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Interaction between *ADH1B*3* and Alcohol-Facilitating Social Environments in Alcohol Behaviors among College Students of African Descent

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Abstract

Background and Objectives—Although alcohol-facilitating social environmental factors, such as alcohol offers and high perceived peer drinking norms, have been extensively studied as determinants of college drinking, their role among college student of African descent drinking remains understudied. Furthermore, gene-environment interaction research suggests that the effects of alcohol-facilitating environments may differ as a function of genetic factors. Specifically, the alcohol dehydrogenase gene's *ADH1B*3* allele, found almost exclusively in persons of African descent, may modulate the association of risky social environments with alcohol behaviors. The current study examined whether the *ADH1B*3* allele attenuated the relationship between alcohol-facilitating environments (i.e., alcohol offers and perceived peer drinking norms) and alcohol behaviors.

Method—Participants were 241 undergraduate students who self-identified as being of African descent (mean age = 20 years [*SD* = 4.11]; 66% female).

Results—Significant interaction effects of *ADH1B*3* with alcohol offers were found on alcohol use frequency (incidence rate ratio [IRR] = 1.14) and on drinking consequences (IRR = 1.21). *ADH1B*3* also interacted with perceived peer norms on drinking consequences (IRR = 1.41). Carriers of the *ADH1B*3* allele drank less frequently and experienced fewer negative consequences than non-carriers when exposed to lower levels of alcohol offers and perceived peer drinking. In contrast, in high alcohol-facilitating environments, no protective genetic effect was observed.

Discussion and Conclusion—This study demonstrates that *ADH1B*3* may protect college students of African descent against alcohol outcomes, although only in low alcohol-facilitating environments

Scientific Significance—Findings add to the growing body of knowledge regarding genetic and social determinants of alcohol behaviors among college students of African descent.

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DECLARATION OF INTERESTS

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of this paper.

INTRODUCTION

College drinking remains a serious public health concern, with college students more likely to drink frequently, drink heavily, and meet criteria for an alcohol use disorder than their non-college peers.^{1,2} Notable racial differences exist in college drinking such that students of African descent are more likely to abstain from alcohol use and, when they drink, engage in fewer heavy-drinking episodes and consume a lower quantity of alcohol than their White peers.^{1,3} Interestingly, despite their lower levels of use, college students of African descent exhibit similar levels of negative drinking consequences as compared to White college students.⁴ Moreover, as college students of African descent advance in age, they become more susceptible to experiencing greater negative drinking consequences. Specifically, adults of African descent exhibit greater negative drinking consequence than Whites, even after accounting for alcohol consumption.⁵ Despite this troubling racial disparity, the unique determinants of college student drinking and negative consequences, among students of African descent, have been under-researched.⁶

Genetic factors impact alcohol behaviors, with up to 55% of alcohol use behavior among adolescents and young adults explained by aggregate genetic factors.⁷ Variations in alcohol metabolism genes (involving two primary alcohol-metabolizing enzymes, alcohol dehydrogenase [ADH] and aldehyde dehydrogenase) are the most consistent and well-replicated genetic variants found to impact drinking behavior.⁸ The *ADH1B* gene has a functional polymorphism found almost exclusively in up to 25% of individuals of African descent – *ADH1B*3*.⁹ The *ADH1B*3* allele increases metabolism of alcohol to acetaldehyde, and acetaldehyde accumulation is associated with unpleasant effects (e.g., nausea,¹⁰ rapid heartbeat, sedation¹¹). Possession of the *ADH1B*3* allele has also been associated with reduced alcohol consumption¹² and lower rates of alcoholism⁹ among adults. What remains understudied, however, is the interactive effect of *ADH1B*3* with risky environmental factors.

