Published in final edited form as:

Drug Alcohol Depend. 2015 August 1; 153: 335–340. doi:10.1016/j.drugalcdep.2015.05.043.

Genetic Overlap between Alcohol Use Disorder and Bulimic Behaviors in European American and African American Women

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Abstract

Background—Despite substantial evidence that alcohol use disorder (AUD) and bulimic behaviors (i.e., binge eating and compensatory behaviors) co-occur, insufficient information exists regarding a possible shared etiology. Moreover, although numerous twin studies of European ancestry individuals have reported moderate heritability estimates for AUD and bulimic behaviors, with little evidence for shared environmental factors, research on genetic and environmental risk in African American (AA) individuals is lacking.

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Contributors: Dr. Munn-Chernoff managed the literature searches, conducted the statistical analyses, and wrote the first draft of the manuscript. Drs. Bucholz, Madden, and Heath designed the study from which data were drawn and oversaw data collection. All authors provided input on the study design and edited the manuscript. All authors have approved the final manuscript.

Conflict of Interest: None of the authors report any conflict of interest.

Methods—We investigated specific and overlapping genetic and environmental influences on AUD and bulimic behaviors in 3,232 European American (EA; 55.38% monozygotic) and 549 AA (42.81% monozygotic) young adult female twins from the Missouri Adolescent Female Twin Study (age range=18–29 years). A structured clinical interview assessed lifetime DSM-5 AUD (minus craving) and bulimic behaviors. Biometrical twin modeling was conducted to generate age-adjusted estimates of genetic and environmental influences on AUD, bulimic behaviors, and their comorbidity.

Results—Estimates of genetic and environmental contributions on AUD and bulimic behaviors could be equated across EA and AA women. Additive genetic effects accounted for 59% (95% CI: 50%, 66%) and 43% (33%, 52%) of the variance in AUD and bulimic behaviors, respectively, with the remainder due to non-shared environmental effects. Shared genetic factors ($r_g=.33$ (.18, .49)) were solely responsible for the correlation between phenotypes; the non-shared environmental correlation was not significant ($r_e=.10$ (–.05, .25)).

Conclusions—Findings indicate similar magnitudes of genetic and environmental effects on AUD and bulimic behaviors for EA and AA women and implicate common genetic mechanisms underlying liability to these problem behaviors.

Keywords

twins; African American; alcohol use disorder; eating disorders; DSM-5; heritability

1. INTRODUCTION

The association between alcohol use disorder (AUD) and its psychiatric correlates that are more commonly found in women, such as eating disorders (e.g., anorexia nervosa, bulimia nervosa) and their symptoms (e.g., binge eating and compensatory behaviors, collectively referred to as bulimic behaviors) has been well-established. For example, 25% of women with bulimia nervosa have had an AUD (Hudson et al., 2007; Kessler et al., 2013) and 36% of women with an AUD report binge eating (Peveler and Fairburn, 1990) in their lifetime. Still, etiological factors common to AUD and bulimic behaviors are relatively unknown. Despite early reports of little overlapping genetic risk between AUD and bulimia nervosa (Wilson, 2010), emerging research has implicated genetic factors in their comorbidity, particularly when considering bulimic behaviors rather than clinical diagnoses (Baker and Munn-Chernoff, 2014). Unfortunately, previous genetically informative studies on AUD, bulimic behaviors, and their comorbidity have rarely included individuals of non-European ancestry. Thus, we do not know whether findings generalize to other racial/ethnic groups, including African American (AA) women.

In European ancestry samples, heritability estimates for AUD range from 50% to 64% (Baker et al., 2010; Dick et al., 2009; Edwards et al., 2013; Grant et al., 2009; Slutske et al., 2013; Trace et al., 2013b), 28% to 83% for bulimia nervosa (Trace et al., 2013a), and 8% to 72% for bulimic behaviors (Baker et al., 2010; Klump et al., 2000; Munn-Chernoff et al., 2013, 2015; Slane et al., 2012; Sullivan et al., 1998; Wade et al., 2008), with generally little evidence for environmental effects shared among family members. Studies including non-European ancestry twins reported possible distinctions in magnitudes of genetic and

