Socioeconomic Status Moderates Genetic and Environmental Effects on the Amount of Alcohol Use

Nayla R. Hamdi, Robert F. Krueger, and Susan C. South

Background: Much is unknown about the relationship between socioeconomic status (SES) and alcohol use, including the means by which SES may influence risk for alcohol use.

Methods: Using a sample of 672 twin pairs (aged 25 to 74) derived from the MacArthur Foundation Survey of Midlife Development in the United States, this study examined whether SES, measured by household income and educational attainment, moderates genetic and environmental influences on 3 indices of alcohol use: amount used, frequency of use, and problem use.

Results: We found significant moderation for amount of alcohol used. Specifically, genetic effects were greater in low-SES conditions, shared environmental effects (i.e., environmental effects that enhance the similarity of twins from the same families) tended to increase in high-SES conditions, and nonshared environmental effects (i.e., environmental effects that distinguish twins) tended to decrease with SES. This pattern of results was found for both income and education, and it largely replicated at a second wave of assessment spaced 9 years after the first. There was virtually no evidence of moderation for either frequency of alcohol use or alcohol problems.

Conclusions: Our findings indicate that genetic and environmental influences on drinking amount vary as a function of the broader SES context, whereas the etiologies of other drinking phenomena are less affected by this context. Efforts to find the causes underlying the amount of alcohol used are likely to be more successful if such contextual information is taken into account.

Key Words: Alcohol, Socioeconomic Status, Gene-by-Environment Interaction.

AS ONE OF the top 10 risk factors for death, disease, and disability (WHO, 2011), alcohol use can be a financial burden on society with economic costs ranging from 1.3 to 3.3% of gross domestic product in middle- and high-income countries (Rehm et al., 2009). Informed policy making and effective intervention are crucial, and both could benefit from research on the genetic and environmental determinants underlying alcohol use. Unfortunately, specific causes have been elusive so far. One reason for this etiological indeterminacy may be that genetic and environmental influences vary by context. For example, a growing literature is showing that the heritability of alcohol use—the proportion of variation in alcohol use explained by genetic factors—is greater in adolescents with more alcohol-using peers (Dick et al., 2007), in girls with less parental closeness (Miles et al., 2005), in urban areas as opposed to rural ones (Legrand et al., 2008; Rose et al., 2001), in females without a religious upbringing compared to ones with such an upbringing (Koopmans et al., 1999), and in unmarried women compared to married women (Heath et al., 1989). These findings have been reported for several different alcohol use variables, including frequency of use, problem use, amount used, and any alcohol used.

When the heritability of a trait varies along a measured environmental dimension (e.g., number of alcohol-using peers), this is a form of gene-by-environment (G × E) interaction known as moderation. The majority of studies exploring G × E in alcohol use seem to find that genetic effects are larger in environments that either increase risk for alcohol use or are less restraining (Young-Wolff et al., 2011). But more research of this kind is needed to confirm existing findings and to uncover additional dimensions that may moderate the etiology of alcohol consumption. This study investigated whether socioeconomic status (SES) moderates total genetic and environmental influences on alcohol use.

Previous studies have begun examining whether genetic and environmental effects on alcohol use vary by education, a commonly used indicator of SES. For example, Latvala and colleagues (2011) examined moderation for maximum alcoholic drinks consumed within 24 hours and found that environmental influences not shared between twins decreased with years of education, whereas shared environmental influences followed a u-shaped pattern. Additionally, Timberlake and colleagues (2007) reported that college attendance enhanced genetic effects on quantity of alcohol consumed. But this result may contrast with findings from adoption studies (e.g., Sigvardsson et al., 1996), which indicate that a genetic predisposition for a common type of alcoholism predicts a severe form of this disorder only in low-SES environments. Specifically, adoptees with a genetic predisposition

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were at elevated risk of severe alcoholism only if their adoptive fathers had an unskilled occupation. Admittedly, this study did not estimate the heritability of alcoholism but inferred adoptees' genetic risk based on their biological parents' histories. To our knowledge, no studies have examined whether socioeconomic variables besides education moderate total genetic and environmental influences on alcohol consumption. Additionally, no studies have investigated the moderating role of SES beyond adolescence and young adulthood.

To fill this gap in the literature, we examined whether SES, measured by household income and educational attainment, moderates genetic and environmental influences on 3 alcohol use variables, including amount of use, frequency of use, and alcohol problems, in a sample spanning all of middle adulthood.

