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REGULAR ARTICLE

Genetic, Psychological, and Personal Network Factors Associated With Changes in Binge Drinking Over 2 Years Among Mexican Heritage Adolescents in the USA

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Abstract

Background Despite prevalent binge drinking and alcohol-dependent symptoms among Hispanics, few studies have examined how multidimensional factors influence Hispanic adolescents' binge drinking.

Purpose This study examines the effects of genetic, psychological, and social network factors on binge drinking over time among Mexican heritage adolescents in the USA and whether there are correlations among genetic variants that are associated with binge drinking and psychological and network characteristics.

Methods Mexican heritage adolescents (n = 731) participated in a longitudinal study, which included genetic testing at baseline, alcohol use assessments at first and second follow-ups, and questionnaires on sensation seeking, impulsivity, and peer and family network characteristics at second follow-up. Logistic regression and Spearman correlation analyses were performed.

Results After adjusting for demographic characteristics, underlying genetic clustering, and binge drinking at first follow-up, two genetic variants on tryptophan hydroxylase 2 (TPH2; rs17110451, rs7963717), sensation seeking and impulsivity, and having a greater fraction of peers who drink or encourage drinking alcohol were associated with greater risk whereas another genetic variant on TPH2 (rs11178999) and having a greater fraction of close family relationships were associated with reduced risk for binge drinking at second follow-up. Genetic variants in TPH1 (rs591556) were associated with sensation seeking and impulsivity, while genetic variants in TPH2 (rs17110451) were associated with the fraction of drinkers in family.

Conclusions Results reveal that genetic variants in the serotonin pathway, behavioral disinhibition traits, and social networks exert joint influences on binge drinking in Mexican heritage adolescents in the USA.

Keywords Binge drinking • Hispanic adolescents • Genetic risk • Sensation seeking • Impulsivity • Peer and family network

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Binge drinking, defined as consuming five or more drinks for men and four or more for women within about 2 hr [1], is a popular form of alcohol use among youth. Adolescent binge drinking is particularly problematic due to its link to negative consequences, including academic problems, risky sexual behavior, physical injuries, driving under the influence, and death [2], as well as to the risk for developing alcohol dependence in adulthood [3–6]. Emerging health research using the biopsychosocial model of health suggests that alcohol use is determined by multidimensional factors including biological, genetic, psychological, and sociocultural characteristics [7, 8]. Few studies that have examined these multidimensional factors for alcohol use among Hispanic

adolescents, despite the higher prevalence of alcohol dependence symptoms in Hispanic adults than non-Hispanic Whites [9].

Alcohol Drinking Among Hispanic Adolescents

Hispanic adolescents, the largest and fastest growing ethnic group in the USA [10], report a higher prevalence of binge drinking and alcohol-related negative consequences, compared with youth from other ethnic groups in the USA. Similar to the binge drinking rate among Hispanic adults [11], 13.5% of Hispanic adolescents (i.e., youth between Years 12 and 17) report engaging in binge drinking during the past month, which is larger than other ethnic minority youth including Native Hawaiians or other Pacific Islanders (12.1%), Black (8.4 %), and Asian (7.6 %) adolescents but is equivalent with non-Hispanic White (16.8%) and American Indian adolescents (13.9%) [11]. Despite the similarities in the rates of binge drinking between Hispanic and non-Hispanic Whites, Hispanic adults who report drinking alcohol tend to experience more symptoms of alcohol dependence than non-Hispanic Whites [9, 12] and have the greatest risk for alcohol-related injuries after alcohol consumption compared with adults of other ethnic groups in the USA. [13]. Therefore, in the present study, we seek to inform the development of alcohol prevention programs designed to reduce health disparities by identifying genetic, psychological, and social factors associated with binge drinking among Hispanic adolescents in the USA.

Impact of Genetic Variants on Adolescent Binge Drinking

Previous studies have shown that genetic variants substantially contribute to the risk of alcohol dependence in adulthood [14]. Candidate genes related to alcohol dependence can be classified into three categories with distinctive pathways underlying the genes and alcohol dependence connection: These are genes related to (a) alcohol metabolism, (b) stress responsivity, and (c) behavioral disinhibition [15, 16]. The first category involves the functional variants on alcohol metabolism genes, including alcohol dehydrogenase (ADH) and aldehyde dehydrogenase (ALDH), which have been widely studied for their protective influence against excessive drinking and alcohol dependence [17]. The second category of candidate genes, related to the stress response system, is considered to play an important role in the initiation and maintenance of binge-drinking behavior and alcohol dependence via the dysregulated activation of the brain's stress and antistress system, notably through actions by corticotropin-releasing hormone (CRH) but also catechol-*O*-methyl transferase (COMT) and neuropeptide Y (NPT) [18, 19]. Finally, the third category of genetic variants in the serotonin and dopamine pathways appears to influence behavioral disinhibition—the inability to inhibit socially restricted actions including alcohol and other substance abuse [20, 21]. Notably, previous studies suggest that polymorphisms on tryptophan hydroxylase (TPH), serotonin transporter (SERT), and serotonin receptor (HTR) genes in the serotonergic system and polymorphisms on dopamine receptors (DRD), dopamine β-hydroxylase (DBH), and tyrosine hydroxylase (TH) have been linked with risk for binge drinking and alcohol dependence in animal and human studies [16, 22–24]. Although the heterogeneity in the relative frequency of genetic variants in candidate genes across populations has been discovered, most studies examined the effects of those genetic variants on alcohol dependence in adults of European heritage. Only a few studies have examined the effects of the polymorphisms in ADH and ALDH on alcohol dependence in adults of other ethnic backgrounds such as Mexican Americans [25, 26].

