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Racial/ethnic discrimination, *ADH1B*3*, and coping-motivated drinking among Black college students

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Abstract

Background and Objectives: Discrimination due to race and/or ethnicity can be a pervasive stressor for Black college students in the United States beyond general negative life events and has demonstrated associations with adverse health and alcohol outcomes. Genetics may confer individual differences in risk of drinking to cope with discrimination-related stress. This study tested whether associations of racial/ethnic discrimination with coping drinking motives and alcohol use differ as a function of a well-documented variant in the alcohol dehydrogenase 1B gene (*ADH1B*3*).

Methods: Cross-sectional data were obtained from 241 Black students (M_{age}=20.04 [range=18–53]; 66% female) attending a predominantly White university in the northeastern United States. Participants provided a saliva sample for genotyping and self-reported on their racial/ethnic discrimination experiences, coping drinking motives, and past-month total alcohol quantity.

Results: Path models demonstrated that associations of discrimination with alcohol quantity directly or indirectly through coping drinking motives did not differ as a function of *ADH1B*3*, after controlling for gender, age, negative life events, and potential confounding interactions of covariates with model predictors. Regardless of *ADH1B*3*, greater experience of negative life events was associated with higher coping drinking motives, which in turn were associated with greater alcohol quantity.

Conclusions and Scientific Significance: Findings represent a novel investigation into gene-environment interplay in associations of alcohol use with racial/ethnic discrimination.

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Declaration of Interest

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

Findings demonstrate coping-motivated drinking associated with negative life events within Black college drinkers regardless of *ADH1B*3*. Future research should leverage longitudinal designs to characterize associations of genetics, stressful experiences, and coping-motivated drinking over time.

Keywords

alcohol metabolism gene; Black students; college drinking; coping motives; racial discrimination

Black college students are at considerable risk for problem drinking. Although Black students drink less than students from other racial groups overall[1], Black drinkers experience alcohol problems (e.g., injuries or accidents, academic/occupational impairment, social problems, symptoms of alcohol use disorder) at similar or higher levels than their other racial peers, even at comparable drinking levels.[2] Despite these disparities, efforts to understand the unique alcohol risk factors within Black students have been considerably limited. Research often comprises samples in which Black students are underrepresented or has focused on comparative relations (i.e., overall racial differences in alcohol use) such that there is a substantial dearth of knowledge on individual differences in alcohol risks within Black students. Research focused on Black drinkers is needed to better capture the likely complex variability in alcohol risk and resilience factors within the Black population.

Racial/ethnic discrimination represents a salient, race-specific stressor for Black students that may increase adverse alcohol behavior. Discrimination can include overt or covert social exclusion, harassment, stigmatization, and unfair treatment due to race and/or ethnicity.[3] Discriminatory experiences represent racially relevant stressors experienced above and beyond general negative life events, thereby capable of further burdening individuals' adaptive coping strategies. Discriminatory experiences have been suggested to elicit powerful stress responses that can increase maladaptive coping behaviors.[4] Discrimination-related stress may increase individuals' motivations to cope with resultant negative affect through alcohol use (i.e., coping drinking motives) and, in turn, their use of alcohol for its potential stress-dampening effects.[5] Discrimination has been associated with greater alcohol behavior among Black Americans[6] as well as within some samples of Black college students[7], yet has more mixed associations within additional college samples.[8,9] Findings underscore possible variability in associations of discrimination-related stress, over and above general negative life events, with Black college student drinking behavior.

Genetics may contribute to individual differences in coping-motivated drinking after discrimination. Research suggests significant variability in associations of discrimination with alcohol behaviors across the Black population.[6] Genetic factors may be meaningful contributors to such variability given their role in individual differences across various alcohol indices.[10] Genetic factors can drive drinking through complex relations with the environment. Gene-environment interactions (G×E) represent one form of such interplay[11] and can be interpreted as environmental modulation of genetic influences or as genetic modulation of environmental influences. Through the latter framework, genetic vulnerabilities (or resiliencies) may render some Black students more (or less) likely to cope with discrimination-related stress through alcohol use.

Genetic variation in alcohol metabolism genes specifically may contribute to individual differences in associations of discrimination with alcohol behavior. Several variants, or alleles, in genes encoding alcohol dehydrogenase can increase conversation of alcohol into acetaldehyde, leading to acetaldehyde accumulation and associated negative physiological responses such as racing heart, increased pulse, and nausea.[12] Such physiological effects may reduce the reinforcing value of alcohol and decrease motivations to cope with stressors through drinking.[13] The *ADH1B*3* allele (rs2066702) on the alcohol dehydrogenase 1B (*ADH1B*) gene has been associated with reduced alcohol consumption among Black Americans.[14] Thus, Black students carrying an *ADH1B*3* allele may be less likely than noncarriers to engage in coping-motivated drinking following discrimination.

