Socioeconomic Status Moderates the Etiology of Alcohol Use

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Abstract

This study examined whether socioeconomic status (SES), measured by household income and educational attainment, moderates genetic and environmental influences on alcohol use. We found that genetic effects were greater in low-SES conditions, while shared environmental effects (i.e., environmental effects that enhanced the similarity of twins from the same families) gained importance in high-SES conditions. This basic pattern of results was found for both income and education and replicated at a second wave of assessment spaced nine years after the first. Our findings indicate that the etiology of alcohol use varies as a function of the broader social context. Thus, efforts to find the causes underlying alcohol consumption are likely to be more successful if such contextual information is taken into account.

Keywords: alcohol, socioeconomic status, SES, gene-by-environment interaction, GxE

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Introduction

As one of the top 10 risk factors for death and the third leading risk factor for disease and disability, alcohol use can be very costly [1]. The total economic costs of alcohol range from 1.3% to 3.3% of GDP in middle- and high-income countries, with costs totaling 2.7% of GDP in the United States [2]. Given the significant economic and public health burdens associated with problem drinking, there is a clear need for informed policymaking and effective intervention. Knowledge of the specific genetic and environmental causes underlying alcohol use would be extremely valuable, but identification of specific causes has been challenging so far. One of the main reasons for this etiological indeterminacy may be that genetic and environmental influences vary based on contextual factors. For example, a growing literature is showing that the heritability of alcohol use—the proportion of variation in use that is explained by genetic factors—is greater in more urban living conditions [3,4], in adolescents with more alcohol-using peers [5], in girls with less parental closeness [6], in females without a religious upbringing compared to ones with such an upbringing [7], and in unmarried women compared to married women [8].

When genetic influences on a trait vary by circumstances separable from the trait per se, this is typically called a gene-by-environment (GxE) interaction (albeit the term 'environment' is slightly misleading in this context because virtually all so-called environments are at least partly genetically influenced). Three theories of individual differences posit GxE interaction: the diathesis stress model, the bioecological model, and the plasticity model. The diathesis-stress model proposes that a genetic vulnerability

interacts with a stressful environment to produce an undesirable outcome [9,10]. In this model, genetic influences are greatest in stressful or high-risk environments. In contrast, the bioecological model predicts that genetic influences are maximized in stable and adaptive environments [11]. Specifically, the model assumes that stable environments are best equipped to help individuals actualize their genetic potentials, as unstable environments may lack supportive resources or may even contain unique stressors that drown out genetic effects. Genetic influences would thus be maximized in the absence of adverse environmental effects. Finally, the plasticity model predicts that genetic influences are greatest on both ends of the environmental risk continuum. In particular, the model suggests that individuals who are more plastic are more responsive to both negative *and* positive environments [12]. Thus, those individuals may have the *worst* outcomes in negative environments yet enjoy the *best* outcomes in positive environments, and their outcomes will be most likely to reflect their genes in both types of environments.

The majority of studies implicate a diathesis-stress etiology in alcohol use, such that genetic influences on drinking are maximized in stressful or high-risk environments. But more GxE research is needed to confirm existing findings and to elucidate further the etiology of alcohol use. There are a few reasons why it is especially important to understand the role of socioeconomic status (SES) in alcohol use: SES has already been found to moderate the etiologies of health-related phenomena, such as internalizing problems [13], externalizing problems [14], and physical health problems [15]. This shows that SES *can* act as a moderator of etiological influences. Of greater relevance to

the current study, two articles have indicated that low SES interacts with a certain form of genetic risk in the prediction of severe alcohol abuse [16,17]; however, questions exist about the robustness and generalizability of this result [e.g., 18]. Research into the moderating role of SES in alcohol use would therefore clarify existing findings. Finally, information about SES is readily available and can be incorporated easily into research and intervention frameworks to fine-tune these efforts. Thus, evidence that SES plays a moderating role in alcohol use can have practical implications. For the above reasons, the present study investigates if SES, measured by household income and educational attainment, moderates genetic and environmental influences on alcohol use.