Effects of Psychological Traits and Social Environments on Adolescent Binge Drinking

Genetic heritability accounts for only a part of the risk for developing alcohol dependence. Psychological traits related to cognitive and emotional susceptibility to substance abuse and social relationships with peers and family are also crucial factors for the initiation and development of adolescent substance abuse [27]. Among various psychological traits, impulsivity and sensation seeking have received the most attention due to their influence on deviant and risky behaviors, including alcohol drinking. Impulsivity can be defined as the tendency to act in an unplanned manner to satisfy a desire and not thinking through the potential impact before carrying out actions or making statements [28, 29]. Sensation seeking can be defined as "the desire for new kinds of sensory experiences and the seeking of excitement and aversion for monotony or boredom by engaging in risky and adventurous activities" [30]. Greater levels of impulsivity and sensation-seeking propensity appear to be consistently associated with neurological activations co-occurring with development of alcohol dependence [16, 29, 31].

While genetic predispositions and psychological traits explain a large amount of risk for heavy alcohol drinking and alcohol dependence at an individual level, social networks within which an individual adolescent is embedded have important implications for opportunities to uptake risky behaviors, including binge drinking. Assessing ego-centered personal networks of adolescents allows us to identify the specific attributes of social ties within one's close personal network that may promote

or prevent alcohol abuse [32]. A few studies that have applied social network analysis to examine social contexts of adolescent alcohol abuse focused almost exclusively on peer influences within networks. For example, a previous study revealed that adolescents tend to have ties with peers who have the same drinking status (a homophily effect) [33]. Also, network properties such as geographic proximity to peer drinkers and centrality in the network were associated with more alcohol use among adolescents [32]. By contrast, few studies on social network structure and adolescent drinking have examined the effects of family network characteristics on adolescent alcohol drinking despite the known impact of family relationships on adolescent alcohol drinking via modeling, permissive parenting, and family conflict and support [27]. To address this gap in the literature, the current research aims to examine the broader social environment by focusing on how personal networks, which contain a mixture of family and peer relationships, may influence binge drinking among adolescents.

The purpose of this study is to identify the genetic, psychological, and social factors associated with the transition to binge drinking over 2 years among Mexican heritage adolescents. Particularly, this study tested whether changes in binge drinking over 30 months (mean = 29.65 and *SD* = 5.21), from first to second follow-up, are affected by genetic variants, psychological traits of sensation seeking and impulsivity, and close personal networks involving family and friends in terms of whether a network member is a drinker, encourages alcohol drinking, and is perceived to have a close relationship with a participant. In addition, gender moderation effects were examined to determine whether genetic, psychological, and personal network factors affect boys and girls with different magnitudes or directions. Also, we explored whether genetic variants that are associated with binge drinking are correlated with psychological traits and personal network characteristics, given that such gene-by-trait or gene-by-social environment correlations may lead to over- or underestimations of the genetic effects on binge drinking [34]. Previous studies suggest that Gene × Environment interactions can be defined as "a different effect of an environmental exposure on disease risk in persons with different genotypes," or, equivalently, "a different effect of a genotype on disease risk in persons with different environmental exposures" [35]. Gene × Environment interactions can be accurately examined only if the genetic variants of interests are independent of environmental factors (e.g., genetic susceptibility to alcohol dependence and randomly assigned roommates in college dormitory). Gene × Environment interactions were not examined because the genetic variants (e.g., having risk alleles on a dopamine receptor) we examined are not independent of social network characteristics (e.g., genetic similarity in dopamine receptors between peers increase the likelihood of them being close and drinking alcohol together) we included in the analysis [36].

Methods

Study Design and Participants

Participants were recruited through their parents who took part in a population-based cohort study, the Mano-a-Mano Mexican American Cohort Study [37]. Participants were recruited and enrolled, completed the baseline survey, and provided saliva samples for genetic testing in 2005–2006. Two comprehensive follow-up surveys were conducted in 2008–2009 and 2010–2011. All parents of our participants self-identified as Mexican or Mexican-American and were initially recruited into the cohort from Houston, Texas, using probability random digit dialing, door-to-door recruitment, intercepts, and network approaches. Of the 1,425 parents or legal guardians with age-eligible adolescents (i.e., between 11 and 13 years of age) contacted, 90% percent (n = 1,328) agreed to enroll their child in the study. The first and second follow-up surveys retained n = 1,154 (86.9% of baseline) and n = 1,002 participants (75% of baseline), respectively. Both follow-up surveys, but not the baseline survey, included the assessments of alcohol use. A detailed description of the study design and nested adolescent cohort recruitment has been previously published [38]. All parents and legal guardians of participants provided informed consent, and all participants provided their informed assent. The study was approved by the Institutional Review Boards at the University of Texas MD Anderson Cancer Center and UTHealth Science Center in Houston.