MATERIALS AND METHODS

Participants

Participants in the current study came from a representative national, random-digit-dial sample of noninstitutionalized English-speaking adults aged 25 to 74 years. The sample was derived from the MacArthur Foundation Survey of Midlife Development in the United States (MIDUS) conducted in 1995 to 1996 to examine physical health, psychological well-being, and social responsibility throughout midlife. In 2004 to 2006, participants were reassessed. At both assessment waves, data were collected via a 30- to 45-minute phone interview and 2 self-administered questionnaires (SAQs).

A subsample of 998 twin pairs, formed by screening 50,000 nationally representative households, was the focus of this study. Approximately 15% of respondents identified a twin in the family, and 60% of those respondents gave the research team permission to contact the twin. For more information on twin recruitment in MIDUS, see Kendler and colleagues (2000). To determine zygosity, twins were queried about the similarity of their eye and hair color and the extent to which others had difficulty telling them apart. Past research has shown that this approach classifies over 90% of twins accurately (Krueger and Johnson, 2002; Lykken et al., 1990). Only 16 twin pairs were unclassifiable due to missing or indeterminate zygosity information. In addition to these twins, we excluded all opposite-sex pairs (n = 263) from the study. Fifty-two singletons did not complete the phone interview or SAQ, and another 42 were dropped from the sample because of missing data on their cotwins. This resulted in a sample size of 672 complete twin pairs, with 350 monozygotic (MZ) pairs and 322 dizygotic (DZ) pairs. Mean age in this sample was 45 years (SD = 12, range = 25 to 74), and 57% of participants were female. At the second assessment wave, 454 of the 672 twin pairs were reassessed (68% of original sample), including 240 MZ pairs and 214 DZ pairs. Mean age at this time was 54 years (SD = 12, range = 34 to 82).

Measures

Amount of Alcohol Use. Amount of alcohol use was assessed via the phone interview. At wave 1, alcohol use was measured as the typical number of drinks that participants had on days on which they drank, during the year in which they drank most. A drink was defined as either a bottle of beer, a wine cooler, a glass of wine, a shot of liquor, or a mixed drink. At wave 2, drinking amount was assessed in the same manner but referred to the past month. Participants who indicated that they never drink were given a score of "0." At wave 2, participants who stated that they did not drink in the past month were also given a score of "0." The distribution of this variable was right-skewed at both waves, so we transformed the variable to normalize its distribution. At wave 1, a natural-log transformation was most effective at normalizing the distribution, while at wave 2 a square-root transformation yielded the best result.

Frequency of Alcohol Use. Frequency of alcohol use was also assessed via the phone interview. At wave 1, participants were asked to indicate how often they typically had at least 1 drink during the year in which they drank most. Possible answers included: Every day, 5 or 6 days a week, 3 or 4 days a week, 1 or 2 days a week, 1 to 4 days a month, less than once a month, or "never drink." Drinking frequency was assessed in the same way at wave 2, except that the assessed period was the past month. Frequency of use was fairly normally distributed at wave 1 (skewness = 0.15) and did not require transformation. At wave 2, frequency of use was right-skewed and was therefore natural-log transformed.

Alcohol Problems. Alcohol problems were assessed via the SAQs, which were mailed to participants following the phone interview. The response rate was high, with over 90% of twins who completed the phone interview at wave 1 returning the initial set of SAQs and over 80% of twins who completed the phone interview at wave 2 returning the second set of SAQs. Alcohol problems were assessed with 7 items inquiring about abuse or dependence symptoms within the past 12 months, such as having a strong desire to use alcohol, using alcohol in hazardous situations, and experiencing emotional problems from alcohol. Six of the 7 items were available at wave 2. A 1-factor principal axis analysis was performed on all available items, with factor loadings ranging from a low of 0.52 to a high of 0.78. The resulting factor score was subjected to an inverse transformation (i.e., $1/x$) because this transformation was most effective at normalizing the distribution of the factor score.

Income. Income was assessed via the SAQs and was measured as total annual household income, including personal earnings, spouses' earnings, government assistance, social security, pensions, and investments. Maximum household income was capped at $300,000. Income was right-skewed at both waves and was consequently square-root transformed.

Educational Attainment. Education was assessed during the phone interview and was measured as the amount of schooling participants had completed. The measure consisted of 12 levels of schooling, with the lowest level equal to "No school/some grade school" and the highest level equal to "Ph.D., Ed.D., M.D., D.D.S., LL.B., LL.D., J.D., or other professional degree." Education was square-root transformed at both waves to normalize its distribution.

