Published in final edited form as:

Alcohol Clin Exp Res. 2013 November ; 37(11): . doi:10.1111/acer.12185.

Are There Differences Between Young African-American and European-American Women in the Relative Influences of Genetics vs. Environment on Age at First Drink and Problem Alcohol Use?

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Abstract

Background—Differences in age at initiation of alcohol use and rates of problem drinking between African Americans (AA) and European Americans (EA) are well documented, but the association between early and problem use – and distinctions by ethnic group in this association – have yet to be examined in a genetically-informative framework.

Methods—Data were derived from a longitudinal study of female twins in Missouri. The sample was composed of 3,532 twins (13.6% AA, 86.4% EA) who participated in the fourth wave of data collection and reported consumption of at least one alcoholic drink over the lifetime. Mean age at Wave 4 was 21.7 (range=18–29) years. Twin modeling was conducted to estimate the relative contributions of additive genetic (A), shared environmental (C), and unique environmental (E) factors to variation in age at first drink and problem alcohol use and the cross-phenotype overlap in these influences.

Results—Early initiation of alcohol use predicted problem use in EA but not AA women. Separate AA and EA twin models produced substantially different estimates (but not statistically different models) of the relative contributions of A and C to problem alcohol use but similar genetic correlations between the phenotypes. Whereas 33% of the variance in the EA model of problem use was attributed to C, no evidence for C was found in the AA model. Heritability estimates for problem alcohol use were 41% in the AA model, 21% in the EA model. Evidence for A and C were found in both AA and EA models of age at first drink, but the A estimate was higher in the EA than AA model (44% vs. 26%).

Conclusions—Findings are suggestive of distinctions between AA vs. EA women in the relative contribution of genetic and environmental influences on the development of problem drinking.

Keywords

alcohol; African Americans; women; twins

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The authors have no conflicts of interest to declare.

INTRODUCTION

Early age at first drink has been associated with elevated risk for alcohol use disorders (AUDs) in numerous studies (Grant and Dawson, 1997; Hingson et al., 2006; Prescott and Kendler, 1999), but few investigations have explored the potential variability across ethnic groups in the strength of this association. Lower rates of alcohol use among African Americans vs. European Americans are well documented (Dauber et al., 2009; Horton, 2007; Wu et al., 2011). Differences in drinking behaviors extend to age at first alcohol use and prevalence of problem drinking as well. African Americans report an older average age at first drink (Rothman et al., 2009; Wagner et al., 2002) and fewer alcohol-related problems than European Americans (Dauber et al., 2009; Horton, 2007; Skidmore et al., 2012; Wu et al., 2011). If these distinctions reflect differential pathways to drinking problems, the association between age at first drink and problem alcohol use may also differ for African Americans vs. European Americans, but this has only rarely been investigated. Findings from a longitudinal study of adolescent males indicating a more pronounced elevation in risk for alcohol-related problems in African American (AA) vs. European-American (EA) male early drinkers (Horton, 2007) lends support to this idea, but it has yet to be explored in female adolescents or women.

Heritability of Initiation of Alcohol Use and Alcohol-related Problems

Twin studies have made major contributions to etiological models of alcohol use and the development of alcohol-related problems, but African Americans are underrepresented in twin studies of alcohol phenotypes, so it is unclear to what extent their findings generalize to African Americans. The review of findings provided here is based on primarily EA samples, a limitation we are attempting to remedy with the current study.