Research has yet to test differences in associations of discrimination with drinking as a function of alcohol metabolism genes. Prior investigations support additional candidate genes modifying alcohol behavior in response to various stressors within Black and mixed race youth and college students[15–17], although no differences afforded by *ADH1B*3* in associations of childhood adversity with alcohol consumption within Black adults.[14] Research is needed to test genetic differences in response to racial/ethnic discrimination—a pervasive and race-related stressor in the Black community. Such efforts should examine any differences in the unique impacts of discrimination on coping-motivated drinking, over and above general negative life experiences. Research also should explore whether *ADH1B*3* modulates stress-related drinking earlier in life before adulthood, given research suggesting gene-environment interaction effects may be somewhat more pronounced earlier in development.[18] Such efforts could better characterize why some Black students may be more likely to cope with discrimination-related stress through drinking during college, a critical time for emergence of problem drinking that can persist much later in life.

The present study tested whether alcohol consumption associated with racial/ethnic discrimination differed as a function of *ADH1B*3* among Black college students. To our knowledge, this represents the first gene-environment interaction test involving discrimination and alcohol consumption. Within a sample of Black college drinkers, the present study examined associations among racial/ethnic discrimination, coping drinking motives, and alcohol consumption as a function of *ADH1B*3*, after controlling for negative life events and demographics. We hypothesized that more frequent experiences of discrimination would be associated with increased coping drinking motives and, in turn, greater alcohol consumption. Further, we hypothesized that *ADH1B*3* would moderate these associations, such that associations of discrimination with coping drinking motives and alcohol quantity would be demonstrated among *ADH1B*3* noncarriers but not among carriers.

Methods

Participants and Procedures

Data were drawn from a cross-sectional study of Black college students attending a predominantly White four-year university in the northeastern United States.[19–21] Eligible participants were undergraduate students ≥18 years of age who self-identified as Black and reported consuming alcohol at least once in the past 30 days. Participants were

recruited through an undergraduate research participation pool, solicitations through student organizations, flyers, and respondent-driven sampling. Participants provided written informed consent at the research laboratory prior to completing questionnaires on diverse health behaviors. Participants also provided a 2.0mL saliva sample for genotyping. The final sample comprised 241 Black students with complete genetic data (mean age=20.04 [*SD* = 4.11; range=18–53]; 66% female). Participants were compensated with research credit or $15, as well as up to an additional $15 for referring future participants ($5 per eligible referral, maximum of three). Study procedures were approved by the university's institutional review board (IRB Protocol #12-288).

Measures

Racial/Ethnic Discrimination—The Perceived Ethnic Discrimination Questionnaire[22] assessed frequency of ethnicity-based discrimination (e.g., offensive comments, unfair treatment) experienced in the past three months using a 1 (*never*) to 7 (*very often*) scale. The original measure construction was premised on using "ethnicity" to capture both culture of origin (i.e., ethnicity) and racial group, such that it is referred to herein as racial/ethnic discrimination.[22] Sum scores were computed (α=.88), with higher scores indicating more racial/ethnic discrimination experiences.[19] The Perceived Ethnic Discrimination Questionnaire has demonstrated good psychometrics in mixed race college students.[22]

Coping Drinking Motives—The Drinking Motives Questionnaire-Revised[23] assessed frequency of drinking to cope with negative affect. Participants responded to each item (e.g., because it helps you when you feel depressed or nervous) using a 1 (*almost never/never*) to 5 (*almost always/always*) scale. Sum scores were computed (α=.84), with higher scores indicating more frequent coping-motivated drinking. Enhancement motives were considered as a covariate to control for positive affect motivations to drink due to demonstrated intercorrelations among drinking motives.[23] However, enhancement motives were not correlated with discrimination or *ADH1B*3* and were dropped from analyses. The Drinking Motives Questionnaire-Revised has demonstrated validity in relation to alcohol behaviors among mixed race youth.[23]