Analytic Plan

We analyzed the data using the extended univariate moderation model outlined by van der Sluis and colleagues (2012). We chose this model over the univariate model by Purcell (2002) because the latter can produce false-positive effects. The van der Sluis extended univariate model reduces the false-positive rate to conventional levels, but it can misspecify the location of moderation when the covariance between moderator and trait is being moderated. For this reason, we also ran our analyses with Purcell's bivariate moderation model, which directly tests for moderation of the covariance. Results from the bivariate model were largely consistent with the findings from the van der Sluis model that are reported in this paper, and any significant differences are noted below. Specific results for the bivariate moderation model are available from the first author upon request.

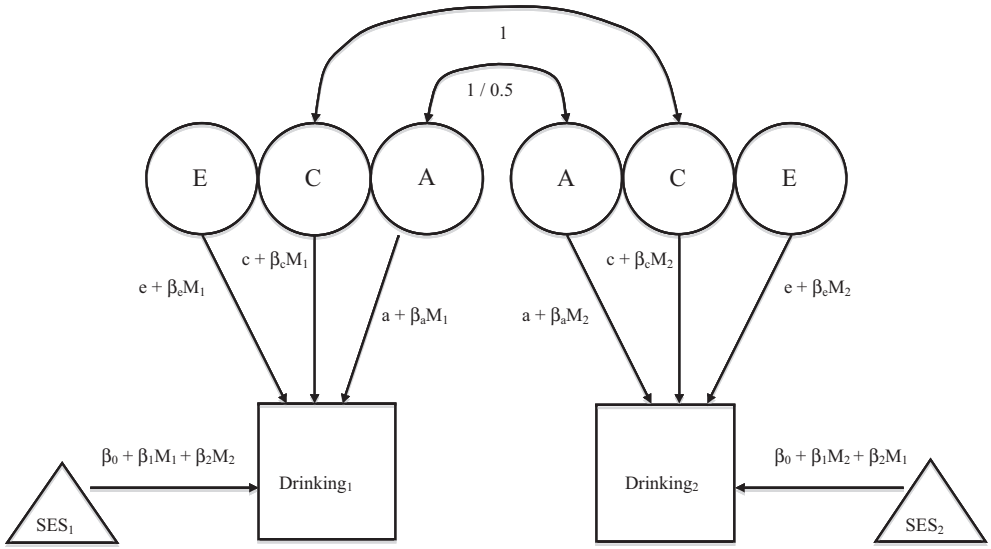

Fig. 1. van der Sluis extended univariate moderation model. The model is shown for both members of a twin pair. It allows genetic and environmental influences on drinking to vary by the moderator M, which is socioeconomic status (SES). *A*, *C*, and *E* represent residual variance in drinking after the variance in common with SES is regressed out. *A* represents influences due to additive genetics, *C* captures common/shared environmental influences, and *E* captures nonshared environmental influences. SES can moderate variance underlying drinking through β_a, β_c, and β_e, which index the direction and magnitude of genetic and environmental moderation. When these β coefficients are set to zero, this represents no moderation effects.

Figure 1 shows the van der Sluis extension of the univariate moderation model for SES and drinking. The figure is depicted for both twins in a twin pair. In this extended univariate model, variance shared between drinking and SES is partialled out of drinking by regressing twins' drinking values on their cotwins' SES values in addition to their own SES values (e.g., with the formula $\beta_0 + \beta_1 M_1 + \beta_2 M_2$, where M_1 is Twin 1's moderator and M_2 is Twin 2's moderator and where the β coefficients are estimated separately for MZ twins and DZ twins). The residual variance in drinking is then decomposed into its *ACE* components. *A* captures additive genetic influences, *C* represents environmental influences that make members of the same family similar, and *E* contains environmental influences that distinguish family members. Of note, variance in drinking is allowed to vary by the moderator, SES. For example, in the formula $a + \beta_a M_1$, a is an intercept capturing genetic effects on drinking, β_a reflects moderation of these genetic effects, and M_1 is the level of the moderator for Twin 1.

To test for moderation, we compared the above moderation model against a no-moderation model that fixes β_a, β_c, and β_e to zero so that genetic and environmental effects on drinking do not vary by SES. Two fit indices were used to compare the 2 models: the log-likelihood ratio test (LLRT) and Akaike information criterion (AIC). LLRT equals the difference between the $-2\ln(L)$ values of the 2 models and is distributed as a chi-square. A statistically significant chi-square indicates that the moderation model fits the data significantly better than the no-moderation model. The formula for AIC is $2k - 2\ln(L)$, where k denotes the number of parameters in the model. Smaller values of AIC indicate a better fit.