Methods

Participants

Participants in the current study came from a representative U.S. national, random-digit-dial sample of non-institutionalized, English-speaking adults between the ages of 25 and 74 years. The sample was derived from the MacArthur Foundation Survey of Midlife Development in the United States (MIDUS), which was conducted in 1995-1996 to examine physical health, psychological well-being, and social responsibility throughout midlife. In 2004-2006, participants were re-located and re-assessed. At both assessment waves, data were collected via a 30-45 minute phone interview and two self-administered questionnaires (SAQs) that combined to around 100 pages in length.

A subsample of 998 twin pairs, formed by screening 50,000 nationally representative households, was the focus of the current 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. Twin pairs were then invited to participate in the MIDUS study. For further information on twin recruitment, see [19]. 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 to zygosity diagnosis classifies over 90% of twins accurately [20,21].

Sixteen twin pairs were unclassifiable due to missing or indeterminate zygosity information. In addition to those twins, we excluded all opposite-sex pairs (*n*=263) from the current study. Fifty-two singletons did not complete the phone interview or SAQ, and another 42 were dropped from the sample because no data were available on their co-twins. This resulted in a sample size of 672 complete twin pairs, with 350 monozygotic (MZ) pairs and 322 same-sex dizygotic (DZ) pairs. The mean age in this sample was 45 years (*SD*=12, range=25-74), and 57% of participants were female. The sample resembled comparably aged individuals in the 1995 US census on important socioeconomic indicators. For example, 88% of individuals in our sample had completed high school (compared to 82% in the census) and 27% had earned a bachelor's degree (compared to 23% in the census).

At the second assessment wave, 454 of the 672 complete twin pairs were re-assessed (68% of our original sample), including 240 MZ pairs and 214 same-sex DZ pairs. The mean age at this time was 54 years (*SD*=12, range=34-82).

Measures

Alcohol Use. Participants' 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. At wave 2, the period assessed was the past month; otherwise drinking was assessed in the same manner. Participants who indicated that they never drank were given a score of "0." The average number of drinks in our sample was 3 (*SD*=3, range=0-30) at wave 1 and 1 (*SD*=1, range=0-10) at wave 2. The distribution of this variable was right-skewed at both waves, so we used a natural log transformation to approximate a normal distribution.

Educational Attainment. Participants provided information about their educational attainment during the phone interview. Education was measured as the amount of schooling that participants had completed. This 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." The variable was measured in the same way at waves 1 and 2. At both waves, the median level of schooling was some college without degree attainment. Education was square-root transformed at waves 1 and 2 to normalize its distribution.

Income. Information about income came from 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. Income 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. Mean household income was $73,484 (*SD*=60,145, range=0-300,000) at wave 1 and $71,159 (*SD*=56,735,

range=0-300,000) at wave 2. Household income was right-skewed at both waves and was consequently square-root transformed.

Analytic plan

Prior to beginning our analyses, we computed standardized residuals for all of our measures, regressing out the effects of age, age^2, sex, sex*age, and sex*age^2. To examine whether SES moderates the etiology of alcohol use, we compared a bivariate moderation model [22] against a no-moderation model. Figure 1 depicts the no-moderation model. This model is akin to a bivariate Cholesky decomposition and shows the additive genetic (A), common/shared environmental (C), and non-shared environmental (E) components underlying SES and Drinking. Shared environmental factors include experiences that make members of the same family similar (e.g., influences of the familial home), whereas non-shared environmental factors make family members different. In Figure 1, variance in SES is fully accounted for by its ACE components, whereas variance in Drinking is influenced by the ACE components underlying SES ("common" components) as well as its own "unique" components.

Figure 2 shows the bivariate moderation model. In this model, the six paths leading to Drinking allow genetic and environmental influences to vary as a function of SES. For example, in the formula $a_C+\beta_{Xac}M$, a_C is an intercept capturing genetic risk for Drinking that is in common with SES, β_{Xac} reflects the direction and size of moderation effects, and M is the level of the moderator (in this case, SES). The main difference between this model and the no-moderation model is that the latter one fixes all β coefficients to be equal to zero, so that genetic and environmental effects on Drinking do

not vary by the moderator variable. In both models, the total phenotypic variance in Drinking can be computed by squaring, and then summing, all of the paths that lead to Drinking.