Trained interviewers visited each participant's home to administer the survey, following identical procedures, at baseline and two follow-ups. English and Spanish versions of the surveys were available. Handheld personal digital assistants were provided to participants to gather their survey responses in a private space at home. In the current analysis, we used data on alcohol use in the past 30 days and binge drinking in the past 30 days provided from both first and second follow-up surveys, while we used data on psychological traits and personal networks provided at the second follow-up survey.

Measures

DNA collection

Saliva samples of participants (n = 1,274) were collected in Oragene vials (DNA Genotek, Ottawa, Ontario,

Canada). DNA extraction was performed using a DNA purifying solution with alcohol precipitation according to the manufacturer's protocol. The median yield of DNA from 2 ml of saliva captured in 2 ml of OrageneDNA was 110 µg.

Single nucleotide polymorphism selection

A total of 58 candidate genes were identified from published reviews and PubMed searches of human genetic studies using the following keywords—sensation seeking, risk taking, gambling, smoking onset, and initiation for the purpose of a larger study [39]. The candidate genes were cross referenced with the Gene Oncology Data Base (http://pid.nci.nih.gov/) and Kegg Pathway (http://www.genome.jp/kegg/). The set of 672 tag SNPs in 58 candidate genes were selected from the International HapMap project (Release 21 with NCBI build 36; http://www.hapmap.org) to maximize the genetic variation captured for each gene. The following selection criteria for SNP location were also considered—located in the respective genes or within 10 kb upstream or downstream of the gene ends to cover the regulatory regions, minor allele frequency (MAF) >5% in various ethnic groups, and not in linkage disequilibrium (LD) of $r^2 < 0.80$ with other tag SNPs. Also, SNPs in coding and regulatory regions including promoter, splicing, and 5'-UTR and 3'-UTR were included. More information on gene selection and genotype information have been previously described [39].

Candidate genes related to alcohol dependence can be classified into three categories with distinctive pathways underlying the genes and alcohol dependence connection: These are genes related to (a) alcohol metabolism, (b) stress responsivity, and (c) behavioral disinhibition. We conducted an additional review for the current analysis and identified 23 candidate genes with 316 tag SNPs specifically associated with these three mechanisms.

Genotyping

An Illumina GoldenGate assay was designed for genotyping 672 SNPs (Illumina, Inc.). Ninety-three percent of the SNPs had Illumina SNP scores of >0.6. A total of 1,274 samples with DNA 250ng were genotyped following the standard 3-day Illumina protocol. The BeadArray Reader (Illumina, Inc.) was used to call the array data. Cluster definitions for each SNP were determined using Illumina BeadStudio Genotyping module v. 2.3.41. When a quality score (i.e., Gencall value) of a genotype is over 95%, genotype calls were made, and 1.2% of the calls (8 of 672) were missing. Therefore, we used 664 SNPs with genotype data for analysis. The concordance of SNP genotype calls was greater than 99% when examined with 70 blind duplicate pairs.

Survey Measures

Demographic information

Participants were asked about their age, gender, country of birth, linguistic acculturation, and subjective social status at baseline. Linguistic acculturation was assessed using four items from the language use subscale of the brief acculturation scale for Hispanics [40]. The four items ask language used when reading, speaking at home, speaking with friends, and thinking with five response options, ranging from 1 = *only Spanish* to 5 = *only English*; a mean score higher than 4 is classified as high linguistic acculturation. Subjective social status was assessed using the MacArther Scale of Subjective Social Status–Youth Version, which uses a ladder to represent social status, and asks the respondent to place him or herself on the ladder relative to others at one's school [41].

Binge drinking and alcohol use in the past month

Binge drinking and alcohol use in the past month were measured at both follow-up surveys by two items from the Youth Risk Behavior Surveillance System [42]. Participants were asked "During the past 30 days, on how many days did you have 5 or more drinks of alcohol in a row?" for binge drinking and "During the past 30 days, on how many days did you have at least one drink of alcohol?" for alcohol use. Both items used seven response options ranging from 1 = *0 days* to 7 = *all 30 days*. The responses for both items were re-coded as dichotomous variables with 1 = *1 or more days of binge drinking or alcohol use in the last 30 days* and 0 = *no days*.

Psychological factors

Barratt Impulsivity Scale–11 was used to assess levels of motor (i.e., acting without thinking), cognitive (i.e., making quick cognitive decision), and nonplanning (i.e., lack of considerations for future) aspects of impulsivity among participants [28]. The scale includes 30 items with response options ranging from 1 to 4 for each item, with four indicating highest impulsivity. The internal consistency was Cronbach's $\alpha = .79$ with the current sample.

In addition, sensation seeking was assessed by the Sensation Seeking Scale for Children (SSSC) [43]. The SSSC includes 26 items that ask respondents to select one of two contrasting sensation-seeking statements that best describe them. Internal consistency of this measure was Cronbach's $\alpha = .78$ with this sample.