In addition to genetic factors, alcohol-facilitating social environmental factors have been identified as important determinants of drinking behaviors, particularly among college students.¹³ Alcohol-facilitating environments include both passive and active social influences. Active social influences include explicit invitations to engage in alcohol use (i.e., alcohol encouragement and offers), while passive social influences are subtler and relate to one's observation and interpretation of other's drinking behavior (i.e., perceived peer norms¹⁴). Regarding active social influences, college students frequently experience overt drinking offers.¹³ Direct alcohol offers have demonstrated positive associations with alcohol use and negative drinking consequences among samples of predominantly White college students.^{15,16} Although research has demonstrated positive associations of peer encouragement of alcohol use with alcohol use frequency among emerging adults of African descent,¹⁷ research has yet to examine the role of direct alcohol offers among a college sample of persons of African descent. Regarding passive social influences, higher levels of peer drinking norms have been associated with greater quantity of alcohol use among college samples of with students predominantly of African descent.^{6,18} Peer drinking norms have also been associated with negative drinking consequences among predominantly White

college students,¹⁹ although it remains unknown whether such findings generalize to college students of African descent.

The effects of passive and active alcohol-facilitating environments may differ as a function of alcohol-metabolizing genes in a gene-environment interaction (GxE). Protective effects of alcohol metabolism gene variants have been strongest in low-risk environments, remitting in high-risk peer or stressful family environments,^{20,21} but see also Meyers and colleagues²². For example, carriers of another protective *ADH1B* variant, *ADH1B*2*, were at lower risk for experiencing symptoms of alcohol use disorder than non-carriers, only when exposed to low levels of childhood adversity. Notably, this GxE interaction was significant among White, but not those of African descent, adults.²³ Similarly, among a sample of racially diverse adolescents and emerging adults, Olfson and colleagues²⁴ found that the protective effects of *ADH1B*2* against major drinking milestones were observed only among individuals reporting low levels of peer drinking. However, to date, research has not yet explored whether the protective effects of the *ADH1B*3* allele are similarly most prominent in low-risk environments.

The current study sought to expand existing GxE findings by testing whether the associations between high-risk environments with alcohol behaviors among college students of African descent differed as a function of a genetic factor, *ADH1B*3*. Specifically, the present study examined whether the influence of active (i.e., alcohol offers) and passive (i.e., perceived peer drinking norms) alcohol-facilitating social environments on alcohol behaviors differs as a function of the *ADH1B*3* allele among college students of African descent. It was hypothesized that carriers of the *ADH1B*3* allele would report lower alcohol use and negative drinking consequences than non-carriers only when exposed to less alcohol-facilitating social environments. However, when exposed to high levels of alcohol-facilitating social environments, it was hypothesized that the protective effect of *ADH1B*3* would diminish, such that carriers and non-carriers report similar levels of alcohol use and consequences. All hypotheses were made a priori.

METHODS

Participants and Procedure

Data were obtained from a cross-sectional study of 251 college students attending a predominantly White 4-year private university in the northeastern United States. Participants were recruited through an undergraduate research participation pool, email invitations through electronic mailing listservs to various student organizations, flyers across campus, and a snowball sampling technique. Students were eligible if they met the following inclusion criteria: (1) currently enrolled in college, (2) 18 years of age or older, (3) self-identified as being of African descent, and (4) reporting alcohol consumption at least once in the past 30 days.

After confirming eligibility, written informed consent was obtained from each participant upon arrival to the lab. Eligible students completed a paper-and-pencil questionnaire on diverse health behaviors and provided a 2.0mL saliva sample for genotyping. Students recruited through the university research pool were compensated with research credit, and

those recruited through other methods were compensated with \$15. Participants could receive up to an additional \$15 for referring future, eligible participants (\$5 per referral, up to 3 referrals permitted). All study measures and procedures were approved by the university's institutional review board.

Ten saliva samples could not be genotyped despite multiple attempts and were dropped from analyses, resulting in a final sample of 241 students. Students whose saliva samples could not be genotyped ($n = 10$) did not differ from students whose samples were genotyped on any study variables at $p < .05$.