All analyses were conducted in the statistical program Mx, using maximum likelihood estimation. Biometric models were fit to the transformed alcohol and SES variables, with age, age^2, sex, sex*age, and sex*age^2 included as predictors in the model. When evidence of moderation was found for wave 1 measures of SES and drinking, we attempted replication at wave 2. Wave 2 measures were thus used to determine the robustness of wave 1 findings. Because the MIDUS sample spans a wide age range, we conducted age group analyses to investigate whether significant findings were consistent across age. We also ran additional analyses in which we left our variables untransformed or subjected them to a different transformation (most frequently a square-root transformation instead of a natural-log transformation, or vice versa). Results were robust in the sense that findings emerging as statistically significant under the original transformation almost always remained significant in these subsequent analyses, and moderation patterns showed little change across the different analyses. Thus, the findings reported in this paper were significant across transformations (and lack of transformation). Complete results are available from the first author upon request.

RESULTS

Descriptive Statistics

We computed descriptive statistics for all wave 1 measures of alcohol use, income, and education. The mean amount of alcohol that participants had on a typical drinking occasion during the year in which they drank most was 3 drinks (SD = 3, range = 0 to 30), and they drank alcohol an average of 1 to 2 days per week. Twenty-two percent of respondents admitted to having used more alcohol than intended within the past 12 months, but only 3% reported having had such a strong desire to use alcohol that they could not resist it or think of anything else. Mean household income was $73,484 (SD = 60,145, range = 0 to 300,000), and the median level of schooling was some college without degree attainment. Table 1 shows the correlations among all alcohol and SES measures. When income and education were categorized into 3 levels (low, moderate, high), mean drinking frequency increased across levels of income and education ($p < 0.01$). Mean drinking amount and mean alcohol problems did not vary along levels of income or education ($p > 0.05$).

Table 1. Correlation Matrix for Wave 1 Alcohol and Socioeconomic Status Measures

	Drinking amount	Drinking frequency	Alcohol problems	Income	Education
Drinking amount	1	0.70**	0.37**	0.06*	−0.01
Drinking frequency		1	0.46**	0.12**	0.08**
Alcohol problems			1	0.06*	0.03
Income				1	0.35**
Education					1

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

Table 2 shows twin correlations and univariate *ACE* estimates for all variables. We decided to retain the full *ACE* model for all alcohol variables despite *C* being (essentially) zero because this does not preclude the possibility of *C* moderation at low or high levels of SES.

van der Sluis Extended Univariate Moderation Model at Wave 1

At wave 1, the moderation model for drinking amount fit better than the no-moderation model according to both LLRT and AIC, and this was found when income was the moderator and when education was the moderator (see Table 3). For drinking frequency and alcohol problems, the no-moderation model almost always fit better. Although AIC suggested that education might moderate genetic or environmental effects on drinking frequency, LLRT was not significant. The bivariate moderation model found no evidence that education moderates the etiology of drinking frequency, so we do not interpret this result here. Table 4 shows the effects of age and sex on drinking amount, as well as the moderation parameters (β_a, β_c, and β_e) estimated for drinking amount. In the analysis with income at wave 1, the confidence interval around β_c indicates that this parameter differs significantly from zero. In the analysis with education at wave 1, β_e was significant.

Below, we present plots of the moderation estimates for drinking amount. We chose to plot the model estimating all moderation parameters as opposed to submodels estimating only a subset of moderation parameters because fixing some parameters to be exactly zero can bias the estimation of other parameters. All figures were plotted for −2 to 2 standard deviations from the moderator mean, which was well within the range of both income and education. Figure 2 shows the unstandardized (*A*) and standardized (*B*) moderation models with income as moderator. The former model allows the variance of drinking amount to change by moderator level, whereas the latter model fixes the variance to 1 at each moderator level. Figure 2*A* shows that, as income increases, genetic effects on drinking amount decline sharply; shared

Table 3. Model Comparison Fit Statistics

	−2ln(*L*)	df	χ^2	Δdf	*p*	AIC
Drinking amount and income						
No-moderation	2809.13	1,092				625.13
Moderation	2794.83	1,089	14.30	3	0.003	616.83
Drinking amount and education						
No-moderation	3361.61	1,300				761.61
Moderation	3339.41	1,297	22.20	3	<0.001	745.41
Drinking frequency and income						
No-moderation	2865.69	1,102				661.69
Moderation	2863.32	1,099	2.37	3	0.501	665.32
Drinking frequency and education						
No-moderation	3451.78	1,313				825.78
Moderation	3444.24	1,310	7.54	3	0.057	824.24
Alcohol problems and income						
No-moderation	2992.76	1,088				816.76
Moderation	2990.36	1,085	2.40	3	0.495	820.36
Alcohol problems and education						
No-moderation	3328.18	1,208				912.18
Moderation	3325.12	1,205	3.06	3	0.382	915.12
Wave 2 drinking amount and income						
No-moderation	1588.11	585				418.11
Moderation	1581.71	582	6.40	3	0.094	417.71
Wave 2 drinking amount and education						
No-moderation	2391.08	884				623.08
Moderation	2376.82	881	14.26	3	0.003	614.82

Smaller AIC values indicate better model fit.

