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Childhood trauma and two stages of alcohol use in African American and European American women: findings from a female twin sample

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Abstract

Aims—The current investigation assessed for moderating effects of childhood trauma on genetic and environmental contributions to timing of alcohol use initiation and alcohol use disorder in African American (AA) and European American (EA) women.

Methods—Data were drawn from diagnostic telephone interviews conducted with 3,786 participants (14.6% AA) in a longitudinal female twin study. Childhood trauma was defined alternately as child maltreatment and more broadly to include other events (e.g., witnessing violence). Phenotypic associations between childhood trauma and alcohol outcomes were estimated using logistic regression analyses. Twin modeling was conducted to test for moderating effects of childhood trauma on the contributions of genetic and environmental factors to timing of initiation and alcohol use disorder.

Results—Under both definitions, childhood trauma was associated with early initiation (relative risk ratios: 1.90, 1.72) and alcohol use disorder (odds ratios: 1.92, 1.76). Yet gene by environment effects were observed only for child maltreatment and timing of initiation in EA women, with heritable influences less prominent in those who had experienced child maltreatment (0.35, 95% CI:0.05–0.66 vs. 0.52, 95% CI:0.30–0.73).

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Compliance with Ethical Standards

Disclosure of potential conflicts of interest

The authors declare that they have no conflict of interest.

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.

Conclusions—We found more similarities than differences in the association of childhood trauma with alcohol outcomes across racial/ethnic groups, trauma type, and stages of alcohol use. However, findings suggest that the relative contribution of genetic factors to alcohol outcomes differs by childhood maltreatment history in EA women specifically in the earliest stage of alcohol use.

Keywords

alcohol; childhood trauma; African Americans; genetics; women

Distinctions between African Americans (AAs) and European Americans (EAs) in rates of alcohol use (Dauber, Hogue, Paulson, & Leiferman, 2009; Jackson, Sher, Cooper, & Wood, 2002; Johnston, O'Malley, Miech, Bachman, & Schulenberg, 2015; Wallace et al., 2003), timing of alcohol use initiation (Jackson, 2010; Malone, Northrup, Masyn, Lamis, & Lamont, 2012; Sartor et al., 2016), and prevalence of alcohol use disorder (AUD) (Alvanzo et al., 2011; Sartor et al., 2016; Wu, Woody, Yang, Pan, & Blazer, 2011) have been documented in numerous studies, with AAs consistently evidencing lower overall rates of alcohol use as well as early and problem drinking than their EA counterparts. However, over 20% of AAs meet criteria for AUD over the lifetime (Gibbs et al., 2013) and some studies indicate that AAs develop AUD at lower levels of alcohol consumption than EAs (Zapolski, Pedersen, McCarthy, & Smith, 2014). Differing levels of exposure to some of the well-known risk factors for alcohol related problems, including childhood trauma - which is far more prevalent in AAs than EAs (Breslau et al., 1998; Lee et al., 2012; Wildeman et al., 2014) - suggest that the pathways to AUD may differ by race/ethnicity, but this possibility has rarely been explored.

Adverse childhood experiences, which include events such as witnessing violence as well as child maltreatment, are associated with elevated risk for early initiation of alcohol use (Dube et al., 2006) and AUD in adulthood (Anda et al., 2002; Enoch, 2011). Childhood physical abuse, sexual abuse, and neglect, with estimated prevalence rates worldwide of 16–22% (World Health Organization, 2016), are robust predictors of a range of alcohol related outcomes, including heavy episodic drinking across adolescence (Shin, Edwards, & Heeren, 2009) and young adulthood (Shin, Miller, & Teicher, 2013) into middle adulthood (Widom, White, Czaja, & Mamorstein, 2007), in addition to AUD (Fergusson, McLeod, & Horwood, 2013; Nelson et al., 2006). The elevated rate of childhood trauma exposure but lower prevalence of problem alcohol use in AAs vs. EAs raises the possibility that AAs are more resilient to the development of early and problem drinking under conditions of trauma exposure, perhaps due to the greater presence of certain protective factors in AA vs. EA families or communities (e.g., involvement in church related activities). Despite its relevance to the refinement of etiological models of AUD in AAs, who have been historically underrepresented in alcohol research, we are unaware of any investigations to date that have directly addressed this question.

