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The association of specific traumatic experiences with alcohol initiation and transitions to problem use in European American and African American women

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Abstract

Background—The aims of this study are to a) characterize racial differences in alcohol involvement and b) examine the risk conferred by specific trauma exposures and PTSD for different stages of alcohol involvement in European American (EA) and African American (AA) women.

Methods—Data are from the Missouri Adolescent Female Twins Study [(N =3787); 14.6% AA; mean age at most recent interview = 24.5 (SD 2.8)]. Trauma exposures (e.g. sexual abuse (SA), physical abuse (PA), witnessing another person being killed or injured, experiencing an accident, and experiencing a disaster) were modeled as time-varying predictors of alcohol initiation, transition to first alcohol use disorder (AUD) symptom, and transition to AUD diagnosis using Cox proportional hazards regression while taking into account other substance involvement, parental characteristics, and commonly co-occurring psychiatric disorders.

Results—In EA women only, SA was associated with alcohol initiation prior to the age of 14, PA predicted transition from initiation to first AUD symptom, and PA, witnessing injury or death, and SA predicted transition to AUD diagnosis. No association was discovered between trauma exposures or PTSD for any stage of alcohol involvement in AA women.

Conclusions—Results reveal trauma experiences as important contributors to all stages of alcohol involvement in EA women only, with different trauma types conferring risk for each stage of alcohol involvement. PTSD was not revealed as a significant predictor of AUD in EA or AA women, suggesting trauma, independent of PTSD, directly contributes to alcohol involvement. Findings highlight the importance of considering racial differences when developing etiologic models of the association of traumatic experiences with alcohol involvement.

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Keywords

alcohol; alcohol use disorder; traumatic stress; racial differences

Introduction

Alcohol use disorder (AUD) is a major public health concern associated with a host of mental and physical health outcomes (Cargiulo, 2007, Grant et al., 2015, Rehm et al., 2009). Recent epidemiologic data show rates of past month problem alcohol use and alcohol use disorder (AUD) appear to be declining across all age groups including adolescent (12–18) and young adult (18–25) subpopulations [Center for Behavioral Health Statistics and Quality (CBHSQ), 2015, Johnston et al., 2016]. However, the rates of past month alcohol use (59.6%), heavy use (10.8%), and binge drinking (37.7%) in young adults are still of concern (CBHSQ, 2015). Furthermore, declining overall trends in alcohol involvement are not reflective of trends in alcohol use behaviors in women. In fact, women are closing the gender gap in AUD (Keyes et al., 2008) as young women are increasingly using alcohol and experiencing alcohol-related problems (Johnston et al., 2016). As recent research has highlighted AUD as more severe in women (Foster et al., 2015, Grant et al., 2015) and sex differences in the pathways to alcohol problems are evident (Kendler et al., 2015), further examination of AUD etiology in women is essential.

Alcohol involvement and the contribution of trauma exposure

Trauma exposure has consistently been reported as a risk factor for alcohol use and related problems (Kendler et al., 2000, Kilpatrick et al., 2000, Sartor et al., 2013b, Zinzow et al., 2009). Both assaultive traumas (i.e. sexual and physical assault) and non-assaultive exposures have been found to increase problem drinking behaviors (Cerda et al., 2011, Keyes et al., 2011, North et al., 2004). Overall, research has reported significantly elevated risk for early initiation of alcohol use and development of alcohol use disorder in trauma survivors (Danielson et al., 2009, Sartor et al., 2010) with mixed findings pertaining to the specificity and strength of the relationship between trauma and alcohol outcomes when accounting for PTSD. Some research suggests the relationship between trauma exposure and problem alcohol involvement is only indirect, mediated through PTSD (Epstein et al., 1998), while others report trauma exposure uniquely contributes to alcohol involvement above and beyond PTSD (Danielson et al., 2009, Kilpatrick et al., 2000, Sartor et al., 2010). Klilpatrick et al.'s (2000) investigation of substance abuse in adolescence reported childhood physical abuse, childhood sexual abuse, and witnessing violence all independently contributed to past year AUD while PTSD contributed no additional variance to the model. A longitudinal study of young adults by Danielson and colleagues (2009) revealed that sexual assault remained a significant predictor, along with PTSD, for past year alcohol problems. Sartor et al. (2010) reported both those with PTSD and those with any trauma exposure but no PTSD were at an increased risk for alcohol dependence even when accounting for commonly co-occurring psychiatric conditions. However, the existing epidemiologic literature linking traumatic experiences with alcohol involvement has only investigated a single stage of alcohol involvement (i.e. alcohol initiation, problem use, or AUD diagnosis) neglecting to consider

alcohol use and transitions to more pathological use concurrently, which may reveal drinking stage-specific contributions of trauma.