Personal network factors

To examine participants' ego-centered personal networks, we asked participants to fill out two "name-generators"

[44]. First, participants enumerated all the people who live with them in the same house. Second, participants listed their three best friends. For each enumerated network member, we asked the person's gender, age, alcohol drinking status, and whether the participant was engaged in alcohol-related social interactions—such as encouraging drinking alcohol—with the member, and general social relations such as closeness and conflict.

Statistical Analysis

Missing data analyses were conducted to examine whether missing cases at the second follow-up showed any systematic differences compared with cases that remained. Demographic characteristics were compared between binge drinkers and non–binge drinking peers, using chi-square tests for categorical variables and one-way ANOVA for continuous variables. Multivariate logistic regression was performed to examine the effects of genetic, psychological, and social network factors on the transition from non–binge drinking to binge drinking. Binge drinking variables were used as outcome variables in the regression models, and alcohol use variables were used in the descriptive analysis and the additional regression analyses to verify if filtering out those who never drank alcohol from non–binge drinkers influenced the results.

Before analyzing the genetic effects on binge drinking, the SNP data were screened for minor allele frequencies ≤5% and Hardy–Weinberg disequilibrium test. Then, a three-step procedure was used to examine the effects of the candidate genes in each of the three functional categories, on changes in binge drinking. First, each of the 262 candidate SNPs was tested for its effect on changes in binge drinking, controlling for age and gender to identify significant SNPs at $p \leq .05$ significance level. Because the mode of inheritance cannot be presumed, allelic data were coded into the number of minor alleles for two potential genetic models (additive and dominant model) and the better fitting model was selected. To verify the possible inflation of the Type I error rate due to multiple testing for the effects of SNPs, a q-value was calculated with the full set of p-values from logistic regression results for the effects of SNPs on binge drinking [45]. Next, principal component analysis was conducted with all available SNPs except for the significant SNPs identified in the previous logistic regressions (i.e., among the total 558, all but the significant 27 SNPs were included in the principal component analysis). The top two factor loadings from the principal component analysis were entered into the multivariate logistic regression models, which were used to control for potential stratification of genetic variants within our sample due to genetic admixture [46]. Finally, the significant SNPs, two principal components, age, gender, and subjective social status were entered together into a multivariate logistic regression model. The multivariate logistic regression model with genetic factors was reduced using a stepwise selection procedure based on the fit index Akaike information criterion (AIC) for the selection criteria to identify the final set of significant SNPs to be entered to the final model. The model for psychological factors or personal network factors for binge drinking at the second follow-up included sensation seeking and impulsivity or personal network variables respectively while adjusting for binge drinking at the first follow-up, age, gender, and subjective social status.

The final model was compared with models with only genetic variants, psychological factors, or personal network factors in terms of parameter estimates and model fit (AIC). To explore if there were associations between genetic variants and psychological/personal network factors (gene-trait/social environment correlations), Spearman correlation was conducted with significant SNPs and psychological/personal network factors.

Finally, power calculations were conducted to confirm if the sample size is sufficient to test the primary hypotheses of this study. The power calculations for genetic variants were calculated based on 66 binge drinkers in the second follow-up. The minimum detectable odds ratios were computed using PS Power and Sample Size Calculations Version 3.0 [47], assuming 80% power and a significance level of 0.05 with the use of two-sided Fisher's exact test. For a common genetic variant with a minor allele frequency of 0.4, we have at least 80% power to detect an odds ratio of ≥2.05 or ≤0.45 between the genetic variant and change in adolescent binge drinking. The sample size (n = 741) was sufficient to examine the final model including genetic variants, psychological, and personal network factors for binge drinking from a general linear modeling point of view.

Results

Data Screening and Missing Data Analyses

Of 1,274 adolescents who provided DNA samples at baseline, 933 who reported their binge drinking at both follow-ups were included in the genetic analysis and 731 who provided data of all key study information including psychological scales and personal network surveys in addition to DNA samples and binge drinking were included in the analysis for the final model with genetic, psychological, and personal network factors. The final set of significant SNPs in the multivariate logistic regression model was identical whether we used the sample with n = 933 or 731. Due to the significant reduction in the sample size at the second follow-up, we conducted a battery of tests to evaluate the effect of missing data on our results. None of these tests revealed any effect that would modify the main findings of this study. First, there were no significant

differences in genetic factors related to missingness (i.e., the missingness appears to be completely at random based on bootstrap sampling results). To evaluate whether exclusion by casewise deletion was related to covariates, we also ran pairwise *t*-tests (for continuous) or tests of proportionality (for discrete data) to see whether exclusion versus inclusion differed in mean variation. Along the margins, excluded individuals appeared to be slightly more likely to have used alcohol at baseline. However, a test of equality of proportions demonstrates that this difference is not statistically significant ($\delta = 0.0397$, $\chi^2 = 2.1815$, $df = 1$, $p = .1397$). We conclude that there were no substantive differences between those participants who were excluded and those who were included in the final analyses.

Participant Characteristics

Table 1 presents the participant characteristics for adolescent binge drinkers and non–binge drinkers at the first and second follow-ups. About 6% ($n = 43$) and 9% ($n = 66$) of participants reported one or more binge drinking episodes in the past 30 days at the first and second follow-ups. Those who reported binge drinking at the first or second follow-up were more likely to be older ($p < .01$), report lower subjective social status ($p \leq .05$), and report drinking alcohol at the first ($p < .001$) and second follow-up ($p < .001$) compared with those who did not report binge drinking at any of the two follow-ups.