environmental effects on problem alcohol use (Sartor et al., 2013) but similar heritability estimates for binge eating (Munn-Chernoff et al., 2015) in EA versus AA women. Determining whether genetic and environmental influences on AUD and bulimic behaviors differ between EA and AA women is critical because observed racial/ethnic differences in prevalence (Crago et al., 1996; Kessler et al., 1994; Marques et al., 2011; Neumark-Sztainer et al., 2002; Wu et al., 2011) and age of onset (Alvanzo et al., 2011; Stice et al., 1998; Taylor et al., 2007; Wagner et al., 2002) for AUD and bulimic behaviors, as well as cultural body ideals (Gordon et al., 2010) relevant to bulimic behaviors, suggest that existing etiological models of these problem behaviors derived from European-ancestry samples may not apply to AA women. Investigating sources of these differences could lead to improvement in AA models of AUD and bulimic behavior development.

To address critical limitations of prior research on AUD and bulimic behavior etiology, we examined potential distinctions in genetic and environmental underpinnings of these problem behaviors in EA and AA young women. Additionally, we aimed to replicate recent findings of shared genetic liability between AUD and bulimic behaviors and to determine whether findings differed by race/ethnicity. Investigating genetic and environmental risks on AUD and bulimic behaviors could yield important information for tailoring prevention and treatment strategies for these problem behaviors to particular racial/ethnic groups.

2. MATERIALS AND METHODS

Data were from the Missouri Adolescent Female Twin Study (MOAFTS; Heath et al., 2002; Waldron et al., 2013). MOAFTS is a population-based longitudinal study of female twins born between July 1st 1975 and June 30th 1985 in Missouri identified from state birth records. Zygosity was determined by standard questions (Nichols and Bilbro, 1966). Two years after the Wave 1 assessment, conducted from 1995 to 1999 (median age=15 years), Wave 3 retest interviews were conducted with a subset of Wave 1 twins. (Wave 2 interviews were limited in scope and are not included here.) Wave 4 interviews were conducted approximately six years after Wave 1 (median age=22 years) and served as the base sample for this study because all twins could be contacted directly for participation. This resulted in 3232 (85%) EA twins (1517 complete pairs, 55.38% monozygotic (MZ)) and 549 (15%) AA twins (253 complete pairs, 42.81% MZ), out of 3998 EA twins (1999 pairs) and 740 AA twins (370 pairs) originally identified from birth records. Overall, 81% of EA and 74% of AA respondents from the original sampling frame were included in the current analyses. The protocol was approved by the Washington University School of Medicine Institutional Review Board; all twins gave informed consent before study participation.

A telephone interview using an adaptation of the Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA; Bucholz et al., 1994) assessed DSM-IV lifetime criteria of AUD and eating disorders. Only women reporting ever trying alcohol at Waves 1, 3, or 4 were included in these analyses. Although the interview was based on DSM-IV, we created a DSM-5 AUD diagnosis to be consistent with the current conceptualization of AUD. Individuals endorsing 2+ of the 10 symptoms (7 dependence plus 3 abuse, legal problems excluded) clustering within the same 12-month period were coded positive for AUD if they

met diagnostic criteria for AUD at Waves 1, 3, or 4. Craving, a new DSM-5 symptom, was not assessed.

Eating disorder information was only collected at Wave 4. Women were coded positive for lifetime bulimic behaviors if they reported ever engaging in binge eating or compensatory behaviors. Binge eating was defined as eating a large amount of food in a short period of time, usually less than 2 hours, *and* during these binge episodes, having a sense of loss of control over the eating. Compensatory behaviors were coded positive if the twin reported ever making herself vomit; taking laxatives; dieting strictly; fasting; exercising vigorously for a long time; and/or taking water pills or diuretics to lose weight or prevent weight gain. Respondents were asked about compensatory behaviors regardless of responses to binge eating questions (i.e., there were no skip patterns), which is important given research suggesting that clinically significant eating pathology is missed when following skip patterns in structured interviews (Swanson et al., 2014).