Role of race/ethnicity

Racial disparities in alcohol use, alcohol use disorder (AUD), and trauma exposure between European Americans (EA) and African Americans (AA) have been reported in female cohorts (Grant et al., 2012, Hawkins et al., 2010, Sedlak et al., 2010). Adolescent and young adult EA women experience higher rates of alcohol use and AUD diagnosis (Grant et al., 2012), but some research suggests AAs who develop AUD problems experience more severe symptoms (Mulia et al., 2009). In addition, EA women report lower rates of sexual abuse (SA), physical abuse (PA), experiencing a life threatening injury and witnessing injury or death (Duncan et al., 2014) than their AA counterparts. That said, studies have revealed significantly elevated risk for early initiation of alcohol use and development of alcohol use disorder following trauma in adolescent girls and women (Sartor et al., 2010, Sartor et al., 2013b). However, despite their lower rates of trauma exposure, problem drinking is more common in EA than AA women (Grucza et al., 2008, Sartor et al., 2013b). This seemingly paradoxical relationship between trauma exposure and alcohol outcomes in EA and AA women suggests existing environmental etiological models of AUD development do not fit as well for AA as EAs. Previous research has identified racial variations in risk and protective factors for alcohol involvement including: exposure to household violence (Bossarte and Swahn, 2008), neighborhood context (Tobler et al., 2011), and alcohol expectancies (Chartier et al., 2009). Furthermore, African American women have been underrepresented in alcohol research (Zemore et al., 2013), and racial differences in specific types of trauma-related risk factors for alcohol use may have gone undetected.

Current Study

The current study builds on previous investigations (Sartor et al., 2010; Sartor et al 2013b) by examining the unique contributions of specific trauma exposure types and posttraumatic stress disorder (PTSD) to increasingly severe levels of alcohol involvement in EA and AA women. In addition, this study will examine racial differences in timing and prevalence of multiple stages of alcohol involvement – alcohol initiation, transition to first AUD problem, and transition to AUD diagnosis. As such the aims of the current study are to a) characterize racial differences in alcohol involvement and b) examine the risk conferred by specific trauma exposures and PTSD for different stages of alcohol involvement – alcohol use, transition to first AUD symptom, and transition to AUD diagnosis – in EA and AA women, while also considering the contribution or other substance involvement and psychiatric factors.

Materials and Methods

The Missouri Adolescent Female Twin Study (MOAFTS) procedures were approved by the Human Research Protections Office at Washington University and have been described in detail previously (Heath et al., 2002, Waldron et al., 2013) with methods as they pertain to the current investigation provided below.

Participants

The sample was composed of 3,787 women who completed Wave 4 (conducted from 2002 to 2005) of the Missouri Adolescent Female Twin Study – a longitudinal investigation of alcohol-related problems and associated psychopathology in female adolescents and young adults. Participants were identified through birth records born between July 1, 1975 and June 30, 1985 to Missouri-resident parents. Cohorts of 13, 15, 17, and 19 year-old twin pairs and their families were recruited from 1995 to 1999. Of those found eligible for participation, parental diagnostic interviews were collected at baseline in 78% of the families recruited. The Wave 4 assessment was chosen for the current investigation as it included the largest number of respondents and most intensive trauma assessment. Data from Waves 1, 3 and/or 5 were available for 95.5% of Wave 4 participants. Participant ages ranged from 18 to 32 years (M = 24.5; SD = 2.8) at the time of the most recent interview; 14.6% percent of participants identified as African-American (AA).

Procedure and assessment battery

Trained interviewers obtained verbal consent prior to the start of the interview and completed the assessment – a modified for telephone administration version of the Semi-Structured Assessment for the Genetics of Alcoholism [SSAGA; (Bucholz et al., 1994)]. The SSAGA assessed the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition [DSM-IV;* (American Psychiatric, 1994)] substance use and other psychiatric disorders and related psychosocial domains. All data from waves 1, 3, 4, and 5 available were utilized and collapsed across waves for analytic purposes. In cases where age at first trauma exposure, age at onset of each stage of alcohol involvement, or age of other individual level covariates was reported in more than one interview, we used the age at first occurrence reported the first time the participant endorsed it.