To determine whether the moderation model or the no-moderation model provides a better fit to the data, we used two fit indices: The Log-likelihood Ratio Test (LLRT) and Akaike Information Criterion (AIC). LLRT is equal to the difference between the -2ln(L) values of the two models, and it is distributed as a *chi-square.* A statistically significant *chi-square* indicates that the moderation model provides a significantly better fit to the data. 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; thus, k acts as a penalty for excess parameters. This means that if the no-moderation model and the moderation model had equal ln(L) values, AIC would favor the no-moderation model because it is more parsimonious.

Results

Table 1 shows the ACE decompositions for Drinking, income, and education from preliminary univariate analyses. As can be seen in the table, variation in Drinking was primarily genetic, with a moderate non-shared environmental influence. Income had a largely non-shared environmental etiology with small genetic and shared environmental contributions, while education was made up of almost equal genetic, shared environmental, and non-shared environmental components. Income and education were correlated 0.34, suggesting that they are related, but not redundant, measures of SES. For this reason, their moderating effects on Drinking were tested in separate analyses. The

correlation between income and Drinking was very small (r = -0.01), indicating little shared variance. The same was true of the correlation between education and Drinking (r = -0.07). The advantage of the moderation model tested here is that it computes genetic and environmental correlations between Drinking, on the one hand, and income and education, on the other, so it is possible to examine moderation effects while controlling for gene-environment correlation; but, because these correlations parse little shared variance in this case, they are not readily interpretable. For this reason, we do not report genetic and environmental correlations here.

As shown in Table 2, LLRT and AIC indicate that the moderation model provides a better fit to the data than the no-moderation model, and this is the case for both income and education. Below, we present results for the full moderation model, which allows free estimation of all six moderation parameters (i.e., all β coefficients in Figure 2). We choose to focus on the full model, as opposed to a model that estimates only some moderation effects, because fixing some parameters to be exactly zero can bias the estimation of other parameters.

Figure 3 shows the no-moderation model (3a), the unstandardized moderation model with income as moderator (3b), and the standardized moderation model with income as moderator (3c). The standardized moderation model fixes the variance of Drinking to equal 1 at each level of the moderator, whereas the unstandardized model allows the variance to change by moderator level. In Figure 3a, the genetic and environmental influences on Drinking do not vary by level of income, consistent with the absence of moderation effects. Here, genetic factors account for slightly less than 60% of

the variance in Drinking, non-shared environmental factors account for about 40%, and shared environmental influences are close to zero. Figure 3b shows what happens when the ACE parameters are allowed to vary with income: As income increases, genetic effects on Drinking decline sharply; shared environmental effects decrease initially but then increase significantly, and non-shared environmental effects decline slightly with higher levels of income. Figure 3c expresses these genetic and environmental influences as proportions of the total variance in Drinking. Figure 4 depicts the same information with education as the moderator. Mirroring the pattern observed for income, genetic and non-shared environmental influences on Drinking decrease while shared environmental influences increase with greater education, although to a lesser degree.

To test the robustness of our results, we examined if they replicated for wave 2 measures of Drinking, income, and education. LLRT and AIC indicated that the moderation model was superior to the no-moderation model for both income and education (Income: χ^2[df=6] = 14, p = 0.0257, AIC[moderation] = 922 compared to AIC[no-moderation] = 924; Education: χ^2[df=6] = 24, p = 0.0005, AIC[moderation] = 1201 compared to AIC[no-moderation] = 1213). In the no-moderation model, genetic influences accounted for approximately half of the variance in Drinking, while non-shared environmental influences accounted for the other half. Figure 5 depicts the unstandardized moderation models for income and education. Much of the basic pattern observed for wave 1 replicated at wave 2, with genetic influences declining sharply and shared environmental influences increasing, although only slightly, with greater income

and education. Non-shared environmental influences decreased with greater income but increased with greater education.

Discussion

The present study examined whether socioeconomic status, measured as household income and educational attainment, moderates etiological influences on alcohol use, measured as drinking amount. We found significant evidence of moderation, with genetic effects decreasing sharply and shared environmental effects increasing with greater socioeconomic status. This pattern of results was observed for income and education and at two waves of assessment spaced nine years apart.

Our findings are consistent with the diathesis-stress model, which posits enhanced genetic effects in stressful or high-risk environments. The assumption that this model makes is that an environmental stressor is necessary to trigger a genetic vulnerability; that is, this vulnerability will not be expressed in less stressful environments. Our results align with this model in that genetic influences were substantially greater in low-SES environments compared to high-SES environments, where these influences were small. According to our results, the etiology of alcohol use is overwhelmingly genetic in low-SES conditions. In contrast, shared environmental factors may be important in high-SES conditions, which suggests the influence of cultural reasons for drinking, particularly ones reflecting the rearing environment, in individuals of a high SES.