Twin studies of alcohol use initiation indicate that genetic factors play a significant role in the timing of first alcohol use. Estimates of the proportion of variance attributable to heritable influences cover a wide range, from 0–72% (Koopmans and Boomsma, 1996; Maes et al., 1999; Pagan et al., 2006), but the majority of studies in this area have reported a more substantial contribution of common environmental than genetic factors. Common – also known as shared – environmental influences, are non-genetic factors that make members of a twin pair similar on a given phenotype, such as familial, school, and neighborhood influences. Shared environment has been found to account for approximately half of the total variance in initiation of alcohol use (Fowler et al., 2007; Koopmans and Boomsma, 1996; Pagan et al., 2006; Rhee et al., 2003). Findings from twin studies of alcohol dependence (AD) indicate a very different model for AD. Consistently across studies of both males and females, 50–60% of variance in AD is attributed to additive genetic influences and there is no evidence for a significant contribution of shared environment (Heath et al., 1997; Kendler et al., 1994; Knopik et al., 2004; Reed et al., 1996; van den Bree et al., 1998)

The examination of intermediate stages of problem alcohol use is critical to understanding how the contribution of genetics vs. environment shifts during the course of alcohol use, but only rarely have such studies been undertaken. Findings from among the few existing studies are somewhat inconsistent. Fowler et al.'s (2007) study of 11 to 19 year-olds reported that 41% of variance in the problem drinking indicator “getting into a situation you later regretted” was attributable to genetics, 16% to shared environment, whereas Rhee et al.'s (2003) study of adolescents in the same age range reported that 78% of variance in AUD symptoms was accounted for by genetics, none by shared environment. Another approach to elucidating the pathway from first use to problem use is to examine the overlap in genetic and environmental influences on the two phenotypes. The evidence suggests that approximately one third of the variance in AD is shared with variance in initiation (Fowler

et al., 2007; Prescott and Kendler, 1999) and a recent study of Australian twins reported a correlation of 0.59 between the heritable factors influencing age at initiation and those influencing AD (Sartor et al., 2009). Prescott and Kendler's (1999) investigation of initiation of alcohol use and AD revealed that the association could be accounted for entirely by familial influences, nearly all genetic, but others have concluded that the association is not attributable in full to familial risk factors (Grant et al., 2006). The degree to which heritable influences on initiation and problem use can be traced to a common source determines the extent to which age at first drink can be treated as a marker for genetic liability to alcohol-related problems. Given the inconsistencies between African Americans and European Americans on lifetime use, age at first drink, rates of alcohol-related problems, and the association between early use and problem drinking, investigation of this question in a sample including both ethnic groups is warranted.

Study Aims

The current study builds on the existing twin research on alcohol phenotypes to explore distinctions between young AA and EA women in age at first alcohol use and development of problem use. The aims of our investigation are: (1) to test for possible differences in rates of early and problem alcohol use and the association of the two; and (2) to quantify the relative contributions of genetic and environmental influences to age at first drink and problem use and the overlapping influences on these phenotypes.

MATERIALS AND METHODS

Participants

The sample was composed of female twins who completed the fourth wave of data collection for the Missouri Adolescent Female Twin Study (MOAFTS), a longitudinal study of alcohol use disorders and related psychopathology in female adolescents and young adults. Female twins born in Missouri to Missouri-resident parents between 1975 and 1985 were identified through birth records and recruited between 1995 and 1999 for the baseline (Wave 1) assessment. Cohorts of 13, 15, 17, and 19 year-old female twin pairs and their families were recruited in the first two years. New cohorts of 13 year-old twins and their families were ascertained in the subsequent two years. Parent interviews were completed by at least one parent in 78% of targeted families. (See Heath et al. (2002) for details on sample ascertainment). Two years after Wave 1 assessments of parents and twins were conducted, Wave 3 retest interviews were administered to a subset of Wave 1 twin participants. (Data from Wave 2 twin interviews were not used in the current study, as they were more limited in scope.) Wave 4 interviews were conducted from 2002 to 2005. Wave 5 interviews were conducted between 2005 and 2008. Of the 4,638 twins identified from birth records, 80% completed the Wave 4 interview ($n=3,787$), the primary source of data for this study. The mean age of participants at Wave 4 was 21.7 ($SD=2.8$; range=18–29) years; 14.6% of participants self-identified as African-American, the remainder as European-American.