Alcohol related behaviors—*Alcohol initiation* was defined as the first time the participant ever used alcohol with age of onset recorded as the age this first occurred. For those who endorsed ever using alcohol at more than one assessment, Cronbach alpha for age of first alcohol initiation was 0.78, indicating acceptable test-retest reliability across waves 1, 3, and 4. We chose the lowest third of the age at initiation distribution (age 15 or younger) to represent early use. *First AUD symptom* was coded positive for individuals who endorsed one or more symptoms of alcohol abuse or dependence with age at first AUD symptom derived from reported age(s) that each endorsed symptom was first experienced. A *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* [*DSM*-5;(Association, 2013)] *AUD proxy* was calculated by identifying individuals who endorsed two or more of the 10 symptoms – 7 dependence and 3 abuse (legal problems are excluded from *DSM*-5) within the same 12-month period at some point during their life [The symptom craving was not assessed; however, the psychometric benefit of adding craving to AUD is unclear (Hasin et al., 2013), as the addition of craving to the *DSM-IV* alcohol dependence has not been found to add significant information (Cherpitel et al., 2010, Hasin et al., 2012).]. Age of onset was defined as the first age at which *DSM-5* AUD proxy was met.

Trauma exposure and PTSD—*Trauma exposure* history was assessed using a checklist of traumatic events adapted from the Revised Diagnostic Interview Schedule (Robins et al.,

1985) used in the National Comorbidity Survey (Kessler et al., 1995). Prior to the interview, respondent booklets that included a list of traumatic events were mailed and participants were asked whether they had experienced each of the traumatic events listed. If a trauma was endorsed, the age at first experience of each was queried. Traumatic experiences included SA, PA, witnessing injury or death, life threatening accident, and natural disaster. Childhood SA and Childhood PA were queried with additional behavioral descriptions of experiences. Endorsement of one or more questions was coded as a positive for SA or PA respectively (see Supplemental Table 1). Ages of onset were defined as age of first reported experience for each event.

Posttraumatic Stress Disorder diagnosis reflects *DSM-IV* criteria and was assessed querying PTSD symptom clusters pertaining to the traumatic event reported as most disturbing which also resulted in feelings of "intense fear, helplessness, or horror." Individuals who reported no trauma or no disturbing feelings were coded as negative for PTSD. Among those who met full PTSD criteria, age of PTSD onset was defined as age at first symptom that lasted at least 1 month.

Covariates—Both other substance use (Guxens et al., 2007) and psychopathology (Pardini et al., 2007) have been associated with alcohol involvement; therefore, cannabis use, tobacco use, conduct disorder (CD), and major depressive disorder (MDD) were considered as covariates. *Tobacco initiation* (age of onset) was defined as the first age at which the participant used/tried smoking tobacco. *Cannabis initiation* (age of onset) was defined as the first age at which the participant used/tried cannabis. *Conduct disorder* was defined as meeting three or more conduct disorder symptoms in a 12-month period prior to the age of 15. *Major Depressive Disorder* criteria was met if 5 or more major depressive symptoms during the same 2-week period (along with dysphoria or anhedonia) were reported as occurring for more than 2 weeks with age of onset defined as age when first met full criteria. *Parental characteristics:* To consider socio-economic status, mother's reports of *familial household income* (defined as < $30,000, $30,000 – $74,999, and ≥ $75,000) and *parental education* were both included in models as a proxy. Variables representing mother's and father's education levels were created representing whether each parent had completed high school (with more than high school as the reference variable). *Maternal and paternal alcohol use problems* were also independently assessed using the SSAGA, by querying the twins: (a) Has drinking ever caused your biological mother/father to have problems with health, family, job or police, or other problems, and (b) Have you ever felt that your biological mother/father was an excessive drinker? Additionally, the mother's report on her own and that of the co-parent's alcohol use disorder symptoms was used to assess parental problem alcohol use. Endorsement of a) one or more alcohol problems by maternal report or b) either alcohol question by either twin was coded as a positive response for alcohol-related misuse in that parent.

Data analysis

Analyses were stratified by race, as pooled analyses, with or without interaction terms, are statistically compromised given the substantial imbalance in socioeconomic status between

AAs and EAs in our sample which could impact the cogency of risk scores (see Imbens and Rubin, 2015).