Modifying Effects of Childhood Trauma on Genetic Liability to Alcohol Outcomes

The substantial contribution of genetic factors to the development of AUD (50–60%) is well established (Goldman, Orosz, & Ducci, 2005) and although heritability estimates for initiation of alcohol use tend to be lower and vary to a greater degree (ranging from 11–78% (McGue, Iacono, Legrand, Malone, & Elkins, 2001; Rhee et al., 2003; Rose, Dick, Viken, Pulkkinen, & Kaprio, 2001; Sartor et al., 2009), twin studies have clearly supported a role for genetic influences on initiation as well. A range of environmental modifiers of (inferred) genetic liability to early and problem drinking have been investigated using twin samples, including religious upbringing (e.g., Koopmans, Slutske, van Baal, & Boomsma, 1999), parental monitoring (e.g., Dick et al., 2007), and urban vs. rural residency (e.g., Legrand, Keyes, McGue, Iacono, & Krueger, 2008; Rose, Dick, Viken, & Kaprio, 2001), which collectively demonstrate the relevance of environmental conditions to the manifestation of genetic liability to alcohol related outcomes (Young-Wolff, Enoch, & Prescott, 2011). Although not examined in twin-based gene by environment (GxE) studies, childhood adversity has been shown to modify genetic risk for alcohol outcomes in numerous studies using measured genes, primarily those associated with stress response and serotonin regulation (e.g., Kaufman et al., 2007; Laucht et al., 2009; Nelson et al., 2010). However, the degree to which the interplay of childhood trauma with genetic liability contributes to alcohol outcomes in AAs remains largely unknown, as AAs have rarely been included in these investigations. Among the few to do so were a study of childhood trauma and variation in GABRA2 on substance dependence exclusively in AA men (Enoch et al., 2010) and a study of AUD symptoms and maximum drinks consumed in a 24-hour period in AA and EA women and men (Sartor, Wang, Xu, Kranzler, & Gelernter, 2014), neither of which found evidence for GxE effects in AAs. Moderating effects of adverse childhood experiences on genetic liability to early alcohol use initiation – which may be stronger, given the closer proximity of initiation than AUD to the environmental exposure - have yet to be explored in genetically informative studies that include AAs.

Study Aims

The current investigation drew data from a large-scale female twin study with a substantial number of AA twins to identify potential distinctions between AA and EA women in the association of childhood trauma - defined alternately as child maltreatment and more broadly to include other types of events (e.g., involvement in a life-threatening accident) - with early alcohol use initiation and AUD. This included testing for moderating effects of childhood trauma on latent genetic liability (i.e., heritability) to these two outcomes. We focused on young women, as women have received less attention than men in the alcohol literature, despite the closing gender gap in alcohol use and problem drinking (Keyes, Grant, & Hasin, 2008), with resulting increases in alcohol related harms specific to women, such as pregnancy complications. More specifically, gender differences such as later age at first drink (York et al., 2004) and elevated rates of some forms of childhood trauma, namely, sexual abuse (Dube et al., 2003), in women vs. men highlight the need to further develop female-specific models of alcohol use. The study was designed to address four aims: (1) to

estimate the magnitude of association between childhood trauma and the two alcohol outcomes in AA vs. EA women; (2) to assess for differences by racial/ethnic group in GxE effects; (3) to gauge the persistence of GxE effects across stages of alcohol use; and (4) to evaluate whether liability to early initiation and AUD associated with childhood trauma is specific to child maltreatment.

Methods

Sample

The Missouri Adolescent Female Twin Study (MOAFTS) is a longitudinal study of substance use and related psychopathology in adolescent girls and young women, conducted with same-sex female twins born in Missouri to Missouri-resident parents between July 1, 1975 and June 30, 1985. Twins were identified through birth records and recruited from 1995 to 1999. The sample was demographically representative of the Missouri population at the time the twins were born, with nearly 15% being African-American (AA) and the remainder European-American (EA). Cohorts of 13-, 15-, 17-, and 19-year-old twin pairs and their families were ascertained in the first 2 years, with new cohorts of 13-year-old twins and their families added in the subsequent 2 years. (Details of sample ascertainment can be found in Waldron, Bucholz, Lynskey, Madden, & Heath, 2013). Wave 1 interviews were conducted with 3,258 twins (median age=15 years). Subsamples of twins completed a brief 1-year follow-up (wave 2) or a 2-year retest interview (wave 3). Between 2002 and 2005, all twins from the target cohort (excluding those who had withdrawn from the study or whose parents asked that the family not be re-contacted) were contacted for wave 4 interviews. All twins were 18 years of age or older at the time of recruitment for wave 4 (median age=22), so parental consent was not needed, and as a result, a greater number of twins completed wave 4 than wave 1 interviews (N=3,787, 80% of those identified from birth records). Wave 5 interviews were conducted between 2005 and 2008 with 3,428 twins (median age=24).