Effects of Genetic Variants on Changes in Binge Drinking

After screening out 108 of the total 664 SNPs (54 of 316 candidate SNPs) with minor allele frequencies ≤5% or Hardy–Weinberg disequilibrium test, 262 candidate SNPs and the total number of 556 SNPs were analyzed for the effects of the candidate genes on adolescent binge drinking. After adjusting for age, gender, and binge drinking at the first follow-up, each of the 27 SNPs was significantly associated with changes in binge drinking ($p \leq .05$). The *q*-value was calculated to evaluate the false discovery rate from conducting 262 tests (≤0.112), and the results of 27 significant SNPs had less than 5% of the false discovery rate ($q \leq 0.05$). Then, 531 SNPs, all 558 SNPs except the 27 significant SNPs, were reduced to

Table 1 Distribution of participant demographics and key study variables by binge drinking at first and second follow-ups ($n = 731$)

	Total	Binge drinking at first follow-up			Binge drinking at second follow-up		
	n (%)	Yes *n* (%)	No *n* (%)	*p*	Yes *n* (%)	No *n* (%)	*p*
Overall	731 (100.0)	43 (5.9)	688 (94.1)		66 (9.0)	665 (91.0)	
Age at the baseline (mean ± *SD*)	**11.82 ± 0.83**	**12.23 ± 0.84**	**11.79 ± 0.82**	**<.01**	**12.11 ± 0.83**	**11.79 ± 0.82**	**<.01**
Gender							
Male	334 (45.7)	26 (44.8)	308 (44.8)	.06	37 (56.1)	297 (44.7)	.10
Female	397 (54.3)	17 (55.2)	380 (55.2)		29 (43.9)	368 (55.3)	
Country of birth							
Mexico	188 (25.7)	9 (20.9)	179 (26)	.58	11 (16.7)	177 (26.6)	.11
USA	543 (74.3)	34 (79.1)	509 (74)		55 (83.3)	488 (73.4)	
Acculturation							
High	601 (82.9)	32 (74.4)	569 (83.4)	.18	8 (12.1)	116 (17.6)	.10[a]
Low	124 (17.1)	11 (25.6)	113 (16.6)		58 (87.9)	543 (82.4)	
Subjective social status (1 = worst, 10 = best), mean ± *SD*	**7.87 ± 1.43**	**7.34 ± 1.23**	**7.90 ± 1.44**	**<.01**	**7.43 ± 1.47**	**7.91 ± 1.42**	**≤.05**
Alcohol drinking in the past month							
First follow-up							
Yes	**91 (12.4)**	**41 (95.3)**	**50 (7.3)**	**<.001**	**30 (45.5)**	**61 (9.2)**	**<.001**
No	**640 (87.6)**	**2 (4.7)**	**638 (99.7)**		**36 (54.5)**	**604 (90.8)**	
Second follow-up							
Yes	**139** (19)	**30 (69.8)**	**109 (15.8)**	**<.001**	**64 (97)**	**590 (88.7)**	**<.001**[a]
No	**592 (81)**	**13 (30.2)**	**579 (84.2)**		**2** (3)	**75 (11.3)**	

The bold values indicate $p \leq .05$.

[a] χ^2 test result may be incorrect due to the small cell size (≤10) in one of four cross-tabulated cells.

two principal components that characterize the underlying genetic variation in our sample.

The multivariate logistic regression model with 27 SNPs as predictors, and binge drinking at the first follow-up, age, gender, subjective social status, and two principal components as controlling variables identified six SNP locations that remained significant at the $p \leq .05$ level including rs2097628 (DBH), rs2224721 (HTR2A), rs591556 (TPH1), rs11178999 (TPH2), rs17110451 (TPH2), and rs7963717 (TPH2).

Effects of Genetic, Psychological, and Personal Network Factors on Changes in Binge Drinking

As presented in Table 2, the final logistic regression analyses examined four models, including "Model 1" with genetic variants, "Model 2" with psychological traits, "Model 3" with personal network factors, and "Final Model" with genetic, psychological, and network factors. The final model with genetic, psychological, and personal network factors for binge drinking revealed that having the AA or AG variant of rs17110451 (TPH2) and having the CC or AC of rs7963717 (TPH2) were significantly associated with a 1.84 and 3 times increased risk for binge drinking, respectively. Having the AA or GA variant of rs11178999 (TPH2) was significantly associated with a 37% decreased risk for binge drinking. Among the psychological traits, both sensation seeking and impulsivity were associated with a 1.10 and 1.05 times increased chance of initiating binge drinking. Finally, having a greater fraction of peers who drink alcohol and peers who encourage alcohol drinking were significantly associated with 8.88 and 15.20 times increased risk for binge drinking, respectively. Having close ties with household members also remained significantly associated, with a 72% reduced risk for binge drinking.