Alcohol involvement, psychopathology, and trauma exposure by race—Logistic regression models adjusting for participant's most recent age, familial household income, mother's and father's education, and mother's and father's lifetime history of alcohol-related problems were conducted to test for differences in rates of alcohol and other substance use, endorsement of one or more AUD symptoms, proxy DSM-5 AUD diagnosis, psychopathology, and trauma exposure by race/ethnicity. Independent t-tests were conducted to test for differences in age at first use, age at onset of first AUD symptom, age at onset of AUD diagnosis, and rates of transition from first use to first symptom and AUD diagnosis by race/ethnicity. All analyses were conducted taking familial clustering into account using the cluster command as implemented in Stata (StataCorp, 2007).

Association of alcohol initiation and transition to AUD with trauma exposure —Cox proportional hazards (PH) regression analyses were conducted to examine associations with a) initiation of alcohol use, b) transition from alcohol initiation to onset of AUD symptoms, and c) transition from alcohol initiation to onset of AUD diagnosis. Cox PH regression analyses allow for the possibility that participants who have not yet experienced a particular outcome may do so in the future. Age at first alcohol use was used as the entry point in the AUD symptom onset and AUD diagnosis analyses. As such, data up until the time of censoring (most recent interview) is used in the calculation of hazard ratios. In addition, the PH assumption that risk remains constant over time was tested with the Grambsch and Therneau test of the Schoenfeld residuals (Grambsch and Therneau, 1994). Trauma exposures, PTSD, MDD, tobacco and cannabis use were modeled as time-varying covariates coded as negative in each year prior to the age at first occurrence and positive for that year and each subsequent year, so that only behaviors that occurred prior to or at the same time as the outcomes counted towards the transition. Analyses were conducted in Stata (StataCorp, 2007) using Huber-White robust standard errors to adjust for the non-independence of observations in twins. Violations of the PH assumption were resolved by estimating piecewise hazard ratios (HRs) for each age range of ≤14, 15–16, and ≥17. AUD symptom onset and AUD diagnosis analyses also included early alcohol use status as a dummy variable as a covariate.

Results

Alcohol involvement, psychopathology, and trauma exposure by race

Adjusting for familial level risk factors, AAs were less likely to use alcohol (77.9% vs. 87.9%, $p < .001$) and started drinking at a later age [AA: $M = 16.8$ years; EA: $M = 15.9$ years; $p < .001$] than their EA counterparts (Table 1). Of those who ever used alcohol, AA women were less likely to experience an AUD symptom (49.0% vs. 68.2%, $p < .001$) and meet DSM-5 criteria for lifetime AUD (24.7% vs. 39.5%, $p < .001$). In the total sample, lifetime prevalence of AUD was 33.2%, with lower rates of AUD in AA (20.7%) compared to EA [35.3%, (AOR=0.44 95%CI: 0.34–0.57)] young adult women. AAs were more likely to be diagnosed with PTSD [total sample: 7.2% vs. 3.6%, (AOR=1.55, 95%CI: 1.02–2.37)];

however, no significant racial differences were found when PTSD was conditioned on trauma exposure and odd ratios were adjusted for familial risk factors [10.1% vs 6.9%, (AOR=1.27, 95%CI: 0.84–1.93)]. AAs also reported higher prevalence of PA (42.5% vs. 18.0%, $p < .001$), witnessing injury or death (26.6% vs. 15.0%, $p < .001$), and experiencing a life threatening accident (21.9% vs. 14.3%, $p < .01$). Although higher prevalence rates were reported in AAs, no significant differences were found in SA exposure (21.2% vs. 14.8%, $p = 0.21$) or experiencing a natural disaster (16.6% vs. 17.7%, $p = 0.15$) between AA and EA women when taking familial risk factors into account.

Cox regression for alcohol related outcomes

Alcohol Initiation—Survival analysis of alcohol initiation revealed that only SA increased risk for alcohol initiation prior to the age of 14 in EAs [HR=1.43 95%CI: (1.08–1.87)] with no association of SA with alcohol initiation in AA women and no further significant increase in hazards of other types of trauma exposure in EA or AA women (Table 2). When considering the contribution of other substances, in EA women, the largest magnitude risk for alcohol initiation was conferred between the ages of 14 and16 by tobacco use [HR= 2.97 95%CI: (2.64–3.33)] and cannabis use [HR= 1.88, 95%CI: (1.70–2.09)] prior to the age of 16. For AA women, tobacco use increased the hazard for alcohol initiation with the highest magnitude prior to the age of 17 [HR= 2.30, 95%CI: (1.67–3.17)] with the increased hazard conferred by cannabis use [HR= 2.26, 95%CI: (1.76–2.90)] consistent across all risk periods. Posttraumatic stress disorder did not significantly increase the hazard for alcohol initiation in EA or AA women.