Procedure and Assessment

The MOAFTS study was approved by the Washington University Human Research Protections Office. Data were collected by trained interviewers through a psychiatric interview modified for telephone administration from the Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA) (Bucholz et al., 1994, Hesselbrock et al., 1999). The SSAGA queries history of substance use and related psychosocial history in addition to diagnostic information for DSM-IV psychiatric disorders. Waves 1, 3, and 4 interviews assessed lifetime psychiatric and psychosocial history. Wave 5 interviews, which were conducted approximately 2 years after Wave 4 interviews, queried the previous 2 years (i.e.,

roughly the period between Wave 4 and Wave 5 assessments). Verbal consent was obtained prior to starting interviews.

Deriving Alcohol Phenotypes—Data from Waves 1 and 3 were available for 78% of participants. In cases where age at first drink or AUD symptoms were reported in more than one wave of data collection, the first report was used.

Age at First Drink—Respondents who reported ever consuming at least one full alcoholic drink in Waves 1, 3, or 4 were asked, “How old were you when you had your very first full drink?” For those women who did not endorse any alcohol use at Wave 4 (or in any prior interview) and completed the Wave 5 interview, we used Wave 5 data to determine lifetime alcohol use status and, among non-abstainers, to estimate age at first use. Wave 5 respondents were asked, “When was the last time that you had a drink with alcohol in it?” Those who responded “never” were coded as non-drinkers. For all others, age at first drink was estimated by subtracting 2 years (the approximate time between Waves 4 and 5) from age at the time of the Wave 5 interview. This method was used to estimate age at first drink in 237 participants. (A total of 6.7% of respondents were lifelong abstainers.)

Age at first drink was broken into 3 categories based on the overall sample distribution. “Early” was defined as the lowest quartile (14 or younger), “average” as the middle 50% (15–17), and “late” as the highest quartile (18 or older). We used a categorical variable because it better represents developmental periods (early, middle, and late adolescence) than does a continuous variable, and it allows for the interpretation of age at initiation of alcohol use in relative terms, i.e., earlier vs. later than individuals in the same birth cohort. Analyses were conducted with all participants who reported an age at first drink (available from all respondents who endorsed lifetime use). The sample analyzed in the current study ($n=3,532$) was composed of 91 monozygotic (MZ) AA pairs, 109 dizygotic (DZ) AA pairs, 80 AA singletons, 785 MZ EA pairs, 602 DZ EA pairs, and 278 EA singletons.

Problem Alcohol Use—Problem alcohol use was defined as the endorsement in any wave of data collection of at least one DSM-IV alcohol abuse symptom or one AD symptom other than tolerance. Respondents with tolerance only, which comprised 30% of cases reporting one or more AUD symptoms, were recoded as non-problem users to create a more conservative definition of problem alcohol use (in keeping with evidence of inflated rates of tolerance endorsement in young drinkers suggestive of misinterpretation of tolerance questions [Caetano and Babor, 2006; Chung and Martin, 2005]).

Data Analysis

All analyses were conducted with singletons as well as complete twin pairs. Although singletons are not informative with respect to the relative contribution of genetic vs. environmental effects, they contribute to estimates of phenotypic variance in twin models.

Phenotypic Analyses—The proportion of the sample endorsing early, average, and late age at first alcohol use and the prevalence of problem use in each of these categories was calculated by ethnicity, using SAS (SAS Institute, 2008). To estimate the risk for problem use associated with early and late (vs. average) age at first drink, logistic regression analyses were conducted separately for the AA and EA subsamples using Stata (StataCorp, 2007). The Huber-White correction was applied to adjust for the non-independence of observations in twins.

Twin Modeling—Data from MZ and DZ twin pairs can be utilized to quantify the relative contribution of additive genetic (A), shared environmental (C) and non-shared

environmental (E) factors to a phenotype. Non-additive (including dominance, and thus denoted as D) genetic influences can be estimated in place of, but not jointly with C when data from twins alone are used. The decision to fit an ACE vs. ADE model is based on the ratio of DZ to MZ twin correlations on the phenotype of interest. A DZ correlation greater than half the MZ correlation indicates that environmental influences that make members of a twin pair similar (C) are contributing to the phenotype and thus provides support for an ACE model. Conversely, a DZ correlation less than half the MZ correlation supports an ADE model. (See Martin et al. (1997) for an overview of twin methodology).