−2ln(*L*), −2 log likelihood; df, degrees of freedom; χ^2, difference in −2ln (*L*) between no-moderation and moderation models; Δdf, difference in df between no-moderation and moderation models; *p*, probability value; AIC, Akaike information criterion.

Table 2. Twin Correlations and Univariate *ACE* Estimates for Wave 1 Measures of Alcohol Use and Socioeconomic Status

Measure	*r*MZ *r* (95% CI)	*r*DZ *r* (95% CI)	*A* % (95% CI)	*C* % (95% CI)	*E* % (95% CI)
Drinking amount	0.59 (0.51 to 0.65)	0.30 (0.20 to 0.40)	61 (42 to 67)	0 (0 to 17)	39 (33 to 46)
Drinking frequency	0.58 (0.50 to 0.64)	0.29 (0.18 to 0.38)	55 (33 to 63)	2 (0 to 22)	43 (37 to 50)
Alcohol problems	0.38 (0.28 to 0.47)	0.18 (0.06 to 0.29)	37 (9 to 46)	0 (0 to 23)	63 (54 to 73)
Income	0.33 (0.23 to 0.43)	0.23 (0.12 to 0.35)	16 (0 to 41)	16 (0 to 35)	67 (58 to 78)
Education	0.68 (0.62 to 0.73)	0.54 (0.46 to 0.62)	41 (25 to 57)	31 (16 to 45)	28 (24 to 33)

*r*MZ, intra-class correlation for monozygotic twin pairs; *r*DZ, intra-class correlation for dizygotic twin pairs; *A*, additive genetic variation; *C*, common/shared environmental influences; *E*, nonshared environmental influences; 95% CI, 95% confidence interval.

Table 4. Moderation Parameters Estimated from Models with Income and Education Moderating Drinking Amount

	β_{sex} (95% CI)	β_{age} (95% CI)	β_{age2} (95% CI)	$\beta_{sex*age}$ (95% CI)	$\beta_{sex*age2}$ (95% CI)	β_a (95% CI)	β_c (95% CI)	β_e (95% CI)
Wave 1 income moderating	−0.27	−0.20	−0.07	−0.05	0.00	−0.16	0.17	−0.02
Drinking amount	(−0.36 to −0.18)	(−0.27 to −0.13)	(−0.14 to −0.01)	(−0.12 to 0.03)	(−0.06 to 0.06)	(−0.27 to 0.01)	(0.03 to 0.29)	(−0.07 to 0.04)
Wave 1 education moderating	−0.28	−0.18	−0.08	−0.05	0.00	−0.09	0.14	−0.06
Drinking amount	(−0.36 to −0.19)	(−0.24 to −0.11)	(−0.14 to −0.02)	(−0.11 to 0.02)	(−0.06 to 0.06)	(−0.19 to 0.02)	(−0.25 to 0.25)	(−0.10 to −0.02)
Wave 2 income moderating	−0.16	−0.15	0.01	0.10	0.00	−0.07	0.00	−0.04
Drinking amount	(−0.28 to −0.04)	(−0.26 to −0.05)	(−0.07 to 0.10)	(0.00 to 0.20)	(−0.09 to 0.09)	(−0.20 to 0.06)	(−0.19 to 0.19)	(−0.13 to 0.05)
Wave 2 education moderating	−0.10	−0.17	−0.03	0.05	0.00	−0.16	0.00	0.05
Drinking amount	(−0.20 to 0.00)	(−0.25 to −0.08)	(−0.10 to 0.04)	(−0.03 to 0.13)	(−0.07 to 0.06)	(−0.25 to −0.06)	(−0.23 to 0.23)	(−0.02 to 0.12)

The β coefficients for sex, age, age^2, sex*age, and sex*age^2 show the effects of age and sex on drinking amount. The remaining β coefficients show the degree to which income and education moderate genetic and environmental effects on drinking amount after all variance that is shared with the moderator (i.e., income or education) is removed. β_a shows moderation of additive genetic effects, β_c indicates moderation of shared environmental effects, and β_e captures moderation of nonshared environmental effects. For clarity of interpretation, the signs on β_a, β_c, and β_e were reversed whenever the intercept was negative.

environmental effects increase significantly, and nonshared environmental effects decline slightly with higher levels of income. Total phenotypic variance, equal to the sum of *A*, *C*, and *E*, also declines with increasing levels of income. Figure 2*B* expresses these genetic and environmental influences as proportions of the total variance in drinking amount. Figure 3 depicts the same information with education as the moderator. Mirroring the pattern observed for income, genetic and nonshared environmental influences on drinking amount decrease, while shared environmental influences increase with greater education, although to a lesser degree. Again, phenotypic variance declines with increasing education.