The results of this study are consistent with those of other GxE studies in implicating a diathesis-stress etiology in alcohol use. Indeed, previous studies have found that the heritability of alcohol use is greater in urban living conditions [3,4], in

adolescents with more alcohol-using peers [5], in girls with less parental closeness [6], in females without a religious upbringing compared to ones with such an upbringing [7], and in unmarried women compared to married women [8]. Additionally, a recent review of GxE in alcohol use concluded that "the relative importance of genetic influences on variance in drinking behaviors was greater in more permissive socio-cultural environments with easier access to alcohol and substance using peers, and lower in more restrictive social environments" [23, p. 803], in line with the diathesis-stress model. The fact that our study agrees with other biometric moderation studies in supporting a diathesis-stress account of alcohol use is reassuring, especially given that different GxE models seem to provide a better fit to other phenotypes. For example, genetic influences on adolescent antisocial behavior [14] and internalizing problems [13] have been found to be greater in socioeconomically advantaged environments, consistent with the bioecologial model. On the whole, our understanding of GxE in psychopathology is still evolving, but existing findings suggest that which type of gene-environment interaction is at play may depend on the particular phenotype under study.

Limitations

This study had a number of limitations. Because alcohol use was originally assessed for the year in which individuals drank the most, its specific timing is unspecified and varies by individual. Also unclear is how it relates to the moderators chronologically. In light of these facts, it is reassuring that our results are essentially the same for income and education, two non-redundant measures of SES that refer to different time points. In addition, our results 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 measures of SES and across time suggests that the observed pattern is robust, is not due to a timing artifact, and captures effects that are common to SES rather than unique to a particular measure of SES. On a related note, the fact that our wave 1 alcohol measure refers to individuals' heaviest drinking year is simultaneously a strength because this is likely to maximize phenotypic variance in drinking behavior, which is optimal for an examination of GxE effects.

Another limitation is that there was 32% attrition in our sample at the second wave of assessment. 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 strongly at wave 2, however, was evidence for shared environmental moderation. The attenuation of this effect may be due to differences in the way in which drinking was measured at the two assessment waves. In particular, our wave 1 measure inquired about typical drinking during the year in which individuals drank the most, whereas our wave 2 measures inquired about typical drinking during the past month. First, 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 large 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). Second, 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 on their drinking.

Implications

Our main finding is that the etiology of alcohol use is not constant across all individuals in the population, but rather, varies as a function of the broader social context. This result has important implications for efforts to find the specific genetic and environmental causes underlying alcohol consumption. Indeed, such efforts will be more successful if contextual information is taken into account. In particular, our results indicate that chances of finding risk genes for alcohol use are maximized in a low-SES sample, given the greater heritability of alcohol use in this population. In contrast, researchers wanting to identify familial environmental factors underlying alcohol consumption may increase their likelihood of success by studying a high-SES sample.

Table 1

Univariate ACE Decomposition

Measure	*A*	*C*	*E*
Drinking	61	0	39
Income	16	16	67
Education	41	31	28

Note. All numbers are percent values from univariate ACE decomposition models, where A captures additive genetic variation, C captures common/shared environmental influences, and E captures non-shared environmental influences.

Table 2

Model Comparison Fit Statistics

		-2ln(L)	*df*	*χ2*	*Δdf*	*p*	*AIC*
Income							
	No-moderation	6091	2222				1647
	Moderation	6070	2216	21	6	0.0019	1638
Education							
	No-moderation	6988	2641				1706
	Moderation	6961	2635	27	6	0.0001	1691

Note. -2ln(*L*) = -2 log likelihood; df = degrees of freedom; $\chi 2$ = 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.

Smaller AIC values indicate better model fit.

Figure Captions

Figure 1. No-moderation model.