Measures

Demographics—Age and sex (0 = *female* and 1 = *male*) were assessed and included as covariates in all analyses due to their potential confounding effects on college drinking.¹

Alcohol Offer—A single item measured frequency of alcohol offers, “In the past 30 days, how many times have you been offered an alcoholic drink,” on a 0 (*never in the past month*) to 6 (*five or more times a week*) Likert scale.²⁵ This single item has been used in studies on racially diverse samples of college students.^{15,16}

Perceived Peer Drinking Norms—Six items measured perceived peer drinking norms, which included two items assessing descriptive norms (e.g., “How many of your close friends would you estimate get drunk at least once a week?”) based on a 0 (*none*) to 4 (*all*) Likert scale and four items assessing injunctive norms (e.g., “How do you think your close friends feel [or would feel] about you drinking four or five drinks regularly”) using a 0 (*strongly approve*) to 4 (*strongly disapprove*) Likert scale.²⁶ (Bachman, Johnston, O'Malley, 2009). These items have been used in studies among racially diverse sample of adolescents.²⁷ An average score of six items was used for main analyses (Cronbach's $\alpha = .74$).

ADH1B*3—The single nucleotide polymorphism (SNP) that defines *ADH1B*3* (i.e., rs2066702) was genotyped through PCR amplification of the region around exon 9 (Arg370Cys) and allele-specific fluorescence using the ThermoFisher TaqMan (SNP) genotyping assay in an Applied Biosystems 7500 real-time PCR instrument. Saliva samples were genotyped twice and demonstrated 100% concordance. The prevalence of the *ADH1B*3* allele did not deviate significantly from Hardy-Weinberg equilibrium, $\chi^2 (1, n = 241) = 0.90, p = .34$. Participants were dichotomized as carriers of the protective, low-risk allele (either heterozygous or homozygous for the *ADH1B*3* allele; $n = 83$) or non-carriers ($n = 158$). Similar to previous studies,^{11,12} a continuous genotype classification (i.e., none, one, or two risk alleles) was not used due to low frequency of the homozygous *ADH1B*3/3* genotype (2–3%).

Alcohol Use—To assess alcohol consumption, a paper-and-pencil version of the Timeline Follow Back calendars was administered.²⁸ Participants were presented with a calendar and asked to record the number of standard drinks they consumed on each of the past 30 days. The definition of a drink was given to participants as a 12-oz. can or bottle of beer, 5-oz. glass of wine, 8-oz. glass of malt liquor, or 1.5 oz. of hard liquor straight or in a mixed

drink; pictures of standard drinks were also provided. Memory aids were provided by noting special events on the calendars (e.g., holidays; major school, community, and sports events, etc.) to enhance recollection of drinking days. The paper-and-pencil administration of the Timeline Follow Back calendars has demonstrated sound validity and reliability in persons of African descent and racially diverse college students.²⁹ Answers were recoded into an alcohol use frequency variable (i.e., how many of the past 30 days the participant consumed at least one standard drink). Moreover, with consideration of the low levels of other quantity-based measures (e.g., alcohol use quantity, heavy drinking) among both national³⁰ and our sample of college students of African descent, frequency of alcohol use may provide a better estimate of the alcohol use behaviors. We note, however, that we conducted ancillary analysis of heavy drinking (i.e., the number of days that participants consumed four or more drinks for women, and five or more drinks for men), and did not find any significant gene-environment interaction effects with neither offer nor peer norms at $p < .05$. These results are available upon request.

Negative Drinking Consequences—The 23-item Rutgers Alcohol Problem Index³¹ was used to assess the frequency of experiencing negative drinking consequences in the past three months, such as “Neglected your responsibility.” Participants responded to the items on a 5-point scale (0 = *none* to 4 = *more than five times*). A sum score was used for analyses. This scale has been used to assess negative drinking consequences among college students of African descent.³²

Data Analytic Strategies

All study analyses were conducted in SPSS, Version 23. Descriptive data analyses were conducted to obtain means/percentages and bivariate correlations among all study variables. As main analyses, generalized negative binomial models were used to account for our over-dispersed (i.e., a variance greater than its mean) count alcohol outcomes. A separate model was analyzed for each alcohol variable. For negative consequences, we estimated two sets of models both with and without controlling for alcohol use frequency to determine whether findings for negative drinking consequences were due to effects on frequency of alcohol use. In all analyses, participant age and sex were included as covariates to control for their potential confounding effects on alcohol behavior.