As wave 4 interviews yielded the largest sample size and covered lifetime psychiatric and psychosocial information in young adulthood, we limited the sample to MOAFTS participants who completed wave 4 interviews (dropping one due to incomplete data). Thus, analyses were conducted with data from 3,786 participants: 1,038 monozygotic (MZ) twin pairs (127 AA, 911 EA), 734 dizygotic (DZ) twin pairs (128 AA, 606 EA) and 242 twins (43 AA, 199 EA) whose co-twins did not participate. Since over 95% of wave 4 participants also completed wave 1, 3, and/or 5 interviews, data from waves 1, 3, and 5 were integrated as well, e.g., in cases where first alcohol consumption was endorsed in the wave 1 interview, we used the age at onset reported at wave 1. (Wave 2 data were not used, as the interview did not cover all relevant domains.)

Procedures

The study was approved by the Washington University School of Medicine Human Research Protections Office. All twins 18 years of age or older gave informed consent prior to study participation. Parental consent and twin assent were obtained prior to participation when twins were under age 18. Data were collected via telephone interview using an adaptation of the Semi-Structured Assessment for the Genetics of Alcoholism (Bucholz et al., 1994;

Hesselbrock, Easton, Bucholz, Schuckit, & Hesselbrock, 1999), which assesses *Diagnostic and Statistical Manual of Mental Disorders (DSM)-IV* psychiatric disorders, history of substance use, and related psychosocial constructs.

Measures

Childhood trauma exposure—*Child maltreatment* was defined as childhood physical abuse, neglect, or sexual abuse. All three forms of maltreatment were queried in the traumatic events section of waves 1, 3, and 4 interviews. Additional sexual abuse items were included in the health problems and health habits section of waves 1, 3, and 4 interviews and the parental discipline and childhood experiences section of the wave 4 interview. *Childhood trauma - broad* was defined as child maltreatment or (1) witnessing someone being killed or badly injured, (2) being involved in a life-threatening accident, or (3) being threatened with a weapon, held captive, or kidnapped. Criteria were met if any qualifying item was reported at any wave of data collection as occurring before age 16. (See Table 1 for list of interview questions).

Timing of alcohol use initiation—Age at alcohol use initiation was defined as age at consumption of first full standard alcoholic drink (definition provided by the interviewer). As we were specifically interested in timing of initiation and onsets of alcohol use tend to developmentally cluster in a discontinuous manner, we created a categorical variable (15, 16–17 and 18) representing roughly equivalently sized groups, to reflect timing of alcohol initiation. The categorical measure was preferred over continuously distributed age as it avoids assumptions regarding linear/proportional relationships between age of onset and other variables and is consistent with the piecewise nature of alcohol initiation, which developmentally clusters in age categories (Wagner & Anthony, 2002).

Alcohol Use Disorder (AUD)—A DSM-5 AUD proxy was defined as endorsement of 2 or more of the 10 DSM-IV symptoms – 7 dependence and 3 abuse (legal problems are excluded from DSM-5) - within the same 12-month period (self-reported or based on ages of onset and recency of individual criteria). [The symptom craving was not assessed. However, the psychometric benefit of adding craving to AUD is unclear, as it has not been found to add significant discriminatory information, particularly within those with mild or moderate levels of AUD (Cherpitel et al., 2010).]