All twin modeling was conducted with the statistical software package OpenMx (2011), which uses maximum likelihood estimation to evaluate the extent to which variance in each phenotype can be attributed to A, C, and E. Twin models were fitted to raw categorical data. Modeling was conducted with the AA subsample, the EA subsample, and the full sample. Three bivariate triangular (also known as Cholesky) decomposition analyses were fitted to assess the degree of overlap in genetic and environmental influences between age at first drink and problem use as well as heritabilities for each phenotype. A series of sub-models were tested to assess the statistical significance of pathways representing additive genetic, shared environmental, and non-shared environmental influences, both within and across phenotypes, to derive the best-fitting bivariate models. The sub-models were tested by calculating the difference between the -2 log likelihood fit of the full model and the nested sub-model, which is distributed as chi-square for the given degrees of freedom.

RESULTS

Age at First Drink and Problem Use by Ethnicity

The distribution of age at first drink and the prevalence of problem use (by age at first drink), are shown separately for AA and EA participants in Table 1. Early use was less common among AA than EA adolescents; 26.6% of AA participants reported first alcohol use before age 15, compared with 41.0% of EA participants. (In a Chi-square test of association between age at first use and ethnicity, $\chi^2_{(1)}=88.88$ ($p<0.01$), this was statistically significant.) Problem use was also less prevalent among African Americans than European Americans (33.1% vs. 43.8%; $\chi^2_{(1)}=31.23$, $p<0.01$). Logistic regression analyses revealed that the risk for problem use associated with early age at first drink was significant for European Americans (OR=1.74, CI:1.46–2.07) but not African Americans (OR=1.42, CI:0.84–2.40), whereas late age at first drink was protective for both (OR=0.50, CI:0.31–0.81 for AAs; OR=0.31, CI:0.25–0.39 for EAs).

Twin Models

Twin correlations were conducted (separately for AA and EA participants) to determine whether an ACE or ADE model would better fit the data. DZ twin correlations were greater than half of MZ twin correlations in all cases except for problem use in African Americans ($r_{MZ}:r_{DZ}$ was 0.36), thus supporting an ACE model. (A summary of the model fitting steps for the AA subsample, EA subsample, and full sample bivariate models (with fit indices, point estimates and confidence intervals for variance components, and results of tests of change in model fit for each sub-model) is available upon request.) The best fitting models, indicated by the lowest Akaike information criterion (AIC), were ACE for both phenotypes in the EA subsample and ACE for first drink, AE for problem use in the AA subsample. The best fitting model using the full sample, in which pathways for the AA and EA participants were set to be equal, also included A, C, and E variance components for both phenotypes. Additional tests conducted with each of the three best-fitting models to assess significance of the cross-phenotype additive genetic (a_{21}), shared environmental (c_{21}), and unique environmental pathways (e_{21}) (i.e., covariances) indicated significance of a_{21} in all three

models, c_{21} in the EA model, and e_{21} in the EA and full sample models. The resulting final models are shown with unstandardized path coefficients in Figures 1, 2, and 3. The standardized variance component estimates and correlations are reported with 95% confidence intervals in Table 2. For both the AA and EA subsamples, age at first drink was attributable to shared environmental as well as additive genetic and unique environmental sources of variance. Very similar estimates of C were observed in the AA and EA models ($C=0.25$ for AA, 0.27 for EA), but A estimates were considerably higher in the EA than AA model (0.44 vs. 0.26). The AA and EA models also diverged on problem alcohol use. No evidence of a shared environmental effect was observed in the AA model, whereas 33% of the variance in problem use was attributed to C in the EA model. Genetic correlations between age at first drink and problem use were comparable in the AA and EA models (0.29 and 0.20 , respectively). As seen in Table 2, the full sample model (with AA and EA pathways equated) closely resembled the EA model.