Wave 2 Extension

We examined whether our findings for drinking amount replicated at wave 2. At this time, participants reported drinking, on average, 1 drink per drinking occasion in the past month (SD = 1, range = 0 to 10). Mean household income was $71,159 (SD = 56,735, range = 0 to 300,000), and median education was still some college without degree attainment. Drinking amount correlated 0.18 with income and 0.06 with education, while income and education correlated 0.36. Mean drinking amount increased across low, moderate, and high levels of income ($p < 0.01$) but did not differ across education levels ($p > 0.05$). The *ACE* components of income and education were virtually unchanged at wave 2. Drinking amount, however, had a slightly reduced genetic etiology (0.52, 95% CI: 0.38 to 0.60) and an increased nonshared environmental etiology (0.48, 95% CI: 0.40 to 0.58) at wave 2 compared to wave 1. Table 3 shows that the moderation model fit well at wave 2, with the exception that LLRT for income moderating drinking amount was only marginally significant. Still, AIC showed evidence for moderation, and the bivariate moderation model provided statistically significant evidence for moderation.

Table 4 shows that none of the estimated moderation parameters at wave 2 differed significantly from zero when income was the moderator, even though the overall moderation model fit well. When education was the moderator, β_a differed significantly from zero. Figure 4 depicts the unstandardized full moderation models for income and education moderating drinking amount at wave 2. As was observed at wave 1, genetic influences declined with greater income and education. Shared environmental influences were flat around zero. Nonshared environmental influences decreased with greater income but increased with greater education. Total phenotypic variance declined with increasing income and education.

Age Group Analyses

To examine whether our results for drinking amount held up across age groups, we split the sample at the median age (45 years at wave 1) and tested for moderation separately in

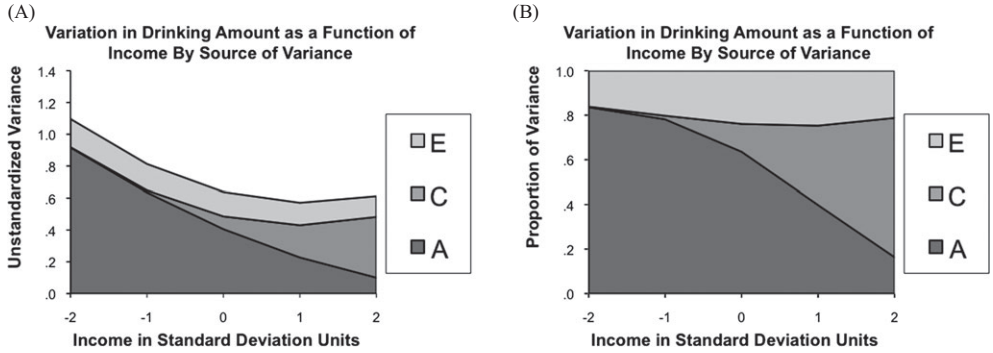

Fig. 2. Drinking amount and income. (**A**) Unstandardized variance in drinking amount from the moderation model with income. (**B**) Proportion of variance in drinking amount from the moderation model with income. *A* = additive genetic variance; *C* = common/shared environmental variance; *E* = nonshared environmental variance.

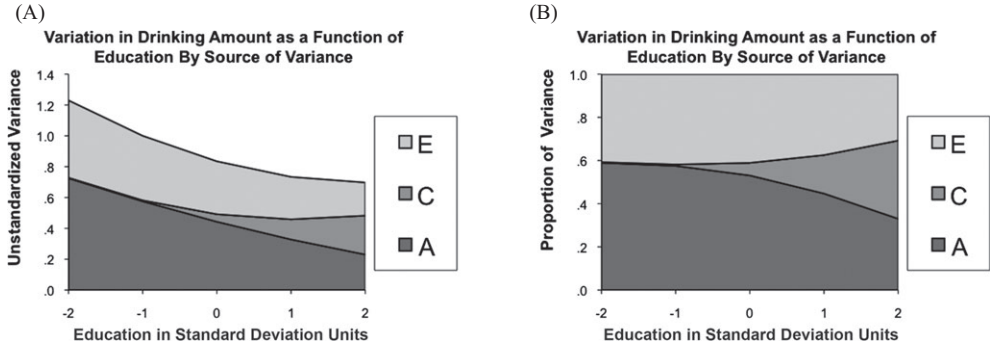

Fig. 3. Drinking amount and education. (**A**) Unstandardized variance in drinking amount from the moderation model with education. (**B**) Proportion of variance in drinking amount from the moderation model with education. *A* = additive genetic variance; *C* = common/shared environmental variance; *E* = nonshared environmental variance.