A total of six models were estimated to test the interaction of each environmental variable (i.e., alcohol offer and peer drinking norms) with *ADH1B*3* on the three alcohol behaviors. These GxE models included all two-way interaction terms of each covariate (i.e., age, sex, and alcohol use frequency) with predictors to account for potential confounding interaction effects.³³

Interaction terms were calculated by multiplying the *ADH1B*3* variable with each grand-mean centered environmental variable. As a measure of effect sizes for individual predictors, incidence rate ratios (IRRs) were estimated, along with their 95% confidence intervals. If interaction effects were significant as hypothesized, post-hoc analyses were conducted to estimate simple effects of environmental factors on alcohol behaviors separately as a function of *ADH1B*3* genotype (carriers versus non-carriers).

RESULTS

Descriptive Statistics

Means (with standard deviations in parentheses) or percentages and bivariate Pearson correlation coefficients of all study variables are presented in Table 1. On average, students drank on approximately six of the past 30 days ($SD = 4.28$), similar to levels observed among national samples of racially diverse college students.³⁴ Bivariate correlation coefficients revealed significant, positive associations of alcohol offers and peer norms with both alcohol frequency and negative consequences. *ADHIB*3* presence was not significantly associated with any environmental factors or alcohol behaviors. As shown in Table 2, compared to female students, male students showed significantly higher levels of alcohol offer, alcohol use frequency, and negative drinking consequences (but not in age, *ADHIB*3* presence or peer norms) at $p < .05$.

Generalized Negative Binomial Models

Alcohol Offers—As shown in the first row of data in Table 3, a significant interaction between *ADHIB*3* and alcohol offers was found on alcohol frequency ($IRR = 1.14$, $p = .04$) and on negative consequences before controlling for alcohol frequency ($IRR = 1.21$, $p = .047$). Thus, the association of alcohol offers with alcohol frequency and negative consequences differed as a function of *ADHIB*3*. However, upon controlling for the effects of alcohol use frequency, the previously significant interaction effect on negative drinking consequences became non-significant ($IRR = 1.03$, $p = .82$; shown in the third column of data in Table 3).

To probe the significant GxE interaction effects, simple main effects of alcohol offer on alcohol frequency and negative consequences were estimated separately among *ADHIB*3* carriers and non-carriers. Results showed that, although alcohol offers were positively associated with alcohol frequency in both genotype groups, this association was stronger among carriers ($B = .41$, $p < .001$) than non-carriers ($B = .28$, $p < .001$). A similar pattern of results was found for negative drinking consequences; although alcohol offers were positively associated with negative consequences in both groups, the association was stronger among carriers ($B = .34$, $p < .001$) than non-carriers ($B = .15$, $p = .01$). This pattern is illustrated in Figure 1, left panel. Carriers reported lower frequency of alcohol use ($M = 4.16$; $SD = 1.50$) than non-carriers ($M = 5.46$; $SD = 4.18$) in the presence of low levels of alcohol offers (i.e., lower 50%). However, in the presence of high levels of alcohol offers (i.e., upper 50%), there appeared to be no differences among carriers ($M = 6.86$; $SD = 3.76$) and non-carriers ($M = 7.80$; $SD = 4.61$). A similar pattern of results was found for negative drinking consequences, as shown in Figure 1, middle panel.

Perceived Peer Drinking Norms—As shown in the second row of data in Table 3, a non-significant interaction effect between *ADHIB*3* and peer norms was found on alcohol frequency ($IRR = 1.21$, $p = .11$). Similar to analyses with alcohol offer, although a significant interaction was found on negative consequences before controlling for alcohol frequency ($IRR = 1.41$, $p = .03$), this interaction became non-significant ($IRR = 1.19$, $p = .31$) when controlling for alcohol frequency.