ADH1B*3—Genomic DNA was extracted from saliva samples using the Qiagen DNeasy Blood and Tissue Kit (Valencia, California) following manufacturer's instructions. Prior to polymerase chain reaction (PCR) analysis, sample quality was assessed using agarose gel electrophoresis. The *ADH1B* alleles (rs2066702: *ADH1B*1* and *ADH1B*3*; G/A) were genotyped using PCR amplification of the region surrounding the *ADH1B* gene on chromosome 4 and allele-specific fluorescence using the ThermoFisher TaqMan® SNP genotyping assay (Assay ID: C_11941896_20) on an Applied Biosystems™(ABI) 7500 real-time PCR instrument by Salimetrics, LLC (Carlsbad, California). Observed genotypes included *ADH1B*1/1* (*n*=158), *ADH1B*1/3* (*n*=77), and *ADH1B*3/3* (*n*=6), and observed allele prevalence did not deviate significantly from Hardy-Weinberg equilibrium, $\chi^2(1, N = 241)=0.90$. Participants were dichotomized as carriers or noncarriers of at least one protective, low-risk allele (0=*ADH1B*1/1*; 1=*ADH1B*1/3* or *ADH1B*3/3*), similar to past research[14], given the low prevalence of the homozygous recessive genotype and concerns of potential false positive or negative findings when modeling a three-category interaction.

Alcohol Quantity—The Timeline FollowBack[24] assessed alcohol consumption over the past 30 days. Participants were provided with the definition of a standard drink (12-oz beer, 8–9oz malt liquor, 5-oz wine, 1.5-oz 80-proof spirits) and reported the number of standard drinks they consumed on each of the past 30 days. Calendars highlighted national, local, and campus events to assist participant recall. Total number of standard drinks consumed in the past 30 days was selected as an outcome, because quantity-based alcohol measures have been more strongly associated with alcohol problems than alternative indices among college drinkers.[25]

Covariates

Participant Characteristics.: Participant self-reported gender (0=*female*, 1=*male*) and age were controlled for in analyses due to their demonstrated associations with college drinking.

Negative Life Events.: The Life Events Scale for Students[26] assessed major life stressors (e.g., personal injury or illness, major change of health in close family member) experienced over the past year. Participants responded to 36 items dichotomously (0=*no*, 1=*yes*), and a sum score of all events endorsed as negative (i.e., rated as −1 or −2) was computed, with higher scores indicating greater experience of negative events. The Negative Life Events Scale for Students does not include any discrimination-related items and was included as a covariate to statistically control for potential confounding effects of non-discrimination-related stressors. The Life Events Scale for Students has demonstrated validity in relation to student distress[26] and has been used in previous samples of Black college students.[16]

Data Analytic Strategies

Descriptive analyses were conducted in SPSS, Version 28 (IBM Corp., 2018). Coping drinking motives were normally distributed (skewness=1.39; kurtosis=1.62). Alcohol quantity was positively skewed (skewness=3.23; kurtosis=15.89); nonnormality was addressed through square root transformation, with the transformed score used in path analyses (skewness = 1.31; kurtosis = 2.55). There was missing data from two students on age (<1%) and complete data for all remaining study variables.

Path analyses were conducted in Mplus, Version 7.4.[27] Path analyses can estimate complex relations among multiple predictors and outcomes in a single model simultaneously. Maximum likelihood estimation with robust standard errors was specified.[27] Path analyses specified a moderated mediation model testing whether *ADH1B*3* moderated the indirect effect of discrimination on alcohol quantity through coping drinking motives (i.e., gene-environment interaction; Figure 1).[28,29] The path from discrimination to coping motives represented the *a* path, and the path from coping motives to alcohol quantity represented the *b* path. Path analyses examined atemporal associations[30] referred to herein as indirect effects. Models controlled for gender, age, and negative life events as well as all two-way interaction terms involving each covariate with both discrimination and *ADH1B*3* (i.e., discrimination × covariate, *ADH1B*3* × covariate) to control for potential confounding interaction effects.[31] Continuous predictors and covariates were mean centered prior to calculating product terms. Significance testing for indirect, direct, and total effects was conducted using 10,000 bias-corrected bootstrapped samples, as the bias-corrected bootstrap

method has outperformed alternative indirect effects tests.[32] Confidence intervals not encompassing zero were interpreted to suggest significant effects. Finally, path analyses were fully saturated and, thus, model fit statistics were not reported.

