

Bulimic Behaviors and Alcohol Use: Shared Genetic Influences

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Abstract Bulimic behaviors are frequently associated with alcohol use disorders. However, extant family and twin study findings have been inconsistent with regard to whether these behaviors share etiologic influences. A sample of 292 young adult, female twins was used to examine genetic and environmental factors underlying the association between binge eating and compensatory behaviors (e.g., vomiting) and alcohol use. Binge eating and compensatory behaviors were assessed using the Minnesota Eating Behavior Survey. Alcohol use was measured using the Alcohol Use Disorders Identification Test. Univariate models indicated that the heritability of binge eating, compensatory behaviors, and alcohol use was 41, 28, and 78%, respectively, with the remaining variance due to nonshared environmental effects. Bivariate models indicated that there was a moderate-to-large degree of overlap (genetic correlation = 0.31–0.61) in additive genetic factors between alcohol use and binge eating and compensatory behaviors, and no overlap in environmental effects. Findings suggest that these phenotypes co-aggregate in families and that similar genes or heritable traits may be contributing to their co-occurrence.

Keywords Bulimic behaviors · Alcohol use · Genetics · Environment

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Introduction

Individuals with eating disorders and substance abuse have the highest risk of premature death of the major mental illnesses (Harris and Barraclough 1998). Within eating disorders, alcohol use disorder severity is one of the most significant predictors of mortality among individuals with anorexia nervosa (AN; Keel et al. 2003), and a history of a substance use disorder is associated with a worse outcome for women with bulimia nervosa (BN; Keel et al. 1999). The co-occurrence of eating disorders and alcohol use disorders is also associated with higher rates of attempted suicide (Duncan et al. 2006) and an increased likelihood of major depressive disorder and drug or tobacco dependence (Duncan et al. 2006). Given the deleterious effects of their co-occurrence, it is important to understand the factors underlying the comorbidity of eating disorders and alcohol use disorders.

Among women with eating disorders, alcohol use disorders are more often comorbid with BN or AN, binge-purge type, than AN, restricting type, (Holderness et al. 1994). Therefore, much of the research focus has been on associations between alcohol use disorders and BN, or more generally, bulimic behaviors (e.g., bingeing and purging). Three family studies that have examined associations between BN and alcohol use disorders have found that these disorders co-occur in families, with higher rates of alcohol disorders in BN probands (Bulik 1987; Kasset et al. 1989; Lilenfeld et al. 1997). However, Lilenfeld et al. (1997) found that only family members of BN probands with comorbid alcohol use disorders had higher rates of alcohol use disorders. This suggests possible independent transmission of the disorders, as alcohol use disorders may be transmitted within families in which alcohol use problems are present, regardless of whether BN is present as well.

Twin studies have begun to examine the issue of etiologic overlap to clarify whether familial aggregation or independent transmission is present, as well as to determine whether relationships are due to common genetic and/or environmental factors. Extant twin data suggests that AN and BN are significantly heritable (>50%), with nonshared environmental factors (i.e., factors unique to co-twins that contribute to their behavioral differences) accounting for the remaining variance in these disorders (Bulik et al. 1998, 2006; Klump et al. 2001; Mazzeo et al. 2010). Genetic and nonshared environmental effects have also been found to influence bulimic behaviors, including binge eating, defined as eating a large amount of food in a short period of time with a feeling of lack of control over the binges, and compensatory behaviors, which are behaviors aimed at weight loss that include self-induced vomiting, laxative use, and diuretic use. Yet, findings for bulimic behaviors are somewhat inconsistent with regard to the magnitude of these factors. With few exceptions (i.e., heritability 8–17%; see Wade et al. 2008), several studies indicated that the heritability of binge eating exceeds 40% (Bulik et al. 1998; Sullivan et al. 1998; Bulik et al. 2003; Reichborn-Kjennerud et al. 2004; Klump et al. 2002) and compensatory behavior ranges from 35 to 70%, depending on whether several compensatory behaviors are examined together (heritability = 50%) or specific types of behaviors are examined separately (e.g., laxative use only; heritability = 43%) are examined (Klump et al. 2000; Mazzeo et al. 2010; Sullivan et al. 1998). Nonetheless, in general, each of these studies indicated that these behaviors are heritable with the remaining variance due to nonshared environmental factors.