The results of the final model were further verified after including only those who ever drank alcohol to see if having heterogeneous mixtures of never drinkers and moderate drinkers in the non–binge drinking status

Table 2 Logistic regression models predicting changes in adolescent binge drinking from the first to the second follow-up (n = 731)

	Model 1	Model 2	Model 3	Final model
	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)
Binge drinking at the first follow-up	**9.67 (4.61–20.36)*** **	**5.50 (2.58–11.56)*** **	**6.51 (2.99–14.03)*** **	**6.08 (2.52–14.70)*** **
Demographics				
Age	**1.40 (1.01–1.96)***	**1.48 (1.06–2.07)***	1.20 (0.84–1.72)	1.30 (0.89–1.89)
Gender	0.71 (0.40–1.24)	1.07 (0.59–1.93)	0.70 (0.39–1.26)	0.87 (0.45–1.69)
Social status	0.85 (0.70–1.03)†	0.94 (0.77–1.14)	0.85 (0.69–1.04)	0.91 (0.72–1.14)
Genetic factors				
rs2097628 (DBH)	0.41 (0.15–0.95)†	–	–	0.41 (0.14–1.04)†
rs2224721 (HTR2A)	1.46 (0.99–2.13)†	–	–	1.37 (0.88–2.11)
rs591556 (TPH1)	**0.60 (0.36–0.95)***	–	–	0.62 (0.36–1.05)†
rs11178999 (TPH2)	**0.63 (0.42–0.93)***	–	–	**0.63 (0.40–0.96)***
rs17110451 (TPH2)	**1.74 (1.05–2.88)***	–	–	**1.86 (1.07–3.27)***
rs7963717 (TPH2)	**2.55 (1.39–4.65)****	–	–	**3.00 (1.50–6.02)****
PC1[a]	1.00 (1.00–1.00)			0.83 (0.69–0.99)
PC2[a]	1.13 (0.99–1.29)†			**1.06 (0.88–1.27)***
Psychological traits				
Sensation seeking	–	**1.14 (1.07–1.23)*** **	–	**1.10 (1.01–1.19)***
Impulsivity	–	**1.05 (1.02–1.09)****	–	**1.05 (1.01–1.08)****
Personal network				
Family drinkers	–	–	1.29 (0.38–4.20)	1.10 (0.30–3.82)
Peer drinkers	–	–	**9.93 (4.88–20.45)*** **	**8.88 (4.04–19.88)*** **
Peer encouragement	–	–	**8.55 (1.42–55.79)***	**15.20 (2.12–110.95)****
Close family members	–	–	**0.30 (0.10–0.87)***	**0.28 (0.08–0.90)***
Close peers	–	–	1.16 (0.51–2.53)	1.02 (0.41–2.44)
AIC criteria	391.35	371.36	355.85	328.65

[a]PC1, PC2 = top two factor loadings from principal component analysis of all available SNPs except 27 SNPs.

†$.05 < p < 1$; *$p \leq .05$; **$p < .01$; ***$p < .001$.

may have obscured the results. All the significant factors in the final model remained significant at $p \leq .05$ after excluding never drinkers ($n = 379$).

Gender Moderation

Significant factors in the final model were further examined for moderation by gender. As presented in Fig. 1, gender appears to moderate the effect of the fraction of peers who drink ($b = -1.597$, $p \leq .05$) though the main effects of gender remain insignificant after adding gender moderation effects. Specifically, there is a greater effect of having a larger fraction of peers who drink on changes in binge drinking for boys than girls. Asymptotically, gender does not moderate the effect of peers on binge drinking until more than two-fifths of one's peers drink and the overall effects sizes were small (i.e., $x <$ 1e-4).

Correlations Among Genetic Variants, Psychological Traits, and Personal Network

Because there might be gene-by-trait or gene-by-social environment correlations among the genetic, psychological, and social factors for changes in binge drinking, the associations among genetic variants, psychological traits, and perceived personal network characteristics were explored. As presented in Table 3, having the TT or CT variant of rs591556 (TPH1) was significantly associated with higher levels of sensation seeking and impulsivity traits ($\rho = -0.11$, $p < .01$; $\rho = -0.09$, $p \leq .05$). In addition, participants with the CC or AC variant of rs17110451 (TPH2) had fewer numbers of alcohol drinkers in their family network ($\rho = -0.09$, $p \leq .05$) than those without. The rest of the SNPs were not associated with any psychological traits or personal network factors in the final model.

Discussion

The current study identified multidimensional risk and protective factors associated with transition to binge drinking, utilizing two follow-up surveys from an under-represented population of Mexican heritage adolescents in the USA. The current study found risk factors for the initiation of binge drinking among Mexican heritage adolescents including having the CC or AC variant of rs17110451 (TPH2), having the CC or AC variant of rs7963717 (TPH2), psychological traits of sensation seeking and impulsivity, and having a greater fraction of peer drinkers and peers who encourage drinking alcohol in one's social network. This study also revealed protective factors against the initiation