Survey commands in STATA (StataCorp, 2005) were used to correct for the twin sampling design. Model-fitting analyses were performed in Mx (Neale et al., 2006). Using raw data, bivariate twin models were fitted to examine three sources of variance/covariance influencing liability to AUD and bulimic behaviors: additive genetic (A), shared environmental (environmental factors shared by twin pairs reared together, C), and non-shared environmental (environmental factors unique to each family member for a trait and measurement error, E) effects. We evaluated the full ACE models for each phenotype within EA (n=3232) and AA (n=549) subsamples. Submodels where parameter estimates (i.e., A, C, or E) were set to zero were compared to the full models to determine whether a given parameter estimate could be dropped. Minus two log-likelihood ($-2LL$) and standard chi-square difference tests (χ^2) (Neale and Cardon, 1992) were used to compare the fit of nested models. Akaike's Information Criterion (AIC) (Akaike, 1987) was also used for model comparisons, where lower AIC values indicate more parsimony and suggest a better model fit. We controlled for age using a median split by allowing prevalence rates to differ by age.

3. RESULTS

A larger proportion of EA than AA women reported ever having at least one full drink of alcohol (88.18% vs. 78.32, $p<.001$) and meeting DSM-5 AUD criteria (among ever-drinkers: 34.99% vs. 18.29%; $p<.001$). There were no racial/ethnic differences in rates of bulimic behaviors (21.86% EA vs 23.62% AA; $p=.39$) or rates of comorbid AUD and bulimic behaviors (among ever-drinkers: 10.21% EA vs. 7.42% AA; $p=.09$). The phenotypic correlation between AUD and bulimic behaviors was .20 (95% confidence interval: .13, .27) in EA women, .33 (.14, .50) in AA women, and .22 (.15, .29) in the full sample.

We first evaluated relative contributions of genetic and environmental factors to the variance and covariance of AUD and bulimic behaviors separately in the EA and AA subsamples (Tables 1 and 2). Given the wide 95% confidence intervals surrounding parameter estimates, we then investigated whether we could equate genetic and environmental effects between EA and AA women (Tables 1 and 2). We started with a model that allowed for all three

sources of variance and covariance because it provided an adequate fit to the data for both subsamples. The constrained model equating all parameter estimates in EA and AA women (Model 9) provided a better fit to the data ($\chi^2=4.21$; $df=7$; $p=.76$) than a model allowing all parameter estimates to differ in both groups (Models 1 and 5). Dropping additional parameters yielded a best-fitting model that included only additive genetic and non-shared environmental effects for AUD, bulimic behaviors, and their comorbidity (Model 11). In the best-fitting model, heritability estimates were 59% (50%, 66%) for AUD and 43% (33%, 52%) for bulimic behaviors. Only the genetic correlation was significant ($r_g=.33$ (.18, .49)), suggesting that 11% of the genetic variance in AUD and bulimic behaviors overlapped for both EA and AA women. Non-shared environmental effects were not significantly correlated ($r_e=.10$ (-.05, .25)).

4. DISCUSSION

Using the largest available AA twin sample, we compared genetic and environmental contributions to AUD and bulimic behaviors in EA versus AA young adult women. There were significant genetic and environmental effects for AUD, bulimic behaviors, and their comorbidity in both racial/ethnic groups. Although point estimates suggested heritabilities higher for AUD and lower for bulimic behaviors in AA women, 95% CIs were wide and included zero; thus, we were able to equate estimates between subgroups.

Corroborating prior research, we found significant genetic and non-shared environmental effects on AUD (Dick et al., 2009; Edwards et al., 2013; Grant et al., 2009; Slutske et al., 2013; Trace et al., 2013b) and bulimic behaviors (Baker et al., 2010; Klump et al., 2000; Munn-Chernoff et al., 2013, 2015; Slane et al., 2012; Sullivan et al., 1998; Trace et al., 2013a, 2013b). Although the heritability estimates for AUD were numerically higher and for bulimic behaviors were numerically lower in AA than in EA women, these differences were not statistically significant. These findings are consistent with those from another study (Sartor et al., 2013), which, although did not show significant differences in genetic and non-shared environmental effects between the two racial/ethnic groups for problem alcohol use, suggest that true racial/ethnic distinctions may exist. Such differences in racial/ethnic groups could reflect cultural differences, whereby, for example, a lower prevalence of AUD is driven more by genetic than environmental factors (Sartor et al., 2013). Additionally, it is possible that gene-by-shared environment interactions, which are included the heritability estimate (Heath and Martin, 1993), exist and account for some of the differences in prevalence and heritability between EA and AA women observed here, whereby the interaction could be stronger in one racial/ethnic group than the other. The current study was underpowered to examine potential effects of specific environmental risk factors, such as trauma exposure, on the heritability of AUD and bulimic behaviors in these subsamples. For example, research has found significantly elevated rates of lifetime alcohol dependence in women who experience assaultive trauma (Sartor et al., 2011) and disparities in trauma exposure have been reported between EA and AA women (Duncan et al., 2014; Roberts et al., 2011). Including AA women in future genetically informative studies is critical to better understand possible racial/ethnic differences in gene-environment interplay on these problem behaviors.