For alcohol use disorders, large-scale twin studies indicate heritabilities of over 60% in women (Heath et al. 1997; Whitfield et al. 2004) with nonshared environmental factors accounting for the remaining variance. Alcohol use disorder symptoms, such as symptom counts that tally the number of abuse and/or dependence symptoms, have also been found to be heritable with estimates exceeding 50% (Kendler et al. 1992, 1994; Prescott et al. 1999). Overall, findings across eating disorder and alcohol use disorder studies suggest that if familial aggregation between these phenotypes is present, it may be due to significant genetic and/or nonshared environmental factors.

Twin studies are able to investigate this possibility by examining the extent to which phenotypic associations between two disorders are due to genetic and/or environmental factors. Using this method, Kendler et al. (1995) only found evidence for a small genetic overlap (i.e., 6%) between BN and “problem drinking” (i.e., “having had or having been considered by others as having a significant drinking problem that is not limited to single isolated incidents”) in a large sample of middle-aged twins (mean age = 30.1 years, SD = 7.6). Indeed, BN was found to

predominately load on a separate genetic factor from problem drinking. Common nonshared environmental influences were similarly small for both disorders. However, it must be noted that Kendler et al.’s (1995) study included several psychiatric disorders which were examined together in the same model to see if a common genetic and environmental factor or several independent genetic and environmental factors influenced the phenotypes. Thus, findings from studies that only examined BN and alcohol use disorders may differ somewhat due to different methodologies and information included in the twin models.

Indeed, recent bivariate studies examining associations between BN and alcohol use disorders only have provided stronger support for shared genetic etiologies, where genetic correlations (i.e., the degree of overlap in genetic factors influencing two phenotypes) ranged from 0.53 to 1.0 (Baker et al. 2010; Mitchell et al. 2010) and nonshared environmental correlations were either small (i.e., -0.03 ; Baker et al. 2010) and/or non-significant (i.e., confidence intervals overlapped with zero; Mitchell et al. 2010). These findings suggest that the association between BN and alcohol use disorders may be primarily due to shared genetic factors. Nonetheless, the above cited studies are few in number ($N = 3$), and confidence intervals have been rather broad. Additional research therefore is needed to confirm genetic associations and provide additional point estimates of these shared effects.

Consequently, the purpose of the current study was to investigate genetic and environmental influences on associations between bulimic behaviors and problematic alcohol use in adult female twins. We examined symptoms of BN (i.e., binge eating and the use of compensatory behaviors), which are much more prevalent than BN diagnoses (Crowther et al. 2008; Hudson et al. 2007). These symptoms frequently predate the development of BN (Patton et al. 1999) and have been found to be associated with increased levels of depression and anxiety (Fitzgibbon et al. 1998; Johnson et al. 2002; Mond et al. 2006) in adolescents and adults. Similarly, alcohol abuse or problem drinking is much more prevalent in the population than alcohol dependence (Hasin et al. 2007) and is strongly associated with other forms of psychopathology, including depression (Marmorstein 2009). Thus, in addition to increasing statistical power in the study, symptoms of BN and problematic alcohol use are important phenotypes to examine in their own right.

Methods

Participants

Participants were drawn from the Adult Twin Study of Behavioral Adjustment and Development from the

Michigan State University Twin Registry (MSUTR; Klump and Burt 2006), a population-based twin registry focused on understanding risk factors for internalizing and externalizing disorders across the lifespan. Although all MSUTR participants are now recruited through birth records, participants in the current study were also recruited from posted flyers and newspaper advertisements. The participants completed in-person assessments privately in individual computer rooms in the laboratory. Regardless of the recruitment methods used, the twins in the current study have been found to be broadly representative of women in Michigan in terms of racial identification (Culbert et al. 2008).

The current study included 292 female twins (164 monozygotic [MZ]; 128 dizygotic [DZ]) between the ages of 18 and 29 years (mean age = 20.92 years; SD = 2.47). Participants identified as Caucasian (83%), African American (11%), Hispanic (2%), Asian American (1%), or “Other” (3%) ethnicity. Socioeconomic status, measured using the four-factor index of social status (Hollingshead 1975), indicated that the majority of twins were in the middle-to-upper socioeconomic classes (i.e., Level 1 = 28.1%, Level 2 = 44.2%, Level 3 = 18.8%, Level 4 = 4.5%, and Level 5 = 2.4%). This breakdown is broadly representative of the socioeconomic status of similar aged adults in the State of Michigan (www.census.gov 2008).

Zygoty determination

Zygoty was determined using a physical similarity questionnaire shown to be over 95% accurate (Plomin et al. 2008). Decisions about indeterminate zygoty were made by the project’s principal investigators (KLK and SAB) who reviewed questionnaire ratings and waist-up photographs of the twins.