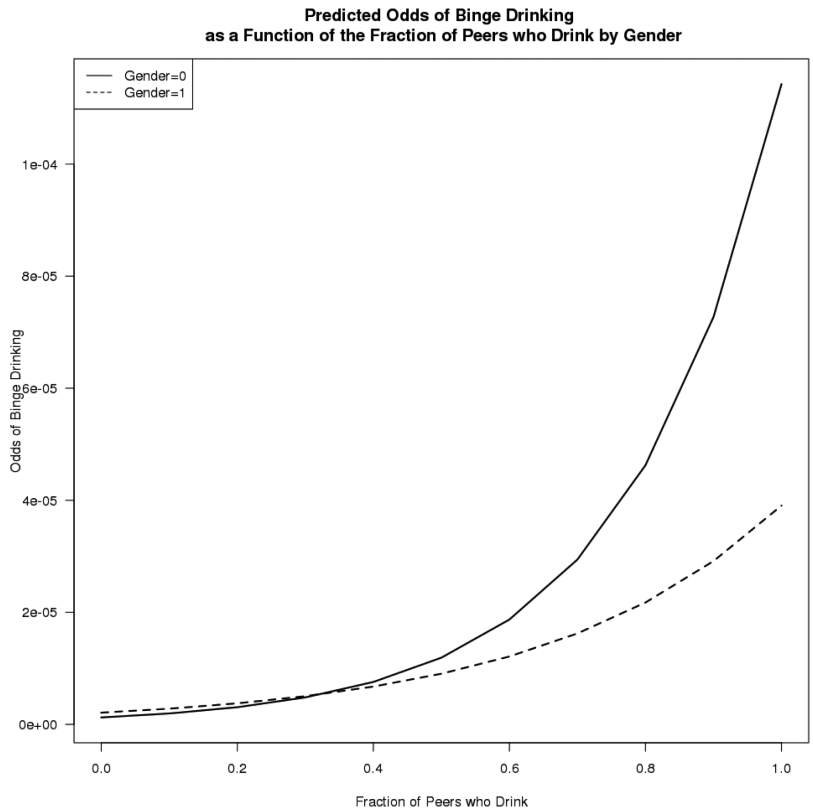

Fig. 1. Effects of gender moderation for the association between fraction of peers who drink alcohol and changes in binge drinking. Gender = 0 represents boys; Gender = 1 represents girls.

Table 3 Spearman correlations between genetic variants and psychosocial variables in the final model (n = 731).

	1	2	3	4	5	6	7	8	9	10	11	12	13
1. rs11178999	1												
2. rs17110451	**−.07***	1											
3. rs2097628	−.03	.01	1										
4. rs2224721	−.01	0	−.06	1									
5. rs591556	−.07	.04	.03	−.03	1								
6. rs7963717	**−.12*****	**−.23*****	−.01	−.01	0	1							
7. Impulsivity	−.07	−.05	−.01	.03	**−.09***	.04	1						
8. Sensation seeking	.05	−.04	.01	.01	**−.11****	.01	.26	1					
9. Family drinkers	0	**−.09***	−.04	.06	−.02	−.03	.07	.2	1				
10. Peer drinkers	−.01	−.02	−.01	.02	−.01	.03	**.11****	.25	.21	1			
11. Peer encouragement	−.03	−.06	−.01	.01	.01	.06	.06	**.1***	.03	.21	1		
12. Close family members	.02	0	.01	.01	.02	−.05	**−.08***	0	−.05	.01	**.08***	1	
13. Close peers	.01	−.04	−.06	−.06	0	.02	−.01	.06	.01	.05	**.09***	.32	1

*$p \leq .05$; **$p < .01$; ***$p < .001$.

of binge drinking, including genetic variants in TPH2 (rs11178999) and having a greater fraction of close family ties. Gender moderation effects showed that boys are more prone to be influenced by peer drinkers to adopt binge-drinking behaviors than girls. Also, gene-by-trait and gene-by-social environment correlation results demonstrated that having either an AA or GA variant of rs591556 (TPH1) was associated with lower levels of sensation seeking and impulsivity traits and having a lower fraction of drinkers in the family network. The findings of the current study indicate the role of biopsychosocial mechanisms in determining the onset of alcohol problems in Hispanic adolescents and may contribute to the development of novel strategies for reducing the health disparity in persistent alcohol problems among Hispanic population in the USA [12]. Also, this study is the first to utilize the ego-centered network survey that assessed the characteristics of social interactions surrounding binge drinking of Mexican heritage adolescents. Given the strong cultural influences on how social relationships are established and influence substance use in Hispanic adolescents [48], this study provides a unique perspective on the role of compositional characteristics of peer and family network influences on changes in health behaviors within the context of Mexican heritage adolescents with low to moderate levels of U.S. acculturation.