Our findings contrast earlier work (Wilson, 2010) but support emerging research in women of European ancestry (Baker et al., 2010; Munn-Chernoff et al., 2013; Slane et al., 2012; Trace et al., 2013) implicating shared genetic risk for problem alcohol use and bulimic behaviors. It is possible that this association is due to unmeasured factors, including other comorbid psychopathology or specific personality traits; however, one study did not find that behavioral disinhibition and emotion dysregulation moderated the genetic correlation between AUD and bulimic symptoms (Slane et al., 2014). Although specific genetic mechanisms underlying this comorbidity are unclear, at minimum, individuals with AUD should be screened for an individual and family history of eating disorders and vice versa, regardless of race/ethnicity, given similarities in shared genetic risk between EA and AA women. Environmental factors contributing to this relation should continue to be explored, especially in the context of genetically informative research designs.

Our results are limited by modest power to detect racial/ethnic differences in the relative contributions of genetic and environmental effects on the phenotypes examined, the absence of craving in the AUD assessment, and that a small proportion of individuals are not yet through the risk period for developing AUD or bulimic behaviors. Future studies should use extended family designs and integrate twin samples with non-twin family designs to more definitively address whether true racial/ethnic differences in genetic and environmental risk for AUD, bulimic behaviors, and their comorbidity exist. Nevertheless, these findings underscore the importance of including AA women and examining eating disorder symptoms -- which are more prevalent in women -- in genetically-informative alcohol research. Clinicians must address eating disorders when treating AUD and vice versa, a practice that is routinely ignored (Dennis et al., 2014), in order to decrease detrimental effects associated with this comorbidity.

Acknowledgments

Author Disclosures

Role of Funding Source: This work was supported by NIH grants: R01 DA023668 and R21 AA021235 (AA); K08 AA017921 (CES); and R01 HD049024, R01 AA009022, R01 AA017915, R01 AA021492, R37 AA007728, P60 AA011988, and K05 AA017688 (ACH). MMC was supported by T32 AA007580 (ACH) and KBW was supported by T32 DA015035 (Cunningham-Williams). CES was also supported by a grant from the Robert E. Leet and Clara Guthrie Patterson Trust. The NIH had no further role in study design; in the collection, analysis, and interpretation of data; in the writing of the report; or in the decision to submit the paper for publication.

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Highlights

- Alcohol use disorder (AUD) and bulimic behaviors co-occur, yet it is unknown why.
- Few studies have included African American women in genetic research.
- We examined their shared etiology in European American and African American women.
- Common genetic and environmental factors underlie liability to these behaviors.
- There were no significant differences between racial/ethnic groups.

Table 1

Model-fitting results for alcohol use disorder (AUD) and bulimic behaviors

Model	Model-Fit				Comparative Fit		
Number	Model	-2LL	df	AIC	χ^2	df	p
European American Subsample							
1	ACE-ACE r_g, r_c, r_e	6257.66	5614	-4970.34	---	---	---
2	CE-CE r_c, r_e	6265.11	5617	-4968.89	7.45	3	.06
3	AE-AE r_g, r_e	6261.97	5617	-4972.03	4.31	3	.23
4	E-E r_e	6442.52	5620	-4797.48	184.86	6	<.001
African American Subsample							
5	ACE-ACE r_g, r_c, r_e	902.14	874	-845.86	---	---	---
6	CE-CE r_c, r_e	903.18	877	-850.82	1.04	3	.79
7	AE-AE r_g, r_e	902.83	877	-851.17	.69	3	.88
8	E-E r_e	919.94	880	-840.06	17.80	6	.01
Full Sample							
9*	ACE-ACE, r_g, r_c, r_e Fully Constrained	7164.01	6495	-5825.99	4.21	7	.76
10	CE-CE, r_c, r_e Constrain C, E	7173.03	6498	-582.97	9.02	3	.03
11	AE-AE, r_g, r_e Constrain A, E	7168.61	6498	-5827.39	4.60	3	.20
12	E-E, r_g, r_e Constrain E	7364.20	6501	-5637.80	200.19	6	<.001

Note: A = additive genetic effects; C = shared environmental effects; E = nonshared environmental effects; r_g = correlation between AUD and bulimic behaviors that is due to additive genetic influences; r_c = correlation between AUD and bulimic behaviors that is due to shared environmental influences; r_e = correlation between AUD and bulimic behaviors that is due to nonshared environmental influences, -2LL = -2 log likelihood; AIC = Akaike's Information Criterion; χ^2 = chi-square difference test statistic.