Caption: Genetic and environmental influences on Drinking do not vary by socioeconomic status (SES). A represents influences due to additive genetics, C captures common/shared environmental influences, and E captures non-shared environmental influences. A_C, C_C, and E_C represent variance components underlying SES that also influence Drinking (i.e., "common" components), whereas A_U, C_U, and E_U represent residual ("unique") variance in Drinking after accounting for the variance in common with SES. The total phenotypic variance in Drinking can be calculated by squaring and summing all of the paths leading to it: $\text{Var(Drinking)} = (a_{C2})^2 + (a_{U2})^2 + (c_{C2})^2 + (c_{U2})^2 + (e_{C2})^2 + (e_{U2})^2$.

Figure 2. Bivariate moderation model.

Caption: Genetic and environmental influences on Drinking vary by socioeconomic status (SES). A represents influences due to additive genetics, C captures common/shared environmental influences, and E captures non-shared environmental influences. A_C, C_C, and E_C represent variance components underlying SES that also influence Drinking (i.e., "common components"), whereas A_U, C_U, and E_U represent residual ("unique") variance in Drinking after accounting for the variance in common with SES. Moderation of Drinking by SES is represented by a coefficient that indexes the direction and magnitude of moderation (e.g., β_{Xac}) multiplied by the level of the moderator (M). The total phenotypic variance in Drinking can be calculated by squaring and summing all of the paths leading to it: $\text{Var(Drinking)} = (a_C+\beta_{Xac}M)^2 + (a_U+\beta_{Xau}M)^2 + (c_C+\beta_{Xcc}M)^2 + (c_U+\beta_{Xcu}M)^2 + (e_C+\beta_{Xec}M)^2 + (e_U+\beta_{Xeu}M)^2$.

Figure 3. Drinking and income.

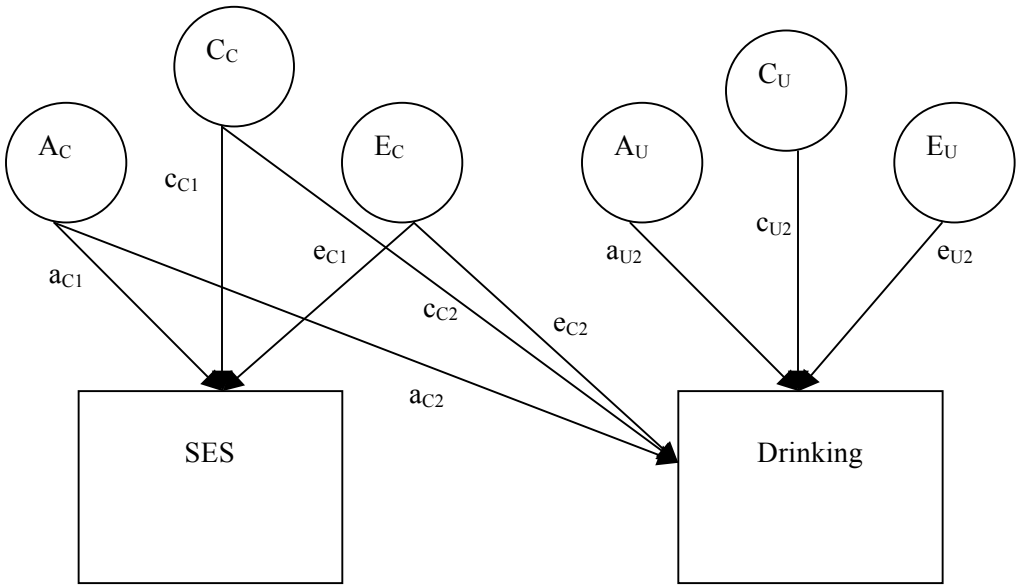
Caption: (a) Proportion of variance in Drinking from the no-moderation model with income. (b) Unstandardized variance in Drinking from the moderation model with income. (c) Proportion of variance in Drinking from the moderation model with income. A = genetic variance; C = common/shared environmental variance; E = non-shared environmental variance.

Figure 4. Drinking and education.

Caption: (a) Proportion of variance in Drinking from the no-moderation model with education. (b) Unstandardized variance in Drinking from the moderation model with education. (c) Proportion of variance in Drinking from the moderation model with education. A = genetic variance; C = common/shared environmental variance; E = non-shared environmental variance.

Figure 5: Wave 2 drinking, income, and education.