Data Analysis

Data sets were constructed in SAS v9 (SAS Institute, 2008). Phenotypic analyses were conducted using binary and multinomial logistic regression in STATA v11 (StataCorp, 2009), with a robust variance estimator to account for familial clustering. Phenotypic analyses and twin modeling were conducted separately for child maltreatment and broad childhood trauma variables. Twin models were fitted to raw ordinal data in Mx (Neale, 2004). Based on twin pair correlations, variance in and covariance between timing of alcohol use initiation and AUD were attributed to additive genetic (A), shared/familial environmental (C) and individual-specific environmental (E) sources (Neale & Cardon, 1992). Evidence for A is derived from the MZ correlation (r_{MZ}) being larger than the DZ (r_{DZ}) correlation, i.e., the more genetically similar pairs are more similar to each other on

that phenotype. Evidence for C arises from $r_{DZ} > \frac{1}{2} r_{MZ}$, i.e., DZ pairs are more similar than would be expected if twin pair resemblance were entirely attributed to genetic influences. Support for E is based on $r_{MZ} < 1$, which indicates that the behavior is not due solely to genetic and shared environmental influences. Moderating effects of childhood trauma were tested by estimating A, C, and E components separately for twin pairs that were (1) concordant for exposure and (2) concordant for the absence of exposure to childhood trauma, with the parameters for members of discordant twin pairs equated to those of the appropriate exposure group. Moderation is indicated by a significant decrement in fit when estimates across exposed and unexposed twins are constrained to be equal. That is, evidence for a gene by environment interaction arises when the heritability of the outcome varies across exposure groups.

All of our results are based on fitting models to raw data using full information maximum likelihood estimation. Assumptions of multivariate normality regarding the distribution of twin and co-twin data (Neale & Maes, 2004) ($p > 0.05$) were met in AAs and EAs. Thresholds were estimated separately for childhood trauma exposed and unexposed groups (and for the exposed and unexposed members of the discordant pairs), and were adjusted for age at wave 4 (binary, age ≥ 22 ; median split to avoid any assumptions regarding a linear relationship between age and the timing of initiation and AUD). Analyses were conducted in EA and AA twins separately, as, even in the absence of differences by race/ethnicity in the phenotypic associations, the sources of variance (i.e., genetic, shared environmental, and individual-specific environmental factors) in alcohol outcomes for women with vs. without a history of childhood trauma may differ between AAs and EAs. Parameters across racial/ethnic groups were compared using multi-group post-hoc statistical tests that examined whether constraining parameters across groups resulted in a statistically significant change in model fit.

Results

Associations between Childhood Trauma and Alcohol Outcomes in AAs and EAs

Prevalence and timing of alcohol outcomes and childhood trauma are shown by race/ethnicity in Table 2. Rates of childhood trauma were higher in AA than EA participants for both broadly defined childhood trauma (41.5% vs. 23.5%; $\chi^2_{(1)} = 78.70$, $p < 0.001$) and child maltreatment (27.3% vs. 15.6%; $\chi^2_{(1)} = 44.25$, $p < 0.001$). Comparisons across racial/ethnic groups for each of the three types of maltreatment included in the study revealed higher rates in AAs vs. EAs for sexual abuse (20.4% vs. 11.7%; $\chi^2_{(1)} = 31.17$, $p < 0.001$), physical abuse (10.8% vs. 6.6%; $\chi^2_{(1)} = 12.45$, $p < 0.001$), and neglect (6.9% vs. 2.3%; $\chi^2_{(1)} = 35.81$, $p < 0.001$). AAs were less likely than EAs to be drinkers ($\chi^2_{(1)} = 44.61$, $p < 0.001$), to have started drinking at age 15 or younger ($\chi^2_{(1)} = 35.62$, $p < 0.001$), and to meet AUD criteria ($\chi^2_{(1)} = 40.77$, $p < 0.001$). They also reported a later age at onset of AUD ($t_{(1019)} = 3.20$, $p < 0.01$). Results of binomial and multinomial logistic regression analyses are shown for the full sample as well as separately by race/ethnicity in Table 3. Regression analyses conducted with the full sample revealed non-significant interactions between race/ethnicity and childhood trauma status, indicating that, across EA and AA twins, both child maltreatment

and broad childhood trauma were associated with increased likelihood of alcohol use initiation before age 16 as well as AUD.

Twin Pair Correlations

Twin pair correlations, calculated separately for AA and EA participants, are shown in Table 4. A substantial contribution of genetic factors and a modest contribution of shared environmental factors were indicated for both timing of alcohol use initiation and AUD. Hence, we proceeded with the full ACE model.