Measures

Bulimic behaviors

The 7-item binge eating and 6-item compensatory behavior subscales from the Minnesota eating behavior survey (MEBS; von Ranson et al. 2005)¹ were used to assess binge eating and the use of compensatory behaviors. These subscales assess thoughts about overeating or the tendency to

binge eat as well several types of compensatory behaviors such as self-induced vomiting and laxative use for weight loss. Items are endorsed as either true (scored as 1) or false (scored as 0), with a sum score for each subscale. The internal consistency of the binge eating and compensatory behavior scales has been adequate in women in past research (von Ranson et al. 2005) as well as in the current sample ($\alpha = 0.75$ and 0.65, respectively). Discriminant validity also has been previously established through the ability of these subscales to differentiate between normal control participants and women with eating disorders (von Ranson et al. 2005).

Importantly, the present sample exhibited a range of pathology on these measures. Specifically, binge eating scores ranged from 0 to 7 and compensatory behavior scores ranged from 0 to 6, with mean scores of 1.56 (SD = 1.76) and 0.51 (SD = 0.97), respectively. Corresponding means for these scales among clinical samples with eating disorders are 2.53 for the binge eating scale and 1.53 for the compensatory behavior scale (von Ranson et al. 2005). Notably, 22% ($n = 65/290$) of women scored a 3 or higher on the binge eating scale, and 31% ($n = 89/290$) endorsed engaging in one or more compensatory behaviors. This suggests that approximately 1/3 of the sample endorses in these behaviors at or above the average level of endorsement for clinical samples (von Ranson et al. 2005).

Problematic alcohol use

The 10-item alcohol use disorders identification test (AUDIT; Saunders et al. 1993) was used to assess harmful and hazardous amounts of alcohol use. The AUDIT items assess alcohol consumption (i.e., “How often do you have a drink containing alcohol”), drinking behavior (i.e., “How often during the last year have you found that you were not able to stop drinking once you had started”), adverse reactions (i.e., “How often during the last year have you had a feeling of guilt or remorse after drinking”), and problems related to alcohol use (i.e., “Have you or someone else been hurt as a result of your drinking”). Total scores range from 0 (non-hazardous consumption) to 40 (the highest severity of hazardous consumption). A score of 8 or higher indicates a strong likelihood that the person is engaging in harmful alcohol consumption. Twenty-two percent ($n = 62$) of women in the present sample had scores of 8 or higher on the AUDIT.

In a study of nearly 1,000 male and female undergraduates, the AUDIT displayed internal consistency reliability of 0.80 (Fleming et al. 1991). Discriminant validity was established by comparing male and female non-drinkers to individuals with alcohol dependence, with 0.5% of the non-drinkers and 99% of the individuals with alcohol dependence scoring over 8 on the AUDIT (Saunders et al. 1993). The AUDIT exhibited good internal consistency in this

¹ The Minnesota eating behavior survey (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.

sample ($\alpha = 0.82$) and scores ranged from 0 to 24 with a mean of 4.48 ($SD = 4.73$).

Body mass index

Body mass index (BMI; weight [kg]/height² [m]) was calculated using height and weight measured using a wall-mounted ruler and digital scale, respectively. Participants were asked to remove their shoes and heavy outerwear before the measurements.

Statistical analyses

Data preparation and descriptive statistics

Log transformations ($\log_{10} X + 1$) were performed for the body mass index, compensatory behavior, and AUDIT scores in order to account for positive skew. Although binge eating was slightly positively skewed, the skewness and kurtosis were within -2 and 2 , suggesting that a transformation was not necessary prior to analysis (see Lewis-Beck et al. 2004). BMI was regressed out of binge eating, compensatory behavior, and AUDIT scores prior to analysis in order to determine whether influences underlying associations between bulimic behaviors and problematic alcohol use were independent of BMI.²

Genetic and environmental influences on bulimic behaviors and problematic alcohol use