Power Analysis

Replicability concerns across gene-environment interaction research underscore the importance of *a priori* power analyses. Since there was no prior gene-environment interaction study involving discrimination among Black college students, effect size estimates were derived from prior research on additional candidate gene variants interacting with general stressful events in Black and mixed race youth and college students[15–17] as well as *ADH1B*3* interacting with childhood maltreatment in Black adults.[14] Statistics reported in text/tables or means extracted from graphs were converted into Cohen's *d* when possible using published formulas[33], and a weighted average effect size was computed (Cohen's d=0.43). Power estimates based on a two-tailed alpha level of .05 suggested that a sample of 200 participants would provide over 99% power to detect similar sized effects within moderated mediation models using bias-corrected bootstrapping.[34]

Results

Descriptive Statistics

Students reported consuming over 27 standard drinks on average in the past 30 days (M=27.54; Table 1). Thirty-four percent of students carried at least one *ADH1B*3* allele, and neither students' coping drinking motives nor alcohol quantity differed significantly as a function of *ADH1B*3* in bivariate analyses (Table 1). Students endorsed a range of racial/ethnic discrimination experiences over the past three months, including offensive comments aimed at the students' racial/ethnic group (i.e., stereotypic statements, offensive jokes; 88%) or directly at the student (71%), implications the student was violent/dangerous (68%) or unintelligent (68%), and low expectations from others (68%). Greater racial/ethnic discrimination experiences were correlated with greater coping motives among *ADH1B*3* noncarriers but not among carriers (Table 2). Greater coping motives were correlated with greater alcohol quantity among *ADH1B*3* carriers but not among noncarriers (Table 2).

Path Analysis

Path models examined associations of racial/ethnic discrimination, *ADH1B*3*, and coping drinking motives with alcohol quantity, after controlling for covariates and two-way interactions of covariates with model predictors (Table 3). Results demonstrated that, contrary to hypotheses, *ADH1B*3* did not moderate the indirect effect of discrimination on alcohol quantity through coping drinking motives. That is, associations of discrimination with coping drinking motives or alcohol quantity did not differ as a function of *ADH1B*3* after controlling for model covariates (i.e., no gene-environment interaction; b = −0.02, β = −.00, p = .62 and b = 0.04, β = .02, p = .07, respectively). There also were no significant interactions of covariates with discrimination or *ADH1B*3* on coping drinking motives or alcohol quantity (bs = −1.20 to 0.48, βs = −.50 to .12, ps = .08 to .98) allowing for the interpretation of predictor and covariate main effects.

Discrimination was not associated with coping drinking motives ($b = 0.03$, β = .10, $p = .26$) or alcohol quantity ($b = -0.02$, β = −.14, $p = .11$), regardless of *ADH1B*3*. Specifically, there was no direct effect of discrimination on alcohol quantity ($B = -0.02$[–0.06,0.01]) and no indirect effect of discrimination on alcohol quantity through coping drinking motives ($B = 0.00$[–0.002,0.02]). Instead, there was an indirect effect of negative life events on alcohol quantity through coping drinking motives ($B = 0.04$[0.01,0.10]). Greater negative life events were associated with greater coping drinking motives ($b = 0.32$, β = .22, $p = .01$), which in turn were associated with greater alcohol quantity ($b = 0.13$, β = .23, $p < .001$). There was no direct effect of negative life events on alcohol quantity ($B = -0.03$[–0.15,0.09]). Finally, regarding additional covariates, students who identified as male reported greater average alcohol quantity than students who identified as female ($b = 1.65$, β = .68, $p < .001$).

Discussion

The present study aimed to contribute to limited research on within-group differences in alcohol behaviors among Black college students. Findings demonstrated that, contrary to hypotheses, associations of discrimination with coping drinking motives or alcohol quantity did not change as a function of *ADH1B*3* after controlling for potential confounding main and interaction effects involving gender, age, and negative life events. Instead, regardless of *ADH1B*3* presence, greater experience of past-year negative life events was associated with greater levels of coping drinking motives that, in turn, were associated with greater past-month alcohol quantity. Findings represent an important step toward better characterizing the likely complex interplay of alcohol risk and resilience factors within Black college drinkers.

Racial/ethnic discrimination may be associated with drinking behavior in more complex and nuanced ways than modeled in the present study. For example, some Black students may attempt to cope with discrimination-related stress through drinking while others instead withdraw from social interactions in an effort to reduce future discriminatory experiences. Social withdrawal may decrease students' exposure to alcohol-promoting peers and/or access to alcohol, thereby limiting their alcohol consumption. Future research could explore whether discrimination alters social interaction patterns, exposure to alcohol-promoting peers, accessibility of alcohol, or additional alcohol-protective mechanisms that may operate in opposing directions to any coping-motivated drinking pathways.