Because the differences between the point estimates for problem use in the AA and EA models were so large, we conducted an additional analysis aimed at determining whether they were likely to be reflective of a true difference that was not detected due to low power resulting from a relatively small number of African Americans in the full sample. In a series of univariate models using the full sample, the estimate for C in the EA subsample was multiplied by a constant ranging from .5–50 in increments of .5 (totaling 100 models), thus setting the relative value of C in the EA subsample to between .5 and 50 times that in the AA subsample. (A more detailed description of this approach by Duncan et al. (unpublished) is available upon request.) The best-fitting model was reached when the constant was set to 18, and it remained at that value for all subsequent models. Results indicate that the most likely model of problem use is one in which the shared environmental component is 18 or more times greater in European Americans than African Americans.

DISCUSSION

Our examination of the timing of alcohol use initiation and subsequent development of problem drinking in AA and EA women using genetically-informative data is a unique contribution to the literature on alcohol-related problems in women. Our study is among the first to test for potential differences between African Americans and European Americans in the degree of association between early age at first drink and problem alcohol use and the first known study to estimate the relative contributions of genes and environment to either phenotype in African Americans. Problem use, which we defined as one or more AUD symptoms (other than tolerance), has received far less attention than alcohol dependence in genetically-informative research, but the high prevalence of problem drinking in the population (Johnston et al., 2011) and the lowered quality of life associated with even one AUD symptom suggest the importance of enhancing our understanding of this alcohol-related outcome.

The higher rates of alcohol use, early initiation of use, and problem drinking in EA vs. AA women observed in our sample is consistent with findings from a growing literature on differences in drinking behaviors between African Americans and European Americans (Dauber et al., 2009; Skidmore et al., 2012; Wagner et al., 2002; Wu et al., 2011). However, very few investigations have compared risk for problem drinking associated with early use across ethnic groups, and the only known study to compare African Americans and European Americans, which was limited to males, reported a weaker association between early age at first alcohol use and risk for problem drinking in AA vs. EA men (Horton, 2007). An even more pronounced difference was observed in our study: a statistically significant increase in risk for developing problem use was observed in European Americans

but not African Americans who began drinking before age 15. However, the protective effect of late initiation of alcohol use was found in both groups.

Twin Models of Age at First Drink and Problem Alcohol Use

Although tests of model fit indicated that estimating variance components separately for the AA and EA subsamples did not provide a better fit to the data, we are focusing our discussion on the distinctions between the two groups for two reasons. First, there is a paucity of research on differences between African Americans and European Americans in the relative genetic and environmental contributions to alcohol-related phenotypes, so exploratory as they are, our analyses provide a rare window into underlying sources of variance on initiation and problem drinking in African Americans. Second, the fact that the twin models conducted with the full sample roughly mirrored the EA models despite substantial differences in estimates from the separate models suggests that true differences may be masked when European Americans so far outnumber African-Americans in a study sample. Our finding that the best fitting single univariate model of problem use with separate estimates for the AA and EA subsamples was one in which the C estimate for European Americans was 18 or more times higher than the C estimate for African Americans supports this assertion.

In both the AA and EA models, the estimated proportion of variance in age at first drink accounted for by additive genetic influences fell within the (broad) range reported in the literature. The difference between the models, 44% vs. 26% for the AA and EA subsamples, respectively, is noteworthy, but given the absence of previous research in this area, it is not easily interpreted. A lower heritability estimate might be expected if age at first drink is a robust marker for genetic liability to a *highly heritable* problem drinking phenotype in European Americans but not in African Americans. However, the estimated heritability of problem use in the EA model was a modest 21%, compared with estimates of 50–60% for the more severe phenotype of alcohol dependence reported in previous studies based on primarily EA individuals (Heath et al., 1997; Kendler et al., 1994; Knopik et al., 2004; Reed et al., 1996; van den Bree et al., 1998). Also unlike AD, the problem use phenotype in our sample did not share a substantial degree of genetic variance with alcohol use initiation. Our results therefore cannot be used to address this hypothesis, nor can the existing literature, as the heritability of AD in African Americans has not been documented.