Twin Models

Results of model fitting are summarized here, with detailed model fitting steps reported in Supplementary Table 1, where final models are indicated with bold text. As seen in Table 5, evidence for moderation was observed only for child maltreatment on the timing of alcohol use initiation in EAs. Among EAs who had not experienced child maltreatment, genetic factors accounted for just over half of the variance in timing of alcohol use initiation ($A=0.52$, 95% CI: 0.31–0.73) while individual-specific environmental factors accounted for 27% (95% CI: 0.22–0.32). These estimates were similar to those observed in the full EA subsample (i.e., not stratified by child maltreatment status). By contrast, for the subset of EAs who experienced child maltreatment, 35% of the variance in timing of initiation was attributable to genetic factors (95% CI: 0.05–0.66) while individual-specific environmental influences explained nearly half ($E=0.46$, 95% CI: 0.31–0.63) of the variance. (Estimates of shared environmental influences did not differ significantly between EA women with vs. without a history of child maltreatment.) In all models other than the one in which we tested for moderating effects of child maltreatment on timing of alcohol use initiation, variance estimates could be equated for AAs and EAs across childhood trauma history status. In the child maltreatment moderation model for initiation, AAs with or without a history of maltreatment could be equated either with EAs with a history of maltreatment or with EAs with no history of child maltreatment, but we lacked the power to differentiate between the two.

Discussion

Our investigation of the association of childhood trauma with early alcohol use initiation and AUD in AA and EA female twins expands our understanding of this link in women in several ways. The inclusion of AAs, which is rare in genetically informative studies of alcohol phenotypes, allowed us to explore potential sources of distinctions between AA and EA women in this association, specifically, the seemingly paradoxical elevated prevalence of childhood trauma but lower levels of early and problem drinking in AAs. The examination of both timing of alcohol use initiation and AUD enabled us to assess the persistence of the link with childhood trauma, including GxE effects, across stages of alcohol use. The examination of both child maltreatment and more broadly defined childhood trauma further allowed us to assess, in a genetically informative design, whether the association of early life traumatic events with alcohol related outcomes in AA and EA women is specific to child maltreatment.

Age at Alcohol Use Initiation, AUD, and Childhood Trauma in AAs vs. EAs

The lower prevalence and later age at onset of alcohol use and AUD in AAs vs. EAs observed in our sample is consistent with the extant literature (Alvanzo, 2011; Johnston et al., 2015; Wallace et al., 2003; Wu et al., 2011). The higher prevalence in AA participants of both child maltreatment (27.3% vs 15.6%) and broadly defined childhood trauma (41.5% vs. 23.5%) is also consistent with prior research (Breslau et al., 1998; Lee et al., 2012; Wildeman et al., 2014). Despite these differences, neither the association of timing of initiation nor AUD with childhood trauma varied across the two racial/ethnic groups. Regardless of how it was defined, childhood trauma was associated with increased risk for early initiation of alcohol use and AUD. Again, our results parallel numerous studies documenting increased risk for early drinking and AUDs among individuals who have experienced child abuse or neglect (Fergusson et al., 2013; Nelson et al., 2006; Shin et al., 2009; Widom et al., 2007) and other adverse childhood events (Anda et al., 2002; Dube et al., 2006; Enoch, 2011) and suggest that these relationships are present in AAs as well as EAs. Thus, it appears that the paradox of higher rates of childhood trauma exposure but lower rates of early and problem drinking in AAs vs. EAs is not accounted for by differences in the presence – or the magnitude - of association between childhood trauma and alcohol outcomes.

Moderating Effects of Childhood Trauma on Genetic Contributions to Alcohol Outcomes

Consistent with prior twin studies, which are based primarily on all-male and mixed gender samples that do not include AA participants, we found evidence for heritable contributions to timing of initiation of alcohol use as well as AUD. A recent meta-analysis of these twin studies (Verhulst, Neale, & Kendler, 2015) also noted no evidence for heterogeneity in AUD heritability estimates by sex, which aligns with our estimate in an all-female cohort. Interestingly, the heritability of initiation was quite similar to that for AUD. The most likely explanation for this similarity is that genetic liability to timing of alcohol initiation is strongly correlated with genetic vulnerability to AUD, thus indexing common heritable variation (Sartor et al., 2009; Ystrom, Kendler, & Reichborn-Kjennerud, 2014).