Simple main effects of peer drinking norms on negative consequences were also estimated separately among *ADH1B*3* carriers and non-carriers. Results demonstrated that among non-carriers, perceived peer drinking norms did not show a significant effect on negative consequences ($B = -.05$, $p > .05$). In contrast, among carriers, peer norms were significantly and positively associated with negative consequences ($B = .30$, $p < .05$). As displayed in Figure 1, right panel, carriers of the *ADH1B*3* allele reported greater negative drinking consequences at high levels of peer norms ($M = 8.98$; $SD = 2.07$) than at low levels of peer norms ($M = 5.77$; $SD = 1.30$), whereas non-carriers appeared to report similar levels of negative consequences at high ($M = 7.86$; $SD = 1.27$) and low ($M = 8.24$; $SD = 1.98$) levels of peer norms.

DISCUSSION

This study extended existing GxE interaction literature by examining whether the *ADH1B*3* allele attenuated the association of alcohol-facilitating social environmental with alcohol behaviors. Results demonstrated that *ADH1B*3* is associated with lower drinking frequency only among college students of African descent exposed to lower levels of alcohol-facilitating (i.e., alcohol offers) environments. Similar protective effects of *ADH1B*3* on negative drinking consequences were found at low levels of environmental risk (i.e., perceived peer norms and alcohol offers) before controlling for alcohol use frequency, although these protective genetic effects disappeared when controlling for alcohol use frequency. Thus, the previously reported protective effects of alcohol metabolism gene variants on negative consequences may be due to genetic protection against frequent alcohol use. Ancillary analyses did not demonstrate any significant findings for our heavy drinking variable. This may be due to infrequent heavy drinking among college students of African descent.^{3,30} Overall, these results suggest that high-risk social environments may overpower the protective impact of this alcohol-metabolizing gene variant on alcohol behaviors among college students of African descent.

Overall, the findings that risky environments attenuate the protective effects of an alcohol metabolism genotype corroborate previous research. Prior research suggests the protective effects of other alcohol metabolizing gene variants (e.g., *ADH1B*2*, *ALDH2*2*) are stronger in low-risk peer and family environments than high-risk environments, including those characterized by childhood adversity and adolescent peer drinking.^{20,23,24} Only one study has found a protective effect of *ADH1B* in high-risk environments (i.e., childhood adversity²²). These discrepancies may have been due to rates of alcohol consumption in each sample; while Meyers and colleagues²² found protective effects of alcohol metabolism gene variants in high-risk environments among a sample of older adults (i.e., mean age of 41) with relatively low rates of alcohol consumption, other research demonstrated protective alcohol metabolism gene effects only in low-risk environments among samples of adolescents and young adults^{20,24} and individuals at high risk for alcohol use.²³ Future research may seek to examine the extent to which differences in age and drinking level impact the attenuating effects of alcohol metabolizing genes in the effect of risky environments on alcohol behaviors.

The current study's nonsignificant genetic protective effects in the presence of high-risk alcohol-facilitating environments may be further explained by the potent alcohol-promoting college drinking culture. Alcohol use is such a prominent aspect of the college experience that neglecting to partake in alcohol use may inhibit students from fully engaging in collegiate social life.³⁵ This may be particularly important for students of African descent, given that adjusting to the main campus culture and forming positive relationships with peers were found to be central to their success.³⁶ When alcohol offers are copious in collegiate settings, students of African descent may be strongly inclined to accept and model majority alcohol use behaviors. In addition, the unpleasant effects experienced by *ADH1B*3* carriers (e.g., nausea,¹⁰ which typically accompanies hangovers) may be deemed as a neutral or even positive outcome and part of the overall social drinking experience for college student drinkers.³⁷ Thus, the strong influence and social rewards of the college drinking culture may overpower possible genetically-based protection afforded by the *ADH1B*3* allele, particularly among young drinkers exposed to alcohol-facilitating social environments.