Negative life events may be a particularly poignant source of coping-motivated drinking among Black college students. Findings support negative reinforcement-based models of alcohol consumption. Specifically, stress response dampening theory suggests that individuals exposed to stressful situations drink alcohol to reduce stress and that such coping-motivated drinking is reinforced by the stress-dampening effects of alcohol.[5] When experiencing negative life events, Black students may turn to alcohol in an effort to relieve associated negative affect. Negative life events endorsed by students in the present study were wide-ranging, including trouble with friends, academic difficulty, and major illness or injury. Future research could explore whether specific stressors are more strongly associated with coping-motivated drinking. Efforts could also examine whether prevention/

intervention delivered in the aftermath of stressful experiences might help reduce adverse coping-motivated drinking among Black students.

Coping-motivated drinking was demonstrated across the present sample of Black students regardless of *ADH1B*3*. Findings may suggest that motivations to cope with negative life experiences through alcohol use could overpower any protection conferred by heightened susceptibility to alcohol's negative physiological effects. That is, results suggest coping-motivated drinking may occur regardless of the examined alcohol metabolism gene variant and, notably, do not suggest or assume any link between genetics and negative life events or discrimination. Findings align with prior research that demonstrated no differences in adult drinking behavior following childhood stress as a function of *ADH1B*3*.[14] However, the mechanism proposed for any *ADH1B*3*-based protection against coping-motivated drinking requires repeated exposure to negative physiological effects of alcohol consumption that serve to reduce alcohol's reinforcing value.[13] The present study was unable to examine such temporal processes given the limitations of cross-sectional data. Future research could explore whether genetic differences emerge when modeling coping-motivated drinking over time as a function of drinking history. Nevertheless, overall, present findings converge with prior research to suggest that alcohol metabolism gene variants may not represent a source of inherited individual differences in alcohol behavior in response to stress among Black college students, in contrast to differences observed as a function of serotonin and corticotropin-releasing hormone gene variants.[16,17]

Findings should be interpreted with respect to several limitations and future directions. First, analyses were based upon cross-sectional data, and causal relationships should not be inferred. Future prospective research should better delineate the temporal orderings among these constructs, and well-controlled experimental work could better test effects of various stressors on alcohol behavior as a function of genetics. Second, despite testing a candidate gene with established biological and empirical relevance to alcohol behavior interacting with a salient environmental stressor, concerns of potential false positive or negative findings necessitate replication efforts. Third, students self-reported on their discrimination experiences, alcohol cognitions, and alcohol quantity. Despite reminding participants of data confidentiality and providing memory aids for alcohol use, over-/under-reporting and recall bias may have influenced results. Relatedly, the original data collection conflated biological sex with gender such that the impact of potential gender misclassification remains unknown. Fourth, *ADH1B*3* noncarriers may have been oversampled relative to carriers; *ADH1B*3* can increase unpleasant physiological effects of alcohol making carriers less likely to report current drinking and meet study eligibility criteria. However, allele prevalence was within Hardy-Weinberg equilibrium, suggesting any selection bias may have been minimal. Finally, future research should examine generalizability of results to Black students in more diverse campus communities (e.g., historically Black colleges and universities), non-college-attending Black young adults, and Black secondary students to evaluate support for broad coping-motivated drinking interventions.

Findings highlights the potential value of universal interventions to reduce deleterious alcohol behaviors associated with stressful life experiences among Black college students. Culturally competent mental health services and coping skills training may help Black

students establish healthy coping strategies and safe outlets for managing stressful experiences in an effort to reduce coping-motivated drinking. Further, findings reveal relatively high rates of racial/ethnic discrimination experienced by Black college students, and discrimination has demonstrated associations with adverse health outcomes. The aforementioned clinical approaches may be adapted to help Black students also manage racially-specific stressors, although such mitigating efforts cannot eradicate discrimination and efforts to do so require large-scale societal advances to enhance this health disparity population.