Estimates of the relative contributions of genetic and environmental influences to timing of alcohol use initiation and AUD independent of childhood trauma status could be equated across racial/ethnic groups, yet tests for GxE effects of childhood trauma on the two alcohol outcomes produced evidence for moderation by child maltreatment only on initiation and exclusively in EAs. The heritability of timing of initiation was lower in EA women who had experienced child maltreatment than in those who had not, indicating that among EA women, child maltreatment is such a potent risk factor that the relevance of heritability is attenuated. Moderating effects were not observed in EAs for AUD under either definition of childhood trauma or for timing of alcohol use initiation by broadly defined childhood trauma. No GxE effects were observed for either outcome in AAs.

The absence of moderation effects with broadly defined childhood trauma is not surprising, as the literature in this area has primarily operationalized childhood trauma as child maltreatment and in the few studies that have incorporated additional indicators of childhood adversity, GxE effects emerged only for child maltreatment (Nilsson et al., 2011; Nilsson et

al., 2007). By contrast, the absence of GxE effects on AUD among EAs is somewhat surprising, as the majority of prior gene by childhood trauma studies have examined relatively severe alcohol phenotypes, i.e., heavy and problem drinking (Baranger et al., 2016; Lieberman et al., 2016; Nilsson et al., 2011) and alcohol dependence (Ducci et al., 2008, Nelson et al., 2010). However, the only known study to test for gene by childhood trauma effects on alcohol use initiation also found evidence that child maltreatment moderates risk for initiation (Kaufman et al., 2007), the stage of alcohol use most proximal to childhood traumatic experiences.

Although low power in the AA subsample does not allow us to interpret non-significant results of moderation tests as evidence for their absence, we tested for racial/ethnic differences, given that the current sample - the largest known twin study with psychiatric data to include AA twins - is among the few that could potentially reveal the presence of gene-environment effects. Thus, we view them as only suggestive, but find merit in considering them in the context of prior related work. The absence of moderation effects for childhood trauma on alcohol outcomes in AAs is consistent with the two known studies in this area to include AA participants. Enoch et al.'s (2010) case-control study examining interactive effects of GABRA2 variation and child maltreatment on alcohol, heroin, and cocaine dependence in African American men yielded non-significant GxE effects for alcohol dependence. An investigation of GxE effects of ADH1B variants and adverse childhood experiences on AUD symptoms and maximum drinks consumed in a 24-hour period in AA and EA women and men similarly did not support GxE effects in AAs (Sartor et al., 2014). However, there was evidence for GxE effects in EAs; EA men who carried the allele associated with reduced risk for alcohol related problems were at equal risk for AUD symptoms as those without the allele. That is, the protective effects appeared to be outweighed by risk conferred by adverse childhood events, much like our finding that the heritability of timing of alcohol use initiation was lower in EA women who had experienced child maltreatment. The apparent absence of such an effect in AA women, while far from accounting for the paradoxical elevated rate of childhood trauma exposure but lower rate of early alcohol use initiation in AAs compared to EAs, indicates that the links between child maltreatment, genetic liability to early alcohol use initiation, and timing of alcohol use initiation vary across the two racial/ethnic groups. Although power limitations due to the size of the AA subsample cannot be ruled out as a source of these distinctions, in light of corroborating evidence from the existing (albeit limited) literature, the specificity of our finding to EAs is suggestive of differential pathways from child maltreatment to early alcohol use in AA vs. EA women.

Limitations

Findings should be interpreted in light of certain limitations, in addition to those related to the size of the AA subsample. First, although the majority of the sample was ascertained in the early to peak years of alcohol use initiation and AUD onset, not all participants had passed through the age of risk for AUD and given the older mean age at onset of AUD in AAs, AAs may be disproportionally represented in this group. Second, given the absence of information on chronicity or severity of traumatic events, we could not determine whether the absence of GxE effects for broadly defined childhood trauma was due to the inclusion of