There is a paucity of clinical interventions designed specifically for drinking among college students of African descent. Findings from the current study may aid in the development of racially tailored clinical prevention/intervention efforts to curtail alcohol use and misuse. As demonstrated in the present study, protective genetic factors, though important, may not be sufficient in reducing alcohol engagement and alcohol-related consequences in high-risk environments. To compensate for diminished genetic protection, college students of African descent may benefit from understanding and learning specific tools to limit their alcohol consumption, particularly when in high alcohol-facilitating environments. For example, harm reduction approaches with protective behavioral strategies (e.g., alternating alcoholic and non-alcoholic drinks, setting a limit on number of drinks, arranging for a designated driver) have been associated with decreased alcohol consumption and negative drinking consequences among college students of African descent.³⁸ Moreover, psychoeducation about possible alcohol-related risks due to genotype (i.e., genetic feedback interventions) may be efficacious in reducing students of African descent's alcohol use.³⁹

Notwithstanding our novel and significant contributions to the literature, our findings should be interpreted within the context of several limitations. First, the present study was comprised of a small college sample; inadequate statistical power due to small sample sizes has been mentioned as one of the main concerns for GxE studies.³³ Second, the self-report nature of the data presents concerns, given the subjective measures of environmental influences and alcohol use behaviors. Although students were provided memory aids to help recall their alcohol use, they may have had difficulty remembering their drinking experiences and either have under- or over-reported their alcohol use and consequences. Nonetheless, self-report measures of personal alcohol use have been shown to be reliable and valid⁴⁰, albeit there is no such support among drinkers of African descent. Lastly, due to the reliance on cross-sectional data and the varying time frames of the used measures, no conclusions about causality among study variables can be drawn.

Future work should attempt to replicate these findings using other social environmental factors, such as involvement in drinking games and social affiliations (i.e., engagement in

Greek affiliated organizations). Additionally, the present study did not examine potential factors that may underlie the identified GxE interaction, such as alcohol expectancies or drinking motives, so the mechanisms of these interaction effects remain unknown. Finally, the present study focused on college students of African descent; other populations, such as non-college young adults or adolescents of African descent, may differ in their environmental exposures and drinking behaviors. Thus, replication of study findings across different samples of large sizes is warranted.

In conclusion, this is the first study to examine the protective effect of the *ADH1B*3* allele and the social drinking context among college students of African descent. The *ADH1B*3* allele may protect against alcohol behaviors in low alcohol-facilitating social environments, although its protective effects may diminish as exposure to alcohol-facilitating social environments increases. Our findings add to the growing body of knowledge regarding genetic and social determinants of individual differences in alcohol behaviors among college students of African descent.

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Figure 1.

Mean levels of alcohol frequency and negative consequences (before controlling for alcohol frequency) as a function of *ADH1B*3* and alcohol-facilitating environments. Error bars represent the standard error of the mean.

Table 1

Descriptive Statistics and Bivariate Correlations Among Study Variables

	<i>M</i> (%)	<i>SD</i>	1	2	3	4	5	6	7
1. Age	20.04	4.10	—						
2. Male sex	34%		.03	—					
3. <i>ADHIB</i> * 3 presence	34%		.00	-.10	—				
4. Alcohol offer	3.73	1.29	-.21**	.13*	-.02	—			
5. Peer drinking norms	2.53	0.72	-.16*	.05	-.02	.35***	—		
6. Alcohol frequency	6.19	4.28	.17**	.16*	-.10	.51***	.28***	—	
7. Drinking consequences	7.84	6.93	.11	.16*	-.04	.29***	.06	.35***	—

N = 239–241 due to missing data.*
p < .05;**
p < .01;***
p < .001.

Table 2

Percentages and Means (Standard Deviations) of Study Variables as a Function of Sex

Variable	Females (<i>n</i> = 159)	Males (<i>n</i> = 82)	Comparison Test Statistics
1. Age	19.97(4.58)	20.19(2.99)	$t(237) = -0.39$
2. <i>ADH1B</i> *3 presence	38%	28%	$\chi^2(1) = 2.25$
3. Alcohol offer	3.62(1.28)	3.96(1.28)	$t(239) = -2.00$
4. Peer drinking norms	2.50(0.72)	2.58(0.72)	$t(239) = -0.84$
5. Alcohol frequency	5.70(3.80)	7.15(4.98)	$t(239) = -2.52$
6. Drinking consequences	7.06(6.69)	9.35(7.16)	$t(239) = -2.46$

N = 239–241. Significant group differences at $p < .05$ are highlighted in bold font.