The most remarkable distinction across ethnic groups in the relative genetic and environmental influences on the two phenotypes was the absence of shared environmental influences on problem alcohol use in the AA subsample vs. the attribution of 33% of the variance in problem alcohol use to shared environment in the EA subsample. The best-fitting model for problem use in the EA subsample was more similar to models of age at first drink than models of severe problem drinking (i.e., AD) reported in previous studies, with shared environment playing a larger role than genetics. The heritability estimate of 21% is much lower than the range of 50–60% reported for AD models in the existing literature, which do not include any shared environmental effects. By contrast, the AA model more closely resembled AD models from the larger literature in its absence of shared environmental influences and its substantial (but slighter lower) heritability estimate of 40%. It was also more similar than the EA model to findings reported in the few prior twin studies examining problem use, which produced heritability estimates of 41% for “getting into a situation you later regretted” (Fowler et al., 2007) and 78% for one or more AUD symptoms (Rhee et al., 2003). Of note, both Fowler et al.’s and Rhee et al.’s studies were conducted with adolescents, for whom there are significant environmental constraints on heavy use and even minor alcohol-related consequences are highly deviant. Environmental constraints on problem alcohol use may exert themselves in African Americans as well.

One likely explanation for the observed distinctions across ethnic groups is a difference in cultural drinking norms, such that among African Americans, for whom problem drinking is less common (Horton, 2007, Skidmore et al., 2012, Wu et al., 2011), alcohol-related problems are driven more by genetic than environmental influences. Although the research on ethnic differences in attitudes toward drinking is sparse, there are some findings in the larger literature that support this hypothesis. For example, stronger parental disapproval of substance use and more conservative attitudes about women's drinking have been reported in African Americans vs. European Americans (Beck et al., 1995; Gillmore et al., 1990; Herd, 1997). There is also consistent evidence for the protective effects of religiosity against alcohol use and misuse (Bahr and Hoffman, 2010; Brown et al., 2001; Michalak et al., 2006; Wills et al., 2003) and religious involvement is more prevalent in African Americans than European Americans (Johnston et al., 1999; Kosmin and Keysar, 2009; Regnerus et al., 2003). The association between religiosity and reduced risk for problem drinking specifically among African Americans has been demonstrated as well (Stevens-Watkins et al., 2010), including in an earlier study based on the current sample (Heath et al., 1999). Furthermore, a high level of ethnic identification has been associated with greater disapproval of substance use, lower intentions to use drugs, lower rates of perceived peer use, and lower rates of alcohol use in African Americans (Chartier et al., 2009; Corneille et al., 2007; Pugh and Bry, 2007; Stock et al., 2012). In short, the differences between the AA and EA subsample twin models of problem use may reflect a protective effect of cultural norms against alcohol-related problems in African Americans, at least in early adulthood. The implication of these findings for European Americans is that, compared to African Americans, the environments to which they are exposed (e.g., peer, school, family) are contributing to a greater degree to problem drinking. Interventions aimed at altering environmental influences, for example, targeting parental attitudes toward alcohol use, or modifying perceptions of peer alcohol use (e.g., Neighbors et al., 2004) may be effective ways of shifting environmental influences such that they become protective against alcohol misuse.