(a) Unstandardized variance in wave 2 Drinking from the moderation model with wave 2 income. (b) Unstandardized variance in wave 2 Drinking from the moderation model with wave 2 education. A = genetic variance; C = common/shared environmental variance; E = non-shared environmental variance.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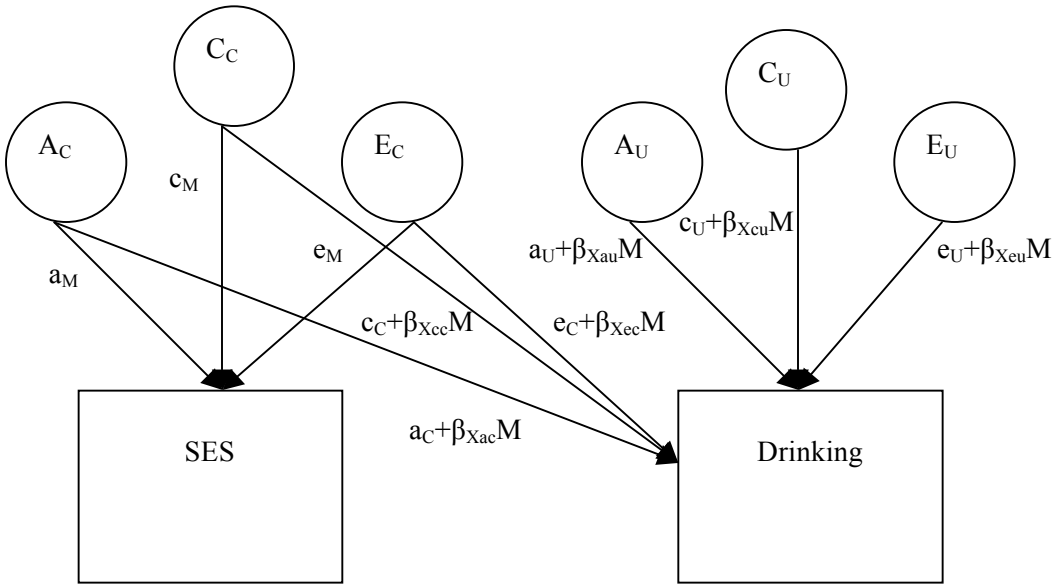
Drinking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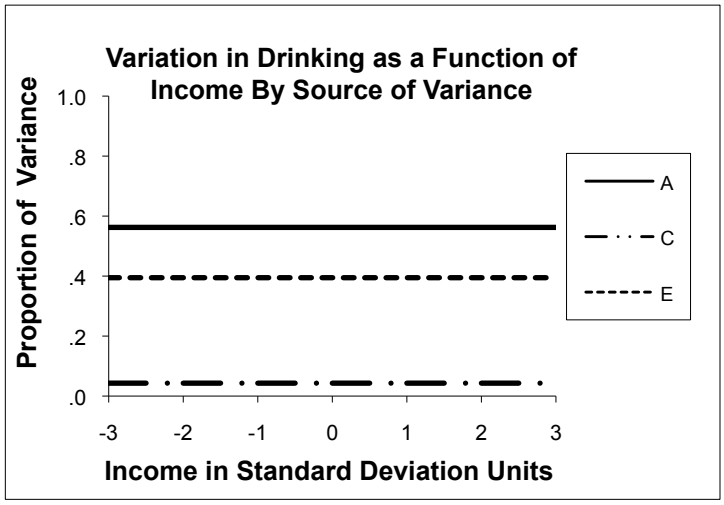
Drinking

(a)


Variation in Drinking as a Function of Income By Source of Variance
Proportion of Variance
1.0
.8
.6
.4
.2
.0
-3
-2
-1
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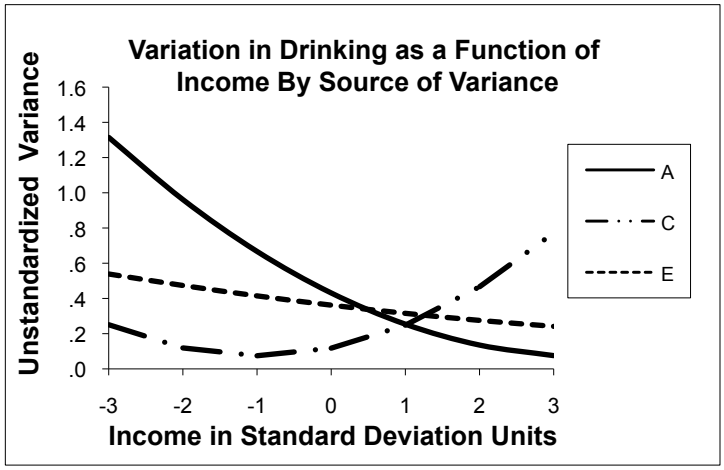
1
2
3
Income in Standard Deviation Units
A
C
E