events less severe than child maltreatment, although the similarity of results across definitions in the phenotypic analyses does not suggest that. Third, interviews queried events that may have occurred several years prior to the time of interview and retrospective assessments are inherently vulnerable to reporting bias. Fourth, findings may not be generalizable to women from other geographic areas in the U.S. that differ substantially in sociodemographic characteristics. Fifth, the estimates of heritable and environmental influences on timing of alcohol use initiation differ somewhat from a prior report based on this sample (Sartor et al., 2013), likely due to differences in the categorization of age at initiation, with early defined as 14 vs. 15 in the current study. Sixth, we used a binary diagnostic measure for AUD. When categorical indices based on the DSM-5 severity definition of mild (2–3), moderate (4–5) and severe (6 or more) were stratified by zygosity, race/ethnicity and trauma, numbers in the severe category were low. Nonetheless, use of a binary measure might obscure associations observable at more severe levels of problem drinking. Future studies of larger cohorts should consider a categorical AUD variable. Seventh, we cannot infer causality in the link between childhood trauma and alcohol outcomes, in part because we opted to retain participants who experienced trauma after the onset of the alcohol outcome since eliminating this subgroup of trauma exposed girls would reduce generalizability of findings and potentially impact estimates of the contribution of familial influences, as childhood trauma frequently co-occurs with other family-level risk factors. Finally, in our statistical analyses, by estimating heritability conditional upon trauma exposure, we ignored the possible effects of gene-environment correlation, a simplification that appeared reasonable given evidence for only modest heritability of the trauma variables.

Conclusions and Future Directions

We found more similarities than differences across racial/ethnic groups in the association of childhood trauma with early alcohol use initiation and AUD and the relative contributions of heritable and environmental influences to those alcohol outcomes. The most noteworthy distinction between AA and EA women – evidence for gene by child maltreatment effects on timing of initiation exclusively in EAs – was only revealed when a robust environmental risk factor that varies in prevalence by race/ethnicity was integrated into the genetically informative design. The emergence of this finding highlights the need to simultaneously consider multiple risk and/or protective factors to identify sources of distinctions in drinking behaviors between AA and EA women, which is critical for refining etiological models of alcohol use and problem use in AA women. Efforts to extend this approach to other potential moderators of genetic liability and other substance use outcomes, which are currently being pursued by our group, will contribute to a more nuanced perspective on the intersection of race/ethnicity with genetic and environmental influences on the course of substance use. These pursuits can inform culturally tailored prevention efforts by revealing potential distinctions by race/ethnicity in the stages of drinking course where alcohol use is under the greatest degree of environmental control – and thus, most malleable – and identify the risk or protective factors to target at those stages.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

Items used to assess child maltreatment and broadly defined childhood trauma by sections of the interview

Child maltreatment	Type of maltreatment
Parental Discipline and Early Childhood Experiences: wave 4 only	
1. Before you turned 16, was there any forced sexual contact between you and any family member like a parent or step-parent, grandfather, etc.? By sexual contact I mean their touching your sexual parts, you touching their sexual parts or intercourse.	Sexual abuse
2. Before you turned 16, was there any forced sexual contact between you and anyone who was 5 or more years older than you (other than a family member)?	Sexual abuse
Health Problems and Health Habits (<age 16): waves 1, 3, and 4	
Has anyone ever forced you to have sexual intercourse?	Sexual abuse
Traumatic Events (<age 16): waves 1, 3, and 4	
<i>Did event # ever happen to you?</i> (definitions provided in respondent booklet, not read by interviewer)	
1. You were raped	Sexual abuse
2. You were sexually molested	Sexual abuse
3. You were physically abused as a child	Physical abuse
4. You were seriously physically attacked or assaulted	Physical abuse
5. You were seriously neglected as a child	Neglect
Additional traumatic events (included in broadly defined childhood trauma)	
6. You witnessed someone being killed or badly injured	
7. You were involved in a life-threatening accident	
8. You were threatened with a weapon, held captive, or kidnapped	

Table 2

Prevalence and timing of initiation of alcohol use, alcohol use disorder (AUD), and childhood trauma in African American and European American women

	African Americans (n=553)	European Americans (n=3,233)
Ever consumed full drink *	86.8%	94.5%
Mean age at initiation (SD) *	17.2 (3.1)	16.1 (2.6)
15	26.6%	41.0%
16–17	26.0%	32.6%
18	47.36%	26.4%
AUD among (ever) drinkers *	16.8%	31.1%
Mean age at AUD onset (SD) *	18.6 (3.2)	17.6 (2.5)
Childhood maltreatment *	27.3%	15.6%
Mean age at 1 st experience (SD)	8.3 (3.9)	8.0 (4.0)
Childhood trauma - broad *	41.5%	23.5%
Mean age at 1 st experience (SD)	9.4 (4.0)	8.9 (4.2)

child maltreatment = childhood physical or sexual abuse or neglect; childhood trauma broad=child maltreatment, witnessing someone being killed or badly injured, being involved in a life-threatening accident, or being threatened with a weapon, held captive, or kidnapped; SD=standard deviation;