Alcohol Initiation to first AUD symptom—The only trauma-related significant hazard associated with transition from first use to first symptom was PA exposure and it was specific to EA women [HR= 1.16, 95%CI: (1.04–1.31)] (Table 3). Consistent with alcohol initiation findings, the largest magnitude risk was conferred by tobacco use [HR= 4.53 95%CI: (1.98–10.35)] for an AUD problem prior to the age of 14 and cannabis use [HR= 1.64, 95%CI: (1.48–1.81)] in EA women. For AA women, cannabis use increased the hazard of transition to an AUD symptom [HR= 2.18, 95%CI: (1.63–2.94)]. Posttraumatic stress disorder did not significantly increase the hazard for alcohol problems in EA or AA women.

Alcohol Initiation to AUD onset—Significant trauma predictors of the transition from first use to AUD were observed in EA women only (Table 4). Sexual abuse [HR= 1.21, 95%CI: (1.01–1.44)], PA [HR= 1.30, 95%CI: (1.12–1.50)], and witnessing injury or death [HR= 2.14, 95%CI: (1.27–3.59)] predicted transition to AUD. In addition, tobacco use [HR= 1.53, 95%CI: (1.26–1.86)] and cannabis use [HR= 1.91, 95%CI: (1.67–2.19)] increased the hazard of transitioning from alcohol initiation to AUD diagnosis in EA women. Only cannabis use increased the hazard of transition to AUD in AA women [HR= 2.81, 95%CI: (1.74–4.54)]. PTSD remained non-significant in both EA and AA women.

Discussion

We investigated the association between specific trauma exposures and PTSD for multiple stages of alcohol involvement – alcohol initiation, transition from first use to first AUD

symptom and transition from first use to AUD diagnosis – in female European American and African American young adults. Consistent with previous research (Grant et al., 2012), EAs were more likely to use alcohol, experience an AUD symptom, develop AUD and reach these milestones at an earlier age, on average, than their AA counterparts. Trauma variables were revealed as risk factors for each stage of alcohol involvement but only in EA women. Specifically, only SA predicted alcohol initiation, PA predicted transition to first AUD symptom, and SA, PA, and witnessing injury or death increased the risk for transitioning to AUD. Our findings build on previous research (Keyes et al., 2011, Sartor et al., 2010) by examining the effects of specific trauma and considering the transitions to first AUD problem and AUD diagnosis from alcohol initiation. By considering transitions from initiation to later stages, we were able to parse out the effects of trauma exposures on alcohol initiation from those on progression to an AUD symptom and AUD diagnosis, given exposure to alcohol use. The current findings suggest, in general, trauma exposure impacts initiation of alcohol use and transition to an AUD symptom and remains a significant risk factor for transition from initiation to AUD in EA women. PTSD did not significantly increase hazard of any alcohol outcome suggesting a direct relationship between trauma exposure and alcohol related behaviors. Our findings lend some support to the self-medication hypothesis – SA increased risk for alcohol initiation and AUD diagnosis and PA increased risk for onset of AUD symptoms and AUD diagnosis – but findings also link more passive trauma exposure (i.e. witnessing injury or death) with transition to AUD diagnosis in EA women. The majority of those who endorsed “witnessing another being killed or injury” (62.4%) first experience this type of trauma after they had already initiated drinking. Therefore, as our analyses relied on time-varying data, the risk conferred by witnessing injury or death for these individuals only contributed to later stages of alcohol involvement. PTSD was not revealed as a risk factor; however, we only considered PTSD diagnosis and not sub-threshold levels of posttraumatic distress.