(b)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Unstandardized Variance
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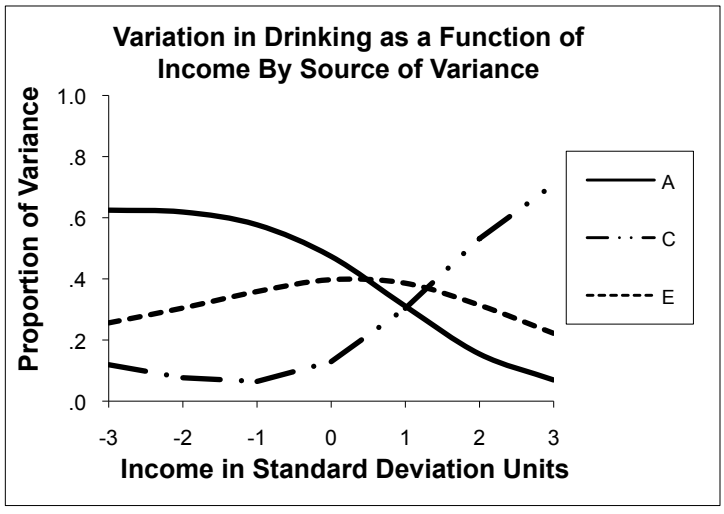
Income in Standard Deviation Units
A
C
E

(c)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Income in Standard Deviation Units
A
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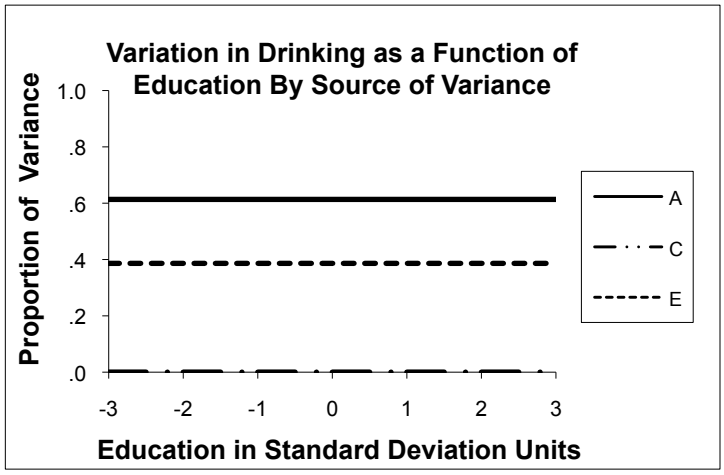
(a)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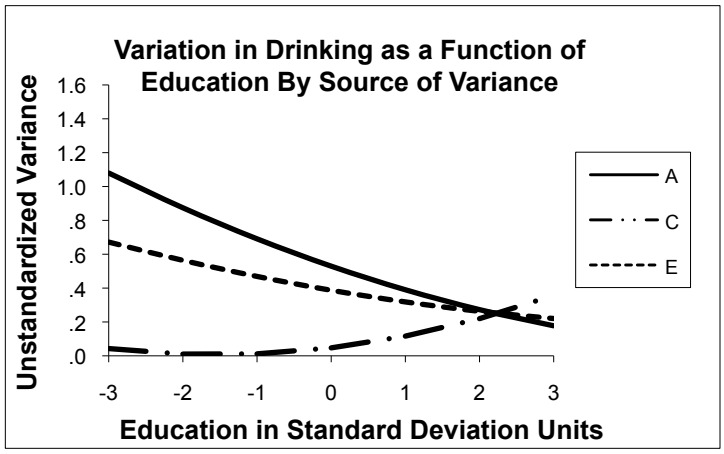
Education in Standard Deviation Units
A
C
E