Limitations

Findings from the current study should be interpreted with certain limitations in mind. First, as indicated by the wide confidence intervals on variance component estimates, the model derived from analyses conducted with the AA subsample is somewhat unstable and should therefore be treated as suggestive rather than definitive. Second, the absence of statistical significance between the equated vs. the separate AA and EA models should be considered when comparing findings across ethnic groups, but not without weighing in results of the additional univariate analyses of problem alcohol use suggesting the absence of significance is likely due at least in part to low power. Third, given our interest in examining the association of initiation of alcohol use with problem drinking, which is contingent on having consumed alcohol, we excluded lifetime abstainers, and African Americans were overrepresented in this group (13.2% of African-American vs. 5.5% of European-American MOAFTS participants). Our conclusions regarding the relative contributions of genetic and environmental factors to the timing of first drink therefore may not generalize to the phenotype of any lifetime use. Fourth, although minimized by the short lag time from first use and/or symptom onset to time of interview (and the use of the first report when more than one report was available), the potential bias introduced by retrospective reporting should be considered, particularly if it varies by ethnicity.

Future Directions

Uncovering the genetic and environmental underpinnings of early alcohol use and problem drinking across ethnic groups is essential to refining etiological models of the course of AUDs in order to develop culturally appropriate intervention strategies. The next logical

step in this line of research is to apply similar methods to samples that include a wider range of ethnic groups as well as males. By doing so, the groundwork will be laid for future investigations to incorporate measured genes and measured environmental risk and protective factors associated with various drinking stages into studies of ethnic differences in the course of AUD development.

Acknowledgments

This study was funded by grants AA17921, AA09022, AA11998, AA17688, AA17915, AA011998_5978, and AA12640 from the National Institute on Alcohol Abuse and Alcoholism, and grant HD049024 from the National Institute of Child Health and Human Development.

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Figure 1.
Bivariate Cholesky Decomposition of Age at First Drink and Problem Alcohol Use: African Americans Only
Unstandardized path estimates shown

Figure 2.
 Bivariate Cholesky Decomposition of Age at First Drink and Problem Alcohol Use:
 European Americans Only
 Unstandardized path estimates shown

Figure 3.
 Bivariate Cholesky Decomposition of Age at First Drink and Problem Alcohol Use: AAs
 and EAs Equated
 Unstandardized path estimates shown

Table 1

Distribution of age at first drink and problem use by ethnicity

	African-Americans (n=480)			European-Americans (n=3,052)		
	Prevalence	Meet Criteria for Problem Use	Odd Ratio* (95% CI)	Prevalence	Meet Criteria for Problem Use	Odd Ratio* (95% CI)
Early (15)	26.6%	46.8%	1.42 (0.84–2.40)	41.0%	58.5%	1.74 (1.46–2.07)
Average (16–17)	26.0%	38.2%	-----	32.6%	44.8%	-----
Late (18)	47.4%	23.7%	0.50 (0.31–0.81)	26.4%	20.2%	0.31 (0.25–0.39)
Across Age Categories		33.1%			43.8%	

Table 2

Bivariate models: estimated proportion of variance accounted for by additive genetic (A), shared environmental (C), and unique environmental (E) influences on age at first drink and problem use, shown with 95% confidence intervals

Age at 1 st drink			Problem Alcohol Use			Correlations		
A	C	E	A	C	E	r _A	r _C	r _E
African-American Subsample								
0.26 (0.07–0.69)	0.25 (0.00–0.46)	0.49 (0.31–0.68)	0.41 (0.14–0.65)	-----	0.59 (0.35–0.86)	0.29 (0.17–0.41)	-----	-----
European-American Subsample								
0.44 (0.26–0.63)	0.27 (0.10–0.43)	0.29 (0.24–0.34)	0.21 (0.01–0.48)	0.33 (0.10–0.43)	0.46 (0.37–0.54)	0.20 (0.03–0.37)	0.16 (0.01–0.30)	0.08 (0.03–0.12)
Full Sample								
0.49 (0.32–0.66)	0.21 (0.05–0.35)	0.30 (0.26–0.36)	0.34 (0.17–0.59)	0.20 (0.06–0.34)	0.46 (0.39–0.55)	0.35 (0.30–0.41)	-----	0.06 (0.02–0.10)