*
 $p<.01$

Table 3

Associations of childhood trauma with alcohol outcomes in African American and European American women*

	Full sample (n=3,786)	African Americans (n=553)	European Americans (n=3,233)
Child maltreatment			
Initiation: RRR ^a (CI)			
15	1.90 (1.51, 2.40)	2.75 (1.67, 4.51)	2.04 (1.55, 2.69)
16–17	1.10 (0.86, 1.42)	1.25 (0.73, 2.13)	1.20 (0.89, 1.61)
Alcohol use disorder ^b : OR (CI)	1.92 (1.58, 2.34)	1.88 (1.07, 3.30)	2.14 (1.73, 2.65)
Childhood trauma			
Initiation: RRR ^a (CI)			
15	1.72 (1.40, 2.09)	2.53 (1.59, 4.04)	1.88 (1.49, 2.37)
16–17	1.19 (0.96, 1.47)	1.69 (1.07, 2.69)	1.26 (0.98, 1.61)
Alcohol use disorder: OR (CI)	1.76 (1.48, 2.08)	1.58 (0.94, 2.65)	2.00 (1.66, 2.40)

child maltreatment = childhood physical or sexual abuse or neglect; childhood trauma broad=child maltreatment, witnessing someone being killed or badly injured, being involved in a life-threatening accident, or being threatened with a weapon, held captive, or kidnapped. RRR=relative risk ratio; CI=95% confidence intervals; OR=odds ratio;

^a 18=comparison group;

^b among (ever) drinkers;

* no statistically significant differences between AA and EA women

Table 4

Twin pair correlations for timing of alcohol use initiation and alcohol use disorder in African American and European American women

	MZ twin <i>r</i> (95% CI)	DZ twin <i>r</i> (95% CI)
African Americans		
Timing of alcohol use initiation	0.57 (0.38–0.76)	0.29 (0.04–0.53)
Alcohol use disorder	0.56 (0.26–0.86)	0.36 (–0.03–0.74)
European Americans		
Timing of alcohol use initiation	0.72 (0.67–0.77)	0.44 (0.35–0.53)
Alcohol use disorder	0.59 (0.50–0.67)	0.42 (0.30–0.55)

MZ=monozygotic; DZ=dizygotic; CI=confidence intervals

Final moderation models of timing of alcohol use initiation and alcohol use disorder in African American (AA) and European American (EA) women, stratified by childhood trauma history: proportions of variance attributable to additive genetic (A), shared environmental (C), and individual-specific environmental (E) influences, with 95% confidence intervals

Timing of alcohol use initiation					
History of child maltreatment			No child maltreatment		
	A	C	E	A	C
AA	Variance can be equated with EAs either with or without a history of maltreatment				
EA	0.35 (0.05, 0.66)	0.19 (0.00, 0.52)	0.46 (0.31, 0.63)	0.52 (0.31, 0.73)	0.21 (0.02, 0.40)
	History of childhood trauma - broad			No childhood trauma - broad	
	A	C	E	A	C
AA	No moderation Variance equated with EAs			No moderation Variance equated with EAs	
EA	0.52 (0.33, 0.72)	0.17 (0.00, 0.34)	0.31 (0.26, 0.36)	No moderation Variance equated across childhood trauma status	
Alcohol Use Disorder					
History of child maltreatment			No child maltreatment		
	A	C	E	A	C
AA	No moderation Variance equated with EAs			No moderation Variance equated with EAs	
EA	0.30 (0.01, 0.60)	0.26 (0.01, 0.51)	0.44 (0.36, 0.53)	No moderation Variance equated across maltreatment status	
	History of childhood trauma - broad			No childhood trauma - broad	
	A	C	E	A	C
AA	No moderation Variance equated with EAs			No moderation Variance equated with EAs	
EA	0.34 (0.05, 0.63)	0.23 (0.00, 0.47)	0.43 (0.35, 0.53)	No moderation Variance equated across childhood trauma status	