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References

1. O'Malley PM, Johnston LD. Epidemiology of alcohol and other drug use among American college students. J Stud Alcohol Suppl. 2002;Suppl14:23–39.
2. Witbrodt J, Mulia N, Zemore SE, Kerr WC. Racial/ethnic disparities in alcohol-related problems: Differences by gender and level of heavy drinking. Alcohol Clin Exp Res. 2014;38:1662–1670. [PubMed: 24730475]
3. Brondolo E, ver Halen NB, Pencille M, Beatty D, Contrada RJ. Coping with racism: A selective review of the literature and a theoretical and methodological critique. J Behav Med. 2009;32:64–88. [PubMed: 19127420]
4. Clark R, Anderson NB, Clark VR, Williams DR. Racism as a stressor for African Americans: A biopsychosocial model. Am Psychol. 1999;54:805–816. [PubMed: 10540593]
5. Sher KJ. Stress response dampening. In: Blane HT, Leonard KE, eds. Psychological Theories of Drinking and Alcoholism. Guilford; 1987:227–271.
6. Desalu JM, Goodhines PA, Park A. Racial discrimination and alcohol use and negative drinking consequences among Black Americans: A meta-analytical review. Addiction. 2019;114:957–967. [PubMed: 30714661]
7. Pittman DM, Kaur P. Examining the role of racism in the risky alcohol use behaviors of Black female college students. J Am Coll Health. 2018;66:310–316. [PubMed: 29448892]
8. Metzger IW, Salami T, Carter S, et al. African American emerging adults' experiences with racial discrimination and drinking habits: The moderating roles of perceived stress. Cultur Divers Ethnic Minor Psychol. 2018;24:489–497. [PubMed: 29975077]
9. Boynton MH, O'Hara RE, Covault J, Scott D, Tennen H. A mediational model of racial discrimination and alcohol-related problems among African American college students. J Stud Alcohol Drugs. 2014;75:228–234. [PubMed: 24650816]
10. Geels LM, Bartels M, van Beijsterveldt TCEM, et al. Trends in adolescent alcohol use: Effects of age, sex and cohort on prevalence and heritability. Addiction. 2012;107:518–527. [PubMed: 21831193]
11. Rutter M, Moffitt TE, Caspi A. Gene–environment interplay and psychopathology: Multiple varieties but real effects. J Child Psychol Psychiatry. 2006;47:226–261. [PubMed: 16492258]
12. Edenberg HJ. The genetics of alcohol metabolism: Role of alcohol dehydrogenase and aldehyde dehydrogenase variants. Alcohol Res Health. 2007;30:5–13. [PubMed: 17718394]
13. Hendershot CS, Witkiewitz K, George WH, et al. Evaluating a cognitive model of ALDH2 and drinking behavior. Alcohol Clin Exp Res. 2011;35:91–98. [PubMed: 21039630]
14. Sartor CE, Wang Z, Xu K, Kranzler HR, Gelernter J. The joint effects of ADH1B variants and childhood adversity on alcohol related phenotypes in African-American and European-American women and men. Alcohol Clin Exp Res. 2014;38:2907–2914. [PubMed: 25410943]