Twin correlations Two types of twin correlations were calculated to provide initial indications of genetic and environmental influences on bulimic behaviors and problematic alcohol use separately, as well as on the association between these phenotypes. Both types of correlations (i.e., twin intraclass and cross-twin, cross trait) were calculated using the double-entry method to remove the variance associated with the ordering of siblings within a pair. Twin intraclass correlations (e.g., Twin 1's binge eating with Twin 2's binge eating) were first calculated for binge eating, compensatory behavior, and problematic alcohol use. These correlations were used to provide indications of genetic and environmental factors on each phenotype individually. Cross-twin, cross-trait correlations (e.g., Twin 1's binge eating with Twin 2's problematic alcohol use) were then calculated to provide indications of genetic and environmental influences on phenotypic associations between bulimic behaviors and problematic alcohol use. For both types of correlations, greater MZ than DZ twin correlations suggest that additive genetic effects (i.e., the

effect of individual genes summed over loci that increase twin similarity relative to the amount of genes shared) influence the trait or phenotypic association in question. By contrast, similar MZ and DZ twin correlations suggest that shared environmental (i.e., the part of the environment common to siblings that acts to make them similar to each other) factors are important. Finally, nonshared environmental (i.e., environmental factors, and measurement error, differentiating twins within a pair) influences are implicated if the MZ twin correlations are less than 1.00.

Univariate twin models Univariate twin models were fit to raw data using the maximum likelihood option in Mx (Neale et al. 2003) to examine additive genetic (A), shared environmental (C), and nonshared environmental (E) influences on binge eating, compensatory behaviors, and problematic alcohol use, separately. The raw data option in Mx considers missing data to be missing at random and can lead to less biased estimates than pairwise or list wise deletion (Little and Rubin 1987). When fitting models to raw data, their variances, covariances, and means are first freely estimated to get a baseline index of fit (minus twice the log-likelihood; $-2\ln L$). The $-2\ln L$ under this unrestricted baseline model is then subtracted from the $-2\ln L$ under more restrictive biometric models. This result is a likelihood-ratio Chi-square test of goodness of fit for the model (χ^2), which is then used to calculate the Akaike's information criterion (Akaike 1987), $AIC = \chi^2 - 2df$, the traditional fit index of behavioral genetics research. The AIC measures model fit relative to parsimony. Better fitting models have lower AIC values. In addition to the AIC, the χ^2 difference test can also be used as a measure of model fit. The χ^2 difference test examines changes in Chi-square values and degrees of freedom between nested models. If the Chi-square difference test is nonsignificant, then the most parsimonious (i.e., reduced) model is preferred. Using this approach, the full ACE model was compared to several submodels (i.e., AE, CE, and E) to ascertain whether a reduced model provided a better fit to the data.

Bivariate models Bivariate models (see Fig. 1) were then fit to raw data using Mx (Neale et al. 2003) to examine additive genetic, shared environmental, and nonshared environmental influences on *associations* between bulimic behaviors and problematic alcohol use. These influences were investigated through genetic and environmental correlations that quantify overlap in genetic and environmental factors between the phenotypes. The genetic (r_a), shared environmental (r_c), and nonshared environmental (r_e) correlations indicate the degree of overlap in genetic factors, shared environmental factors, and nonshared environmental factors, respectively, influencing each phenotype. Thus, these correlations estimate the extent to which a common

² Analyses using raw MEBS subscales and the AUDIT score did not differ from those with BMI regressed out.

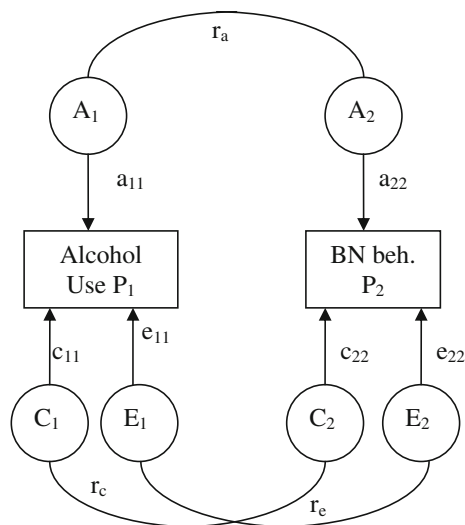


Fig. 1 Path diagram of the bivariate model examining bulimic behaviors and alcohol use. Variance estimates are comprised of additive genetic (A), shared environmental (C), and nonshared environmental (E) effects, including: (1) genetic and environmental influences on alcohol use (a_{11} , c_{11} , e_{11}); (2) genetic and environmental influences on bulimic behaviors (a_{22} , c_{22} , e_{22}); (3) genetic (r_a), shared environmental (r_c), and nonshared (r_e) environmental correlations that examine the degree of overlap in genetic and environmental factors between bulimic behaviors and alcohol use. Alcohol Use P_1 = Alcohol Use as Phenotype 1; BN beh. P_2 = Bulimic behaviors as Phenotype 2

set of genes and/or environmental factors contribute to phenotypic associations between two traits/behaviors.