This study partially addresses the paradox of higher trauma exposure but less alcohol involvement in AAs as findings suggest trauma is not as strongly related to alcohol outcomes in AAs compared to EAs. Differential reporting of trauma across races could partially account for discrepancies as, although AAs report higher rates of PA, racial differences in the reporting of trauma exposure have been identified. That is, AA women are more likely to report childhood PA on behavioral items without also explicitly endorsing the abuse on the trauma interview, which may point to differences in acculturation of physical punishment between EA and AA women (Werner et al., 2016a). Furthermore, racial differences in risk and protective factors (which were not assessed here) may mediate the relationship between trauma and alcohol outcomes, but have remained relatively unexplored. Additionally, we need to increase our understanding of what drives early and problem drinking behaviors in AAs as, although these behaviors are less common, they are not absent. Given the research suggesting that AAs who do use alcohol experience more severe AUD symptoms (Mulia et al., 2009) than their EA counterparts, understanding AUD etiology in this population remains an important step in the development of effective prevention efforts. We did not foresee the lack of association between trauma exposure and PTSD with alcohol outcomes in AA women. This lack of relationship could be due to limited AA sample size, but may also be explained by different patterns of choices of

substances. AAs are more likely to use cannabis prior to alcohol (Sartor et al., 2013a), and recent findings in the same sample cohort did find trauma exposure increased the risk of cannabis initiation (but not the transition to cannabis problems) in AA women (Werner et al., 2016b). To address the possibility that cannabis use mediates the relationship between traumatic exposure and alcohol involvement, we re-ran all three models for AAs removing cannabis use from the model. Although hazard ratios associated with traumatic exposure slightly increased, the results did not reach statistical significance. Future research would benefit from including a larger sample of AA women, which allow for the development of well powered, racially distinct etiological models of alcohol involvement.

Racial differences were also apparent in the association of tobacco and cannabis use with alcohol related outcomes. Tobacco use and cannabis use both increased the risk of alcohol initiation in EA and AA women; however, the relationship was clearly associated with early initiation in EA compared to AA women. In AA women, tobacco and cannabis use conferred a consistent, significant risk for alcohol initiation, whereas the association between tobacco and cannabis use and alcohol initiation in EA women varied by risk period. Cannabis use continued to be a significant predictor of transitions to an AUD symptom and to AUD in both EA and AA women, while tobacco use conferred statistically significant risk for problem alcohol involvement in EA women only, with only a trend in AA women.

Limitations and future directions

Limitations to the current study include the inability to take into account severity and chronicity of trauma exposure. Additionally, the current study did not investigate if heavier alcohol use increased the risk of experiencing a trauma. That is, heavy alcohol use and problem involvement may increase the risk for additional trauma exposure by increasing the likelihood of engaging in high-risk behaviors (Haller and Chassin, 2014, Baskin-Sommers and Sommers, 2006). Third, there is epidemiological evidence that AUD occurs later in AA women (Grant et al., 2012). Therefore, further follow-up with the AA participants may clarify the relationship between trauma exposure and AUD in AA women, as in our sample, it is likely that not all the AA women have passed through the age of risk. Additional methodological limitations include the potential for recall bias of ages of onset for both substance and trauma related measures as well use of a Midwestern cohort that might not be generalizable to national and global populations. The current findings suggest etiological models of alcohol involvement for AA and EA women must be considered discretely as risk and protective factors may differ across race – although protective factors not considered in these analyses. Lastly, the current findings are based on a female-only sample and cannot be generalized to men. It is likely that men differ from women with respect to the types of trauma experienced, reactions to trauma (Breslau, 2002, Tolin and Foa, 2006) and involvement with alcohol (Johnston et al., 2016).

Conclusion

The current investigation extends previous research by identifying unique trauma-related and substance use risk factors of alcohol involvement for EA women. Our results highlight trauma exposure as a risk factor for alcohol initiation, transition to an AUD symptom, and AUD diagnosis but only in EA women. Future research should include a larger cohort of

AAs and more specific trauma-related measures in a longitudinal design that can examine bi-directional influences of trauma exposure and other heavy consumption measures as an intermediate step to provide further insight into this relationship.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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Table 1