15. Kaufman J, Yang B-Z, Douglas-Palumberi H, et al. Genetic and environmental predictors of early alcohol use. Biol Psychiatry. 2007;61:1228–1234. [PubMed: 17123474]
16. Kranzler HR, Scott D, Tennen H, et al. The 5HTTLPR polymorphism moderates the effect of stressful life events on drinking behavior in college students of African descent. Am J Med Genet B Neuropsychiatr Genet. 2012;159B:484–490. [PubMed: 22488930]
17. Goyal N, Aliev F, Latendresse SJ, et al. Genes involved in stress response and alcohol use among high-risk African American youth. Subst Abus. 2016;37:450–458. [PubMed: 26751645]
18. Kendler KS, Gardner C, Dick DM. Predicting alcohol consumption in adolescence from alcohol-specific and general externalizing genetic risk factors, key environmental exposures and their interaction. Psychol Med. 2011;41:1507–1516. [PubMed: 20942993]
19. Desalu JM, Kim J, Zaso MJ, et al. Racial discrimination, binge drinking, and negative drinking consequences among Black college students: Serial mediation by depressive symptoms and coping motives. Ethn Health. 2019;24:874–888. [PubMed: 28931304]
20. Desalu JM, Zaso MJ, Kim J, Belote JM, Park A. Interaction between ADH1B*3 and alcohol-facilitating social environments in alcohol behaviors among college students of African descent. Am J Addict. 2017;26:349–356. [PubMed: 28494133]
21. Zaso MJ, Desalu JM, Kim J, Suryadevara K, Belote JM, Park A. Interaction between the ADH1B*3 allele and drinking motives on alcohol use among Black college students. Am J Drug Alcohol Abuse. 2018;44:329–338. [PubMed: 28662358]
22. Contrada RJ, Ashmore RD, Gary ML, et al. Measures of ethnicity-related stress: Psychometric properties, ethnic group differences, and associations with well-being. J Appl Soc Psychol. 2001;31:1775–1820.
23. Cooper ML. Motivations for alcohol use among adolescents: Development and validation of a four-factor model. Psychol Assess. 1994;6:117–128.
24. Sobell LC, Sobell MB. Timeline follow-back: A technique for assessing self-reported alcohol consumption. In: Measuring Alcohol Consumption: Psychosocial and Biochemical Methods. Humana Press; 1992:41–72.
25. Borsari B, Neal DJ, Collins SE, Carey KB. Differential utility of three indexes of risky drinking for predicting alcohol problems in college students. Psychol Addict Behav. 2001;15:321–324. [PubMed: 11767264]
26. Clements K, Turpin G. The Life Events Scale for Students: Validation for use with British samples. Pers Individ Differ. 1996;20:747–751.
27. Muthén LK, Muthén BO. Mplus User's Guide. 8th ed. 1998–2017.
28. Edwards JR, Lambert LS. Methods for integrating moderation and mediation: A general analytical framework using moderated path analysis. Psychol Methods. 2007;12:1–22. [PubMed: 17402809]
29. Hayes AF. Introduction to Mediation, Moderation, and Conditional Process Analysis: A Regression-Based Approach. Guilford; 2013.
30. Winer ES, Cervone D, Bryant J, McKinney C, Liu RT, Nadorff MR. Distinguishing mediational models and analyses in clinical psychology: Atemporal associations do not imply causation. J Clin Psychol. 2016;72:947–955. [PubMed: 27038095]
31. Keller MC. Gene × environment interaction studies have not properly controlled for potential confounders: The problem and the (simple) solution. Biol Psychiatry. 2014;75:18–24. [PubMed: 24135711]
32. MacKinnon DP, Lockwood CM, Williams J. Confidence limits for the indirect effect: Distribution of the product and resampling methods. Multivariate Behav Res. 2004;39:99–128. [PubMed: 20157642]
33. Lipsey MW, Wilson DB. Practical Meta-Analysis. Sage; 2001.
34. Preacher KJ, Rucker DD, Hayes AF. Addressing moderated mediation hypotheses: Theory, methods, and prescriptions. Multivariate Behav Res. 2007;42:185–227. [PubMed: 26821081]

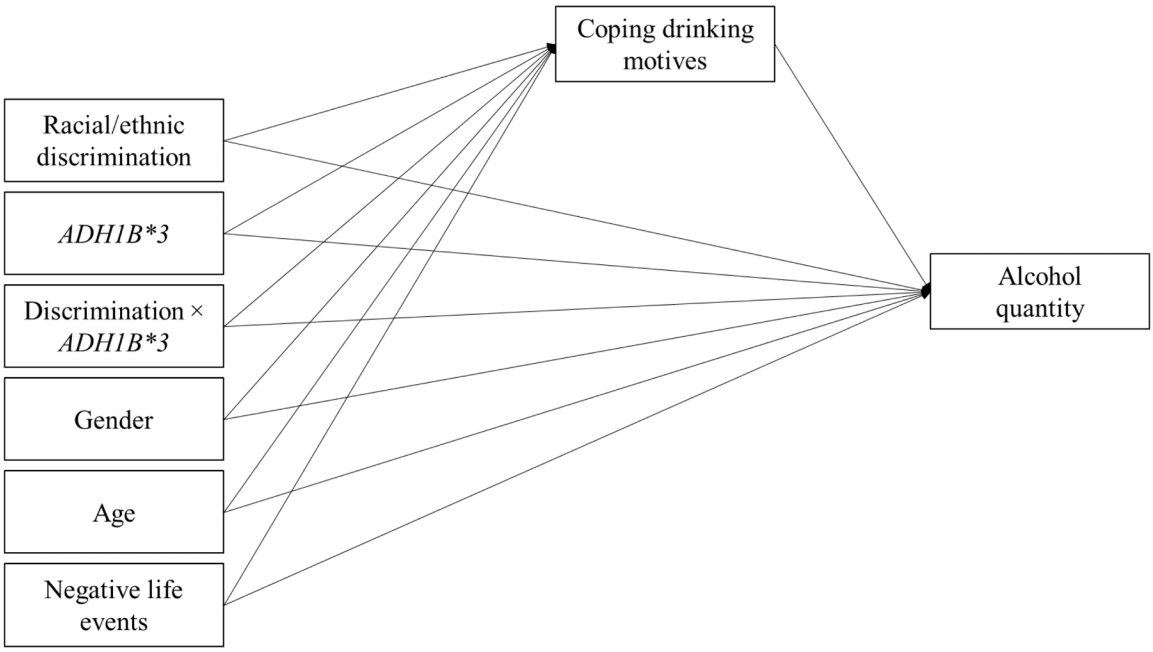

Figure 1.