Similar to the univariate models, we initially estimated variances, covariances, and means of the bulimic behaviors

and problematic alcohol use to determine a baseline fit ($-2\ln L$) of the data. The $-2\ln L$ under this unrestricted baseline model was then compared with $-2\ln L$ under more restrictive biometric models. This comparison provides a likelihood-ratio Chi-square test of goodness of fit for the model, which is then converted to the AIC. The AIC and changes in χ^2 were used to examine model fit. Additional submodels, based on the best-fitting univariate models, were then fit to the data to examine whether constraining the genetic, shared, or nonshared environmental correlations to 0 would provide a better fit to the data. These models tested whether there is statistically significant overlap in genetic/environmental influences on bulimic behaviors and problematic alcohol use.

Results

Univariate models

Twin intraclass correlations suggested significant genetic, but no shared environmental, influences, on binge eating, compensatory behavior, and problematic alcohol use (see Table 1). That is, in all cases, MZ correlations were nearly double the DZ correlations. Additionally, nonshared environmental factors also appeared important for all three phenotypes, as the MZ twin correlations were less than 1.00.

Univariate twin models confirmed these impressions. The best fitting model for binge eating, compensatory behavior, and problematic alcohol use was the AE model

Table 1 Within-person, twin intraclass, and cross-twin, cross trait correlations

Variables	Within-person	MZ ($n = 77-80$ pairs)	DZ ($n = 51$ pairs)	Test of equality z^a	p
Twin intraclass correlations					
Binge eating	–	0.41** (0.28, 0.53)	0.21* (0.01, 0.38)	1.26	0.10
Compensatory behavior	–	0.32** (0.17, 0.45)	–0.01 (–0.20, 0.19)	1.83	0.03
Alcohol use	–	0.76** (0.69, 0.82)	0.40** (0.23, 0.55)	3.10	0.001
Cross-twin, cross-trait correlations					
Alcohol use					
Binge eating	0.18**	0.21** (0.05, 0.35)	0.07 (–0.12, 0.26)	1.08	0.14
Compensatory behavior	0.28**	0.31** (0.16, 0.45)	0.14 (–0.05, 0.33)	0.96	0.17

Note. Ninety-five percent confidence intervals are in parentheses. Sample size is decreased from the original sample due to a lower number of twins with available BMI data (MZ = 162; DZ = 111). Significant differences between MZ and DZ correlations are indicated by bold type

^a Test of significant differences between MZ and DZ correlations

* $p < 0.05$, ** $p < 0.01$

(see Table 2). This model had the lowest AIC and did not provide a significantly worse fit to the data than the full ACE model for each phenotype. Genetic factors were the highest for problematic alcohol use, with parameter estimates of 78% for genetic influences and 22% for nonshared environmental effects. Binge eating had the second highest heritability of 41%, with the remaining variance accounted for by nonshared environment. Compensatory behaviors were accounted for, in part, by genetic factors (i.e., 28%) with the remaining variance attributed to nonshared environmental effects (i.e., 72%).

Notably, the heritability estimate for compensatory behaviors is somewhat lower than what might be suggested by the intraclass twin correlations (see Table 1). We tested whether this was due to our modeling of compensatory behavior on a continuous rather than a categorical scale. When a univariate twin model is conducted using a categorical compensatory behavior variable (i.e., 0–6 with no transformations), the variance due to genetic effects (i.e., 0.23–0.38, depending on the constraints used in the model) fell within the range of the heritability estimate in the continuous univariate model (i.e., 0.28). Given the relative

Table 2 Univariate twin models examining genetic and environmental influences on binge eating, compensatory behavior and alcohol use