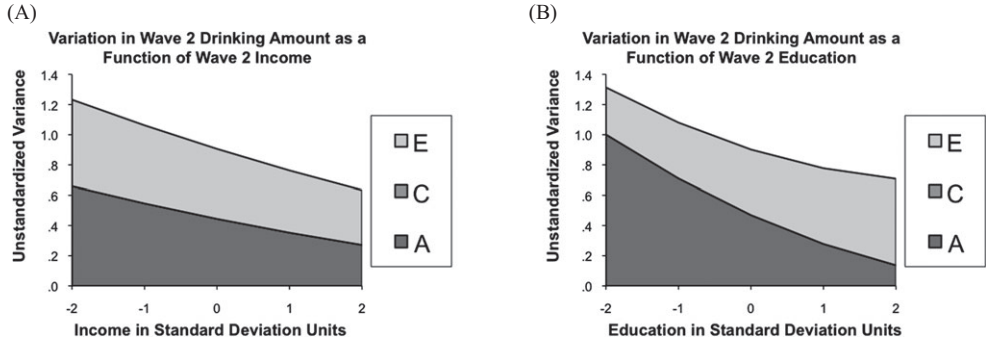

Fig. 4. Wave 2 drinking amount, income, and education. (**A**) Unstandardized variance in wave 2 drinking amount from the moderation model with wave 2 income. (**B**) Unstandardized variance in wave 2 drinking amount from the moderation model with wave 2 education. *A* = additive genetic variance; *C* = common/shared environmental variance; *E* = nonshared environmental variance.

each age group. At wave 1, there was significant moderation in all combinations of age groups and moderators, except for the younger age group when income was the moderator, where there was a trend (*p*-value for $\chi^2 < 0.1$). The original pattern of results observed at wave 1 replicated for both income and education, with the exception that shared environmental influences decreased with greater education in the older age group. At wave 2, the median age split resulted in samples that were generally too small to allow statistically significant detection of moderation; thus, the moderation model was significant only for the younger age group when education was the moderator, and it was marginally significant for the younger age group when income was the moderator. Still, when the parameter estimates from the moderation model were plotted, results showed once again that genetic influences declined with higher levels of SES.

Shared environmental influences were very small and fairly flat, but they declined slightly with higher education in the older age group. Nonshared environmental influences declined with greater income but increased with greater education.

DISCUSSION

This study examined whether SES moderated etiological influences on drinking amount, drinking frequency, and alcohol problems. Moderation was generally evident only for drinking amount. Genetic and nonshared environmental influences on drinking amount tended to decrease with greater SES, while shared environmental effects tended to increase, although the latter was observed only at the first wave of assessment and generally only in individuals below age 45. This general pattern of results was found for both income and education and largely replicated at a second wave of assessment spaced 9 years after the first.

Our results prompt the question why the moderating effects of SES are rather specific to drinking amount. Effects for alcohol problems may have been harder to detect because participants may have been reluctant to disclose alcohol-related problems. Still, this does not explain why effects were generally not found for drinking frequency. An alternative explanation is that SES is differentially related to specific facets of alcohol use, an explanation that is consistent with previous research (e.g., Casswell et al., 2003; Huckle et al., 2010). Thus, SES may moderate factors underlying the amount of alcohol consumed (e.g., ability to exercise restraint or metabolic factors) rather than risk for frequent use or problematic use.

We found that genetic variance in drinking amount is greatest in low-SES conditions, as is total phenotypic variance. Thus, individuals from low-SES environments vary considerably in the amount of alcohol they drink, and this variation is largely explained by genetic factors. In contrast, high-SES individuals vary less in the amount of alcohol they drink, and drinking in this environment is also influenced by familial environmental factors. Of note, there were few mean-level differences in drinking amount by SES. This means that SES does not explain much phenotypic variation in drinking amount, but the underlying determinants of drinking amount do depend on SES.