Path model examining associations of racial/ethnic discrimination, *ADH1B*3*, and their interaction with coping drinking motives and alcohol quantity. Models also controlled for two-way interaction terms involving each covariate with both discrimination and *ADH1B*3* (i.e., discrimination × covariate, *ADH1B*3* × covariate), although these paths are not shown for simplicity.

Table 1.

Percentages and Means (with Standard Deviations) of Study Variables as a Function of *ADH1B*3*

Variable (observed range)	Total sample (*N* = 241)	*ADH1B*3* noncarriers (*n* = 158)	*ADH1B*3* carriers (*n* = 83)	Test statistic comparing *ADH1B*3* groups
Gender (0 = female; 1 = male)	34%	37%	28%	$\chi^2(1) = 2.25$
Age (18 – 53)	20.04 (4.11)	20.03 (3.81)	20.06 (4.66)	$t(237) = -0.05$
Negative life events (0 – 14)	3.83 (2.89)	3.88 (2.91)	3.72 (2.87)	$t(239) = 0.40$
Racial/ethnic discrimination (17 – 86)	36.87 (14.94)	36.97 (15.10)	36.66 (14.71)	$t(239) = 0.16$
Coping drinking motives (5 – 24)	9.07 (4.20)	9.30 (4.41)	8.61 (3.74)	$t(239) = 1.21$
Alcohol quantity (1 – 271)	27.54 (32.02)	29.64 (34.44)	23.54 (26.54)	$t(239) = 1.41$

N = 239 – 241, due to two cases with missing data on age.

Table 2.

Bivariate Correlations among Study Variables for *ADH1B*3* Carriers (Shown Above the Diagonal) and Noncarriers (Shown Below the Diagonal)

	Gender	Age	Negative life events	Racial/ethnic discrimination	Coping drinking motives	Alcohol quantity
Gender	–	.05	−.05	**.32****	−.00	**.22***
Age	.01	–	−.09	−.06	−.04	−.02
Negative life events	**−.16***	.05	–	.15	**.25***	.09
Racial/ethnic discrimination	.09	.03	**.30*****	–	.14	.20
Coping drinking motives	−.15	.11	**.29*****	**.23****	–	**.30****
Alcohol quantity	**.27*****	**.17***	−.09	−.10	.09	–

N = 239 – 241, due to two cases with missing data on age. Pearson's correlation coefficients presented for associations between two continuous variables and point-biserial coefficients presented for associations between a continuous and dichotomous variable. Correlations significant at $p < .05$ are shown in bold.

Table 3.

Path Model Examining Associations Among Racial/Ethnic Discrimination, Coping Drinking Motives, and Alcohol Quantity as a Function of *ADH1B*3*

Predictor	Coping drinking motives			Alcohol quantity		
	b	β	*p*	*b*	β	*p*
Racial/ethnic discrimination	0.03	.10	.26	−0.02	−.14	.11
*ADH1B*3*	−0.98	−.23	.13	−0.05	−.02	.88
Coping drinking motives	--	--	--	**0.13**	**.23**	**<.001**
Racial/ethnic discrimination × *ADH1B*3*	−0.02	−.00	.62	0.04	.02	.07
Gender	−1.18	−.28	.06	**1.65**	**.68**	**<.001**
Gender × Racial/ethnic discrimination	0.04	.01	.18	0.00	.00	.98
Gender × *ADH1B*3*	0.48	.12	.63	−1.20	−.50	.08
Age	0.11	.10	.40	0.08	.13	.07
Age × Racial/ethnic discrimination	0.00	.01	.94	0.00	.07	.42
Age × *ADH1B*3*	−0.13	−.03	.43	−0.07	−.03	.47
Negative life events	**0.32**	**.22**	**.01**	−0.03	−.04	.58
Negative life events × Racial/ethnic discrimination	0.01	.05	.44	−0.00	−.03	.56
Negative life events × *ADH1B*3*	−0.04	−.01	.83	0.09	.04	.38

N = 239, due to two cases with missing data on age. Coefficients significant at $p < .05$ are shown in bold.