Model	Standardized parameter estimates			Test statistics				
	a ²	c ²	e ²	−2lnL (df)	χ ² (df) ^a	Δχ ² (df) ^b	p	AIC
Binge eating								
Baseline	–	–	–	749.13 (263)	–	–	–	–
ACE	0.41 (0.00, 0.56)	0.00 (0.00, 0.43)	0.59 (0.44, 0.78)	755.24 (269)	6.11 (6)	–	–	−5.89
AE	0.41 (0.23, 0.56)	–	0.59 (0.44, 0.77)	755.24 (270)	6.11 (7)	0.00 (1)	0.99	−7.89
CE	–	0.33 (0.17, 0.47)	0.67 (0.53, 0.83)	757.24 (270)	8.11 (7)	2.00 (1)	0.16	−5.89
E	–	–	1.0 (1.0, 1.0)	772.32 (271)	23.19 (8)	17.08 (2)	<0.01	7.19
Compensatory behavior								
Baseline	–	–	–	751.41 (263)	–	–	–	–
ACE	0.28 (0.00, 0.46)	0.00 (0.00, 0.30)	0.72 (0.54, 0.92)	765.50 (269)	14.09 (6)	–	–	2.09
AE	0.28 (0.08, 0.46)	–	0.72 (0.54, 0.92)	765.50 (270)	14.09 (7)	0.00 (1)	0.99	0.09
CE	–	0.19 (0.02, 0.35)	0.81 (0.65, 0.98)	767.79 (270)	16.38 (7)	2.29 (1)	0.13	2.38
E	–	–	1.0 (1.0, 1.0)	772.79 (271)	21.38 (8)	7.29 (2)	0.03	5.38
Alcohol use								
Baseline	–	–	–	681.65 (259)	–	–	–	–
ACE	0.74 (0.35, 0.85)	0.04 (0.00, 0.41)	0.22 (0.15, 0.32)	685.49 (265)	3.84 (6)	–	–	−8.16
AE	0.78 (0.68, 0.85)	–	0.22 (0.15, 0.32)	685.51 (266)	3.86 (7)	0.02 (1)	0.89	−10.14
CE	–	0.63 (0.51, 0.72)	0.37 (0.28, 0.49)	701.26 (266)	19.62 (7)	15.78 (1)	<0.01	5.62
E	–	–	1.0 (1.0, 1.0)	761.67 (267)	80.02 (8)	76.18 (2)	<0.01	64.02

Note. a² = additive genetic effects; c² = shared environmental effects; e² = nonshared environmental effects; 2lnL = −2 times log likelihood of data; df = degrees of freedom; AIC = Akaike's information criterion. The p value compares the relative fit between the ACE model and submodels. Ninety-five percent confidence intervals are in parentheses. The best-fitting model for each scale is indicated by bold type

^a χ² is computed by subtracting the −2lnL of the baseline model from the −2lnL of the submodel; the AIC is calculated using this fit statistic (i.e., AIC = χ² − 2*df)

^b Δχ² compares sub-models to the full ACE model

similarity in the magnitude of these effects, and the difficulty of accounting for BMI using a categorical compensatory behavior variable, we retained the continuous compensatory behavior variables in all analyses.

Bivariate models

Within-person, phenotypic associations between BN behaviors and problematic alcohol use were statistically significant and in the small-to-moderate range (see Table 1). Cross-twin, cross trait correlations revealed that these phenotypic associations were likely mediated by common genetic factors. Higher MZ relative to DZ cross-twin, cross-trait correlations were observed between binge eating and problematic alcohol use, as well as between compensatory behavior and problematic alcohol use, suggesting the presence of genetic influences on relationships between these phenotypes.

Results from the bivariate models are presented in Table 3. The AE model with r_e constrained to 0 provided the best fit to the data for associations between binge eating and problematic alcohol use as well as compensatory behavior and problematic alcohol use. These models evidenced a non-significant $\Delta\chi^2$ (see Table 3) relative to the baseline model, and a lower AIC than the AE model or the AE submodel with r_a constrained to 0. Parameter estimates for the best-fitting AE model with r_e constrained indicated some overlap ($r_a = 0.31$) in additive genetic factors between binge eating and problematic alcohol use, and a larger overlap ($r_a = 0.61$) for compensatory behavior and problematic alcohol use.

Discussion

The current investigation examined etiologic associations between bulimic behaviors (i.e., binge eating and compensatory behaviors) and problematic alcohol use in an effort to clarify past discrepant results regarding shared/independent transmission of these commonly comorbid phenotypes. Each behavior was separately influenced by genetic and nonshared environmental factors. However, associations between bulimic behaviors and problematic alcohol use appeared to be entirely mediated by common genetic rather than nonshared environmental factors. This suggests that the phenotypes do, in fact, aggregate in families, and that similar genes or heritable traits may be contributing to the co-occurrence of these disorders.

At the univariate level, we examined genetic and environmental influences on all three phenotypes, controlling for BMI. Heritabilities were 41, 28, and 78% for binge eating, compensatory behaviors, and problematic alcohol use, respectively, with the remaining variance due to

nonshared environmental effects. These estimates are within the ranges found in previous research (Bulik et al. 1998, 2003; Kendler et al. 1992, 1994; Klump et al. 2000; Mazzeo et al. 2010; Prescott et al. 1999; Reichborn-Kjennerud et al. 2004; Sullivan et al. 1998) and suggest that between 1/4 and 3/4 of the variance in individual differences in these phenotypes is due to genetic effects.

