.62, .71)	.05 (-.08, .18)	.76 (.68, .86)	-.10 (-.43, .28)	.22 (-.25, .51)
Compensatory behaviours										
AE	.74 (.67, .81)	.17 (.00, .35)	.28 (-.22, .50)	.23 (-.13, .57)	.63 (.04, 1.17)	.66 (.62, .71)	.06 (-.10, .21)	.92 (.83, 1.01)	-.11 (-.38, .21)	-.44 (-.63, .04)
DSM BN symptoms										
AE	.75 (.67, .82)	.04 (-.08, .17)	.60 (.44, .72)	.10 (-.14, .35)	-.29 (-.60, .08)	.66 (.61, .71)	.03 (-.07, .13)	.71 (.63, .81)	.10 (-.15, .34)	.57 (.39, .73)
Cross-sectional models at age 25 years										
MEBS total score										
AE	.58 (.49, .67)	.14 (-.04, .31)	.74 (.65, .84)	.06 (-.24, .37)	.09 (-.11, .27)	.78 (.73, .84)	.07 (-.03, .17)	.58 (.51, .65)	-.10 (-.30, .11)	.10 (-.05, .25)
Binge eating										
AE	.58 (.49, .67)	.06 (-.11, .23)	.62 (.51, .72)	.10 (-.18, .38)	.16 (-.06, .36)	.78 (.73, .84)	.20 (.09, .31)	.67 (.60, .75)	-.15 (-.35, .06)	.11 (-.05, .27)
Compensatory behaviours										
AE	.58 (.49, .67)	.13 (-.05, .30)	.67 (.56, .76)	.02 (-.31, .37)	-.25 (-1.25, .04)	.78 (.73, .84)	.09 (-.01, .20)	.63 (.56, .71)	-.09 (-.34, .15)	.40 (.23, .56)
DSM BN symptoms										
AE	.59 (.50, .67)	.05 (-.12, .21)	.53 (.40, .64)	.23 (-.01, .48)	-.16 (-.56, .10)	.78 (.73, .84)	.07 (-.05, .18)	.80 (.73, .88)	-.11 (-.30, .08)	.16 (.03, .29)

Note: Alcohol use disorder symptoms were assessed using DSM-III-R criteria at age 17 years and DSM-IV criteria at age 25 years. DSM BN symptoms were assessed using DSM-IV criteria at both time points. Ninety-five percent confidence intervals are in parentheses. Significant effects are indicated by boldface.

MEBS = Minnesota Eating Behaviors Survey; BN = bulimia nervosa, AUD = alcohol use disorder; DSM = Diagnostic and Statistical Manual of Mental Disorders; a_{AUD} = AUD symptoms unique genetic path; a_C = common genetic path; a_{BUL} = bulimic behaviours unique genetic path; β_{aC} = common genetic moderation effect; β_{aBUL} = unique genetic moderation effect; e_{AUD} = AUD symptoms unique nonshared environmental path; e_C = common nonshared environmental path; e_{BUL} = bulimic behaviours unique nonshared environmental path; β_{eC} = common nonshared moderation effect; β_{eBUL} = unique nonshared moderation effect.