(b)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Unstandardized Variance
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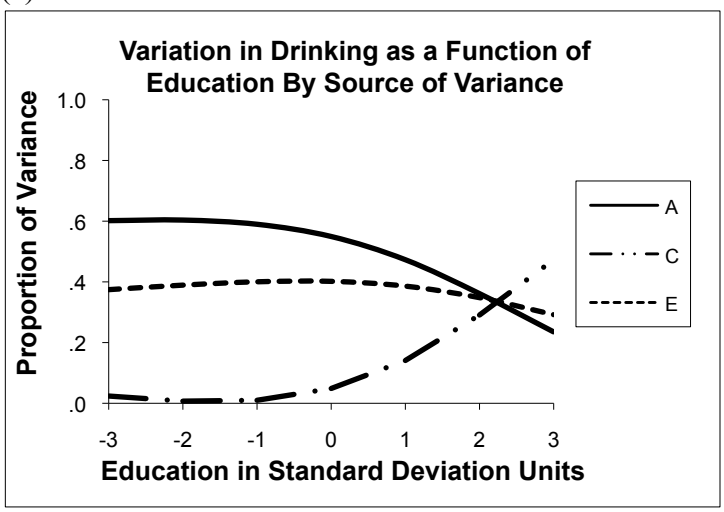
Education in Standard Deviation Units
A
C
E

(c)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Proportion of Variance
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Education in Standard Deviation Units
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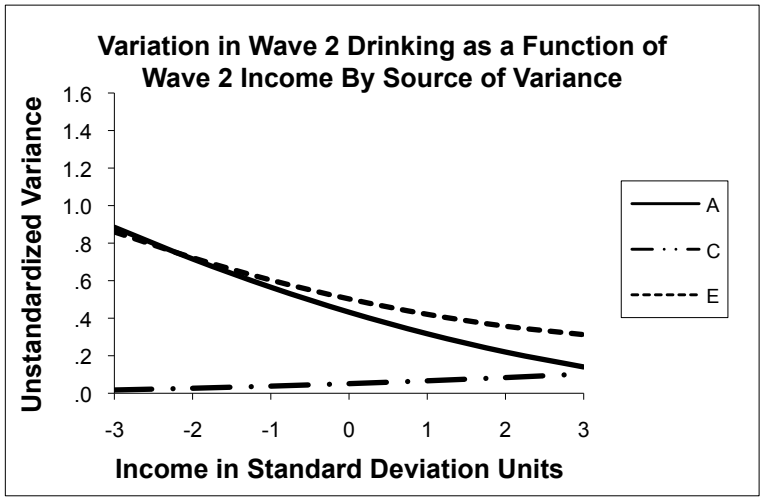
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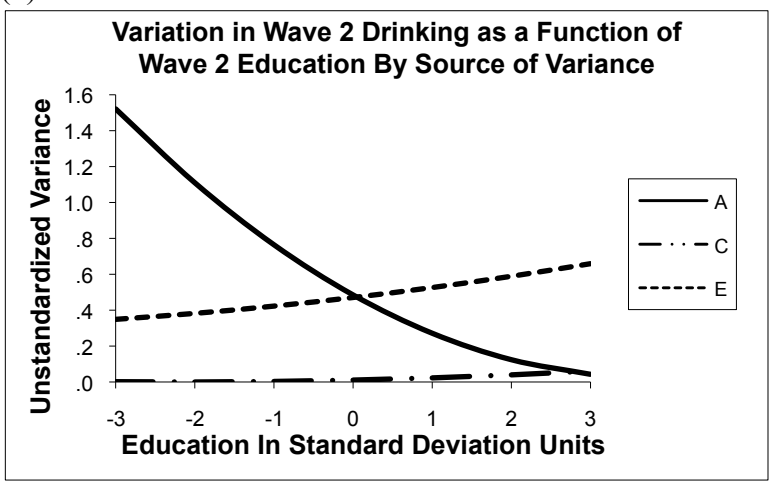
1.4
1.2
1.0
.8
.6
.4
.2
.0
-3
-2
-1
0
1
2
3
Income in Standard Deviation Units
A
C
E

(b)


Variation in Wave 2 Drinking as a Function of Wave 2 Education By Source of Variance
Unstandardized Variance
1.6
1.4
1.2
1.0
.8
.6
.4
.2
.0
-3
-2
-1
0
1
2
3
Education In Standard Deviation Units
A
C
E

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