Bivariate twin models extended our univariate results by indicating that common genetic factors primarily influence associations between binge eating and problematic alcohol use ($r_a = 0.31$) and compensatory behaviors and problematic alcohol use ($r_a = 0.61$). Findings support those of Baker et al. (2010) and Mitchell et al. (2010) who found that shared genetic factors underlie associations between BN and alcohol use disorders. Our results complement and add to this work by showing that shared genetic factors are present for bulimic behaviors/problematic alcohol use symptoms, and by highlighting the fact that genetic overlap appears to be greater for problematic alcohol use and compensatory behaviors than it is for problematic alcohol use and binge eating. Taken together, these data suggest that etiologic associations between BN and problematic alcohol use disorders may be carried largely by associations with compensatory behaviors. One way to examine this possibility would be to compare genetic overlap between alcohol use disorders and BN to alcohol use disorders and purging disorder (i.e., a diagnosis that is characterized by the use of compensatory behaviors in the absence of binge eating; Keel et al. 2005). Based on findings in our study, we would expect stronger etiologic associations between alcohol use disorders and purging disorder than BN. Notably, in one study, women with purging disorder had higher current and lifetime rates of substance use disorders than women with BN and controls (Keel et al. 2005).

Future research should examine the mechanisms that might account for greater phenotypic and genetic associations between problematic alcohol use and compensatory behaviors than problematic alcohol use and binge eating. One potential explanation for this difference is impulsivity. Previous studies have indicated that women exhibiting bulimic pathology tend to score higher on measures of impulsivity compared to women with AN, restricting type, and controls (Cassin and von Ranson 2005). More specifically, however, a recent study of women exhibiting bulimic pathology found that higher levels of novelty seeking (a measure of impulsivity) was predictive of purging, but not binge eating (Wade et al. 2008). Further, extant research has found a significant genetic correlation between impulsivity and the use of compensatory behaviors ($r_a = -0.23$; note that correlation is negative due to low scores on the scale indicating high impulsivity; Klump et al. 2002). Notably, Klump et al. (2002) did not examine

Table 3 Standardized parameter estimates and model fit statistics for AE bivariate models for binge eating and alcohol use and compensatory behavior and alcohol use

	Heritability		Nonshared environmentality		Bivariate correlations		Test statistics				
	Alcohol use	BN symptom	Alcohol use	BN symptom	Genetic (r_a)	Nonshared (r_e)	-2lnL (df)	χ^2 (df) ^a	$\Delta\chi^2$ (df) ^b	p	AIC
Binge eating											
Baseline	-	-	-	-	-	-	1419.47 (514)	-	-	-	-
AE model	0.78 (0.68, 0.84)	0.41 (0.23, 0.56)	0.22 (0.16, 0.32)	0.59 (0.44, 0.77)	0.33 (0.08, 0.58)	-0.04 (-0.24, 0.17)	1433.32 (534)	13.85 (20)	-	-	-26.15
AE model, r_a constrained	0.77 (0.67, 0.84)	0.40 (0.21, 0.56)	0.23 (0.16, 0.33)	0.60 (0.44, 0.79)	-	0.08 (-0.12, 0.27)	1440.11 (535)	20.64 (21)	6.79 (1)	0.01	-21.36
AE model, r_e constrained	0.78 (0.68, 0.84)	0.41 (0.23, 0.56)	0.22 (0.16, 0.32)	0.41 (0.23, 0.56)	0.31 (0.09, 0.53)	-	1433.43 (535)	13.96 (21)	0.11 (1)	0.74	-28.04
Compensatory behaviors											
Baseline	-	-	-	-	-	-	1409.11 (514)	-	-	-	-
AE model	0.77 (0.68, 0.84)	0.26 (0.07, 0.44)	0.23 (0.16, 0.32)	0.74 (0.56, 0.93)	0.62 (0.32, 1.0)	0.00 (-0.21, 0.21)	1431.03 (534)	21.92 (20)	-	-	-18.08
AE model, r_a constrained	0.76 (0.64, 0.83)	0.23 (0.01, 0.42)	0.24 (0.17, 0.36)	0.77 (0.58, 0.99)	-	0.21 (0.01, 0.39)	1446.80 (535)	37.69 (21)	15.77 (1)	<0.01	-4.31
AE model, r_e constrained	0.77 (0.68, 0.84)	0.26 (0.07, 0.44)	0.23 (0.16, 0.32)	0.74 (0.56, 0.93)	0.61 (0.34, 1.0)	-	1431.03 (535)	21.92 (21)	0.00 (1)	0.99	-20.08