Among the candidate genes that have been linked to alcohol dependence, the current results suggest that the genetic polymorphism on TPH1 and 2 predict the risk for the transition from non–binge drinking to binge drinking. TPH1 and 2 are two isoforms of the rate limiting enzyme for biosynthesis of serotonin and catalyze the biopterin dependent monooxygenation of tryptophan to 5-hydroxytryptophan (5HT). TPH has been extensively studied for its association with impulsivity, psychiatric disorders, and alcohol dependence in non-Hispanic White population, but those associations have not been examined much in Hispanic or other populations [49]. Previous work on TPH polymorphisms showed that TPH1 A218C polymorphism (rs1800532) and TPH I7 A779C polymorphism (rs1799913) are associated with lower 5-hyroxyindoleacetic acid, serotonin metabolite. The low turnover rate of serotonin is found to be associated with deficit in impulse control, risk of suicidality, and alcoholism in a European sample [49, 50]. The present study supports the previous findings that variants in TPH (rs11178999, rs17110451, and rs7963717) are associated with risk for binge drinking [50] and extends them by reporting the associations between TPH variants (rs591556) and sensation seeking or impulsivity traits among Mexican heritage adolescents in the USA. Although the findings should be interpreted with caution due to the relatively small sample size for a genetic study and genetic admixture among people with Mexican heritage that might not have been fully captured by the current methodology, the association between TPH and binge drinking might be partially explained by the role of TPH polymorphisms (rs591556) in impulse control and sensation-seeking tendencies.

As expected, both sensation seeking and impulsivity traits slightly elevated the risk for changes from no binge drinking to binge drinking among Mexican heritage adolescents. The results are consistent with other studies including a meta-analysis with mostly non-Hispanic populations, and suggest that sensation seeking and impulsivity are risk factors for binge drinking and other types of substance abuse among adolescents across

different ethnic backgrounds [31, 51]. Although more studies are needed, this pattern may imply that personality traits of sensation seeking and impulsivity may help early identification of Mexican heritage adolescents who are more vulnerable to binge drinking than others.

Finally, the results suggest that social relationships with both peers and family members influence whether Mexican heritage adolescents initiate binge drinking. In particular, adolescents whose peers drink alcohol themselves and encourage drinking are 8–15 times more likely to initiate binge drinking. The findings are consistent with previous studies that exposure to alcohol drinking friends and selection of peers who have similar drinking behaviors are associated with increased alcohol use among adolescents [32, 33]. Interestingly, boys appear to be more vulnerable to peer influence on initiation of binge drinking than girls, which is consistent with the results in a previous study with adolescents of diverse ethnic backgrounds including Hispanics [52]. In contrast, results indicate that having close social ties with family was protective against binge drinking, which is consistent with protective role of family closeness and parental monitoring in preventing binge drinking among Mexican American youth [53]. Overall, the present results suggest that peer and family network may play critical roles in triggering or discouraging binge drinking among Mexican heritage adolescents.

The present study has several limitations. First, this study assessed binge drinking only at the two follow-up surveys, which limited our ability to analyze different trajectories in alcohol use over longer periods of time. Also, the peer network characteristics were asked only at the second follow-up, which prevents us from examining the effects of dynamic changes in peer network of adolescents on binge drinking. Second, we focus on initiation of binge drinking rather than on variation in patterns of binge drinking (never binge, decreased, increased, and persistent) due to the lack of power with the present sample size. The alternative approach of classifying binge drinking into never drinking, moderate drinking, and binge drinking was not selected because adolescent alcohol drinking may not necessarily follow sequential developments from never, moderate to binge drinking; an adolescent may binge on his or her first time drinking alcohol. Third, Gene × Environment interactions could not be tested because the measures of the social environment in this study were perceived social interactions with others that might be endogenous with individuals' genetic and psychological predispositions, and thus violating necessary independence assumptions of environment and genes for examining Gene × Environment interaction [36]. Our results with genetic variants for binge drinking should be interpreted with caution because they were not confirmed with a replication sample. This is often difficult to do because Hispanic populations are under-represented in health research; despite this limitation, this study serves to fill this gap, as it is one of a few studies on this topic using a Hispanic adolescent sample. Finally, the assessments in this study are subject to respondent biases as they are based on self-report. Previous studies have shown reliability of those self-reported measures, for example, in assessing adolescent substance use [54]. Future studies with a large sample size of Mexican heritage adolescents and more extensive longitudinal assessments of changes in personal network characteristics and alcohol drinking of adolescents would be beneficial to advance findings of the current study.

In conclusion, this study found that three genetic variants in TPH2 (rs1178999, rs17110451, and rs763717), traits of sensation seeking and impulsivity, and peer drinking and peer encouragement to drink, and family closeness are uniquely associated with risk for initiation of binge drinking in Mexican heritage adolescents. The results confirm previous findings and advance our understanding of how genetic, psychological, and social factors can jointly affect adolescent binge drinking within the contexts of Mexican American families. Results may inform how to improve alcohol prevention programs by tailoring them to the unique combination of these multidimensional risk and protective factors for binge drinking on health and adaptation of Hispanic adolescents in the USA.

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Compliance with Ethical Standards

Authors' Statement of Conflict of Interest and Adherence to Ethical Standards: All authors of the manuscript state that they have no conflict of interest. The research described in the manuscript conforms to APA ethical standards on the treatment of human subjects.

Authors' Contribution: S.Song devised the research hypotheses, analyzed the data, and took the lead in writing the manuscript. C.S.M. helped with data analysis and interpretation of the results. A.V.W. and L.M.K. designed and directed the project; S.Shete supervised the overall genetic data analysis and interpretation of genetic results. All authors provided critical feedback and helped shape the research, analysis and manuscript.

Ethical Approval: All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed Consent: Informed consent was obtained from all individual participants included in the study.

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