The results of this study are partly consistent with the diathesis-stress model, which posits that environmental stressors activate or trigger genetic vulnerabilities for undesirable outcomes. The model has been interpreted to indicate that total genetic influences are greater in more high-risk environments (Vendlinski et al., 2011). Our results align with this model to the extent that (i) amount of alcohol use is conceptualized as an undesirable outcome, (ii) low-SES environments are a trigger of its genetic diatheses, and (iii) these diatheses are the reason why total genetic variance in drinking amount is greatest in low-SES environments. Conversely, our results diverge from the diathesis-stress model in that the phenotypic correlation between drinking amount and SES is positive (albeit very small), contrary to what the model would predict. Overall, existing theoretical models for G × E are often limited in their focus on genetic liability for undesirable outcomes, whereby it is unclear how this genetic risk generalizes to total genetic variance. Additionally, most models do not consider that multiple processes could be unfolding simultaneously (e.g., while stressors in low-SES environments trigger genetic liabilities for drinking, familial customs prominent in high-SES environments could explain the variation in drinking observed in these settings). Additional empirical results are needed to build more comprehensive and nuanced theories of G × E.

Our results contrast with those from previous twin studies, which found that the heritabilities of maximum drinks (Latvala et al., 2011) and quantity of alcohol use (Timberlake et al., 2007) were greater among more educated individuals. At the same time, our findings resemble those of Latvala and colleagues (2011) in showing that nonshared environmental influences tend to decline with greater SES. Diverging findings may be due to differences in participant age, as we assessed adults across midlife, whereas Timberlake and colleagues (2007) and Latvala and colleagues (2011) assessed young adults. Another, related possibility is that education may either enhance or reduce genetic variance in alcohol use contingent on context (e.g., current college attendance may enhance genetic variance, whereas a history of greater educational attainment may reduce genetic variance, especially later in life). On the whole, our understanding of how G × E affects behavioral traits is still evolving, and it remains to be seen how different measures of SES either enhance or reduce genetic effects on related phenotypes.

Limitations

This study had several limitations. Because the amount and frequency of alcohol use were originally assessed for the year in which individuals drank the most, the timing of use is unspecified and varies by individual. Also unclear is how alcohol use relates to the moderators chronologically. In light of these facts, it is reassuring that our moderation results for drinking amount are essentially the same for income and education, 2 nonredundant measures of SES that refer to different time points. In addition, our results largely replicate at a second assessment wave, wherein the timing of alcohol use is well-defined (i.e., past month) and succeeds the timing of the SES measures. The similarity in results across time and measures of SES suggests that the observed moderation pattern is robust, is not due to a timing artifact, and captures effects that are common to SES rather than unique to a particular SES measure. Relatedly, the fact that our wave 1 alcohol measure refers to individuals' heaviest drinking year is simultaneously a strength because this likely maximizes phenotypic variance, which is optimal for an examination of G × E.

Another limitation is that there was 32% sample attrition at the second assessment wave. Still, this attrition makes it all the more compelling that the wave 2 pattern of results matched the wave 1 pattern fairly closely and was statistically significant despite the loss in power. One aspect of our original results that did not emerge at wave 2, however, was shared environmental moderation. The attenuation of this effect may be due to differences in the way in which drinking amount was measured at the 2 assessment waves. Specifically, our wave 1 measure inquired about typical drinking during the year in which individuals drank the most, whereas our wave 2 measure inquired about typical drinking during the past month. It is possible that SES moderates shared environmental effects on heavier drinking only. This would mean that, in high-SES environments, familial environmental factors explain a significant amount of variation in heavier drinking but less variation in more moderate drinking (while in low-SES environments, familial environmental factors explain little variation in either measure of drinking). Additionally, past month drinking may show a reduced shared environmental etiology because the past month refers to different time points for twins not interviewed at the same time. In this case, twins would be reporting on different sociocultural occasions for drinking, which could reduce our ability to detect shared environmental effects.

Finally, the sample size in this study was modest. As a result, we had limited power to detect small effects, and it is possible that we could have detected additional moderation effects (e.g., for drinking frequency or problematic alcohol use) in a larger sample. Future research should examine G × E interplay as it affects various alcohol use phenomena in a larger sample and with a longitudinal replication such as the one that we provided here.

Implications

Our main finding is that the etiology of drinking amount is not constant across all individuals in the population, but rather, varies as a function of the broader socioeconomic context. This finding has important implications for efforts to locate the genetic and environmental causes underlying how much alcohol is consumed—efforts that are more likely to be successful if contextual information is taken into account. Our results indicate that the likelihood of finding genes for the quantity of alcohol used is maximized in a low-SES sample, given the increased heritability in this group. In contrast, researchers wanting to identify familial environmental factors underlying drinking amount should consider studying a high-SES sample.

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