Note. r_a = genetic correlation; r_e = nonshared environmental correlation; -2lnL = -2 times log likelihood of data; df = degrees of freedom AIC = Akaike's information criterion; BN = bulimia nervosa; The "AE model, r_a constrained" constrains the genetic correlation to be 0. The "AE model, r_e constrained" constrains the nonshared environmental correlation to be 0. The p value compares the relative fit between the AE model and submodels. Ninety-five percent confidence intervals are in parentheses. The best-fitting model for each scale is indicated by bold type

^a χ^2 is computed by subtracting the -2lnL of the baseline model from the -2lnL of the submodel; the AIC is calculated using this fit statistic (i.e., $AIC = \chi^2 - 2*df$)

^b $\Delta\chi^2$ compares sub-models to the full AE model

genetic associations between impulsivity and binge eating due to a nonsignificant phenotypic correlation between the scales. Emerging data from our lab replicates these findings by showing a higher genetic correlation between impulsivity and compensatory behavior ($r_a = 0.37$) than impulsivity and binge eating ($r_a = 0.17$; Spanos et al. in preparation).

Importantly, impulsivity is associated with alcohol use disorders both cross-sectionally and prospectively (Sher et al. 2000) and predicts early initiation of alcohol (McGue et al. 2001) as well as frequency and quantity of use (Grau and Ortet 1999). Therefore, it may be that stronger phenotypic and genetic associations between problematic alcohol use and compensatory behavior are due to impulsivity as a “third variable” underlying both phenotypes. It is unclear why impulsivity might be more strongly linked phenotypically and genetically to compensatory behavior rather than binge eating. However, given the emerging data, this is an important area of research to pursue along with seeing if the impulsivity link underlies differential associations between problematic alcohol use, compensatory behavior, and binge eating.

Despite the potential importance of these findings for understanding in the comorbidity between bulimic behaviors and problematic alcohol use, some limitations must be noted. First, the sample size was relatively small, which may have decreased the power and precision in detecting genetic effects. However, none of the confidence intervals in the best-fitting models overlapped with zero, suggesting that we had adequate power to detect significant etiologic effects. Nonetheless, additional research with larger samples of female twins is needed to confirm our results.

Second, the internal consistency was only adequate for binge eating and compensatory behavior ($\alpha = 0.75$ and 0.65 , respectively). However, both interview and self-report questionnaires have been found to produce similar heritability estimates in twin studies (see Bulik et al. 1998; Sullivan et al. 1998; Reichborn-Kjennerud et al. 2004; Klump et al. 2002), despite clinical interviews being considered the “gold standard” for diagnosing bulimia nervosa (Fairburn and Beglin 1994). Nonetheless, future research should examine these phenotypes using clinical interviews to determine whether findings can be replicated.

Third, the majority of our twins were not entirely through the period of risk for the development of clinical eating disorders (i.e., through age 25) or alcohol use disorders (i.e., 20s to mid-30s; American Psychiatric Association 2000). Notably, however, the symptoms assessed typically predate the development of clinical eating disorders (Patton et al. 1999) and alcohol dependence (Conigrave et al. 1995) and so would be expected to be present even in “at risk” individuals. Indeed, research suggests that the “peak risk” of onset is 16 years old for binge eating

and 18 years old for purging symptoms (Stice et al. 1998). The present sample has an age range of 18–29 years with a mean age of 21; thus, the majority of participants are past the age of “peak risk” for these symptoms. Further, the AUDIT measure was specifically developed to identify individuals with problematic alcohol use before they develop more severe problems, such as alcohol dependence (Saunders et al. 1993). Thus, even though our subjects have not passed the peak period of risk for eating disorders and alcohol use disorders, findings are still informative for the types of symptoms that put individuals at risk for more severe eating and alcohol problems.

Finally, the direction of genetic associations between binge eating and problematic alcohol use, as well as compensatory behavior and problematic alcohol use, cannot be determined in our study, as our data were cross-sectional. Future research should examine prospective associations between these bulimic behaviors and problematic alcohol use in twins in order to further understand the direction and nature of the genetic and environmental associations.

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