Alcohol Involvement and Trauma Exposure by Race

	African American	European American	
	n **= 554**	***n*** **= 3233**	
	%/M (SD)	***%/M (SD)***	***AOR (95% CI)***
Alcohol Related Behaviors			
Ever used Alcohol	77.9%	87.9%	0.45 (0.34–0.60) ***
Age at Alcohol Initiation (years)	16.8 (3.2)	15.9 (2.5) ***	
≥1 Alcohol Related Problem †	49.0%	68.2%	0.44 (0.35–0.55) ***
Age at Alcohol Initiation (years) ‡	16.5 (3.5)	15.6 (2.6) ***	
Age at 1st Alcohol Problem (years) ‡	19.6 (3.7)	18.0 (2.9) ***	
Alcohol Use Disorder (AUD) †	24.7%	39.5%	0.47 (0.36–0.62) ***
Age at Alcohol Initiation (years) ¥	16.0 (3.2)	15.2 (2.4) *	
Age at 1st Alcohol Problem (years) ¥	18.7 (3.4)	17.2 (2.7) ***	
Age at AUD diagnosis (years) ¥	20.0 (3.8)	18.5 (3.1) ***	
Trauma Exposure			
Sexual Abuse	21.2%	14.8%	1.20 (0.90–1.58)
Sexual Abuse Age of Onset (years)	9.5 (4.7)	11.1 (5.5)	
Physical Abuse	47.2%	21.7%	2.67 (2.13–3.35) ***
Physical Abuse Age of Onset (years)	10.2 (3.4)	10.7 (4.3)	
Witnessed another being killed or injured	26.6%	15.0%	1.86 (1.46–2.37) ***
Witnessing Age of Onset (years)	14.3 (4.9)	15.7 (4.4) **	
Involved in a Disaster	16.6%	17.7%	0.81 (0.61–1.08)
Disaster Age of Onset (years)	12.7 (5.7)	11.4 (5.6)	
Experienced an accident	21.9%	14.3%	1.52 (1.18–1.94) **
Accident Age of Onset (years)	15.9 (5.1)	14.9 (5.1)	

Note: European American race is reference level;

† Conditional only includes those who ever used alcohol;

‡ Conditional only includes those who endorsed at least 1 AUD symptom;

¥ Conditional only includes those with AUD diagnosis; Adjusted Odds Ratios (AOR) adjust for age, familial household income, mothers and fathers education, and mother's and father's lifetime history of alcohol-related problems

* p<0.05,

** p<0.01,

*** p<0.001

Table 2

Results of Cox proportional hazards regression analysis predicting alcohol initiation

		African American	European American
		HR (95% CI)	**HR (95% CI)**
Sexual Abuse	(<14)	1.22 (0.92–1.63)	**1.43 (1.08–1.87)** *
	(≥14)		1.08 (0.95–1.22)
Physical Abuse		1.01 (0.82–1.24)	0.98 (0.89–1.07)
Witness		0.97 (0.29–3.29)	1.02 (0.44–2.37)
Accident		1.24 (0.94–1.64)	1.02 (0.90–1.15)
Disaster		1.06 (0.79–1.43)	0.95 (0.86–1.05)
Tobacco Use	(<14)	**2.30 (1.67–3.17)** ***	**1.93 (1.47–2.53)** ***
	(14–16)		**2.97 (2.64–3.33)** ***
	(≥17)	**1.69 (1.28–2.24)** ***	**0.72 (0.61–0.85)** ***
Cannabis Use	(≤ 16)	**2.26 (1.76–2.90)** ***	**1.88 (1.70–2.09)** ***
	(≥17)		**1.55 (1.31–1.84)** ***
PTSD		1.22 (0.80–1.86)	0.91 (.068–1.19)

Note: Models adjusted for age, conduct disorder, lifetime major depression, familial household income, mother's and father's education, and mother's and father's lifetime history of alcohol-related problems;

* $p<0.05$,

** $p<0.01$,

*** $p<0.001$

Table 3

Results of Cox proportional hazards regression analysis predicting rate of transition from alcohol initiation to first AUD symptom

		African American	European American
		HR (95% CI)	**HR (95% CI)**
Sexual Abuse		1.23 (0.83–1.82)	1.08 (0.95–1.24)
Physical Abuse		1.07 (0.80–1.43)	**1.16 (1.04–1.31)** *
Witness		1.60 (0.68–3.76)	0.79 (0.36–1.75)
Accident		1.29 (0.93–1.78)	1.10 (0.96–1.26)
Disaster		1.03 (0.71–1.50)	0.95 (0.83–1.08)
Tobacco Use	(< 14) (≥ 14)	1.45 (0.99–2.11) †	**4.53 (1.98–10.35)** *** **1.51 (1.34–1.71)** ***
Cannabis Use		**2.18 (1.63–2.94)** ***	**1.64 (1.48–1.81)** ***
PTSD		1.37 (0.76–2.48)	0.96 (0.74–1.25)

Note: Models adjusted for age, early alcohol use, conduct disorder, lifetime major depression, familial household income, mothers and fathers education, and mother