Table 3

Results from Negative Binomial Regressions Predicting Alcohol Behaviors from *ADH1B* Genotype and Alcohol-facilitating Environments

	Alcohol use frequency	Negative consequences, before controlling for alcohol frequency	Negative consequences, after controlling for alcohol frequency
	IRR [95% CI] <i>p</i>	IRR [95% CI] <i>p</i>	IRR [95% CI] <i>p</i>
Model of Alcohol offer			
Age	1.06 [1.04,1.09] .00	1.04 [1.00,1.08] .04	1.02 [0.98,1.05] .42
Age × <i>ADH1B*3</i>	0.98 [0.94,1.02] .31	1.00 [0.93,1.07] .89	1.02 [0.95,1.10] .61
Age × Alcohol offer	1.01 [0.99,1.02] .47	1.02 [1.00,1.04] .03	1.03 [1.01,1.05] .01
Male sex	1.14 [0.95,1.37] .15	1.27 [0.96,1.68] .09	1.16 [0.89,1.52] .28
Male sex × <i>ADH1B*3</i>	0.89 [0.64,1.25] .51	1.02 [0.61,1.70] .94	1.12 [0.68,1.83] .67
Male sex × Alcohol offer	0.95 [0.84,1.07] .39	1.02 [0.85,1.22] .87	1.13 [0.94,1.36] .21
Alcohol use frequency	—	—	1.06 [1.02,1.10] .01
Alcohol use × <i>ADH1B*3</i>	—	—	1.05 [0.98,1.12] .19
Alcohol use × Alcohol offer	—	—	0.97 [0.95,0.99] .00
<i>ADH1B*3</i>	1.39 [0.57,3.36] .47	1.01 [0.25,4.12] .99	0.48 [0.12,2.01] .32
Alcohol offer	1.23 [0.94,1.60] .14	0.73 [0.47,1.12] .15	0.68 [0.43,1.06] .09
<i>ADH1B*3</i> × Alcohol offer	1.14 [1.01,1.30] .04	1.21 [1.00,1.46] .047	1.03 [0.82,1.29] .82
Model of Peer drinking norms			
Age	1.07 [1.04,1.10] .00	1.02 [0.98,1.07] .34	0.98 [0.94,1.03] .48
Age × <i>ADH1B*3</i>	0.96 [0.92,1.00] .05	0.95 [0.90,1.01] .10	0.96 [0.91,1.03] .24
Age × Peer norm	1.03 [1.01,1.06] .01	1.00 [0.96,1.03] .78	0.98 [0.95,1.02] .35
Male sex	1.22 [1.00,1.50] .05	1.37 [1.04,1.82] .03	1.30 [0.99,1.72] .06
Male sex × <i>ADH1B*3</i>	0.71 [0.49,1.03] .07	1.03 [0.62,1.73] .90	1.16 [0.70,1.91] .58
Male sex × Peer norm	1.07 [0.84,1.36] .57	0.94 [0.68,1.30] .70	0.92 [0.67,1.27] .62
Alcohol use frequency	—	—	1.06 [1.03,1.10] .00
Alcohol use × <i>ADH1B*3</i>	—	—	1.07 [1.01,1.14] .02
Alcohol use × Peer norm	—	—	0.98 [0.95,1.02] .30
<i>ADH1B*3</i>	2.12 [0.90,4.95] .08	2.45 [0.75,8.01] .14	1.27 [0.37,4.32] .71
Peer norm	0.65 [0.40,1.07] .09	1.07 [0.53,2.15] .86	1.43 [0.71,2.86] .31
<i>ADH1B*3</i> × Peer norm	1.21 [0.96,1.53] .11	1.41 [1.03,1.94] .03	1.19 [0.85,1.67] .31

N = 239 – 241 due to missing data. Significant statistics at *p* < .05 are highlighted in bold.