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This model was compared with an unconstrained model that allowed for all parameter estimates to differ between subsamples ($-2LL=7159.80$; $df=6488$; $AIC=-5816.20$); it provided a better fit to the data compared with the unconstrained model ($\chi^2=4.21$; $df=7$; $p=.76$) and was included in further model testing. The best-fitting model is indicated in bold-type.

*

Table 2

Proportions of variance due to genetic and environmental factors for bivariate twin models

Model	AUD			Bulimic Behaviors			Correlations			
Number	Model	a ²	c ²	e ²	a ²	c ²	e ²	r ^g	r ^c	r ^e
European American Subsample										
1	ACE-ACE r _g , r _c , r _e	.31 (.03, .61)	.24 (.00, .48)	.45 (.36, .55)	.28 (.00, .53)	.15 (.00, .39)	.57 (.46, .68)	.68 (-.29, 1.0)	-.11 (-1.0, 1.0)	.07 (-.09, .23)
2	CE-CE r _c , r _e	---	.48 (.40, .56)	.52 (.44, .60)	---	.37 (.28, .46)	.63 (.55, .72)	---	.29 (.13, .45)	.14 (.01, .26)
3	AE-AE r _g , r _e	.58 (.49, .66)	---	.42 (.34, .51)	.45 (.35, .55)	---	.55 (.45, .65)	.31 (.15, .47)	---	.09 (-.07, .24)
4	E-E r _e	---	---	1.00	---	---	1.00	---	---	.20
African American Subsample										
5	ACE-ACE r _g , r _c , r _e	.29 (.00, .85)	.33 (.00, .73)	.38 (.15, .70)	.27 (.00, .54)	.00 (.00, .37)	.73 (.46, 1.0)	.77 (-1.0, 1.0)	.98 (-1.0, 1.0)	.22 (-.24, .64)
6	CE-CE r _c , r _e	---	.54 (.28, .74)	.46 (.26, .72)	---	.18 (.00, .39)	.82 (.61, 1.0)	---	.48 (-.20, 1.0)	.29 (-.04, .59)
7	AE-AE r _g , r _e	.67 (.35, .87)	---	.33 (.13, .65)	.27 (.00, .54)	---	.73 (.46, 1.0)	.52 (-.07, 1.0)	---	.23 (-.25, .64)
8	E-E r _e	---	---	1.00	---	---	1.00	---	---	.33
Full Sample										
9	ACE-ACE, r _g , r _c , r _e Fully Constrained	.31 (.03, .60)	.25 (.00, .48)	.44 (.36, .54)	.31 (.02, .51)	.10 (.02, .35)	.59 (.49, .70)	.64 (.20, 1.0)	-.17 (-1.0, 1.0)	.09 (-.07, .24)
10	CE-CE, r _c , r _e Constrain C, E	---	.49 (.41, .56)	.51 (.44, .59)	---	.34 (.26, .42)	.66 (.58, .74)	---	.30 (.14, .46)	.16 (.04, .27)
11	AE-AE, r _g , r _e Constrain A, E	.59 (.50, .66)	---	.41 (.34, .50)	.43 (.33, .52)	---	.57 (.48, .67)	.33 (.18, .49)	---	.10 (-.05, .25)
12	E-E, r _g , r _e Constrain E	---	---	1.0	---	---	1.0	---	---	.21

Note: AUD = alcohol use disorder; a^2 = additive genetic influences; c^2 = shared environmental influences; e^2 = nonshared environmental influences; r_g = correlation between AUD and bulimic behaviors that is due to additive genetic influences; r_c = correlation between AUD and bulimic behaviors that is due to shared environmental influences; r_e = correlation between AUD and bulimic behaviors that is due to nonshared environmental influences. 95% confidence intervals are presented in parentheses. The best-fitting model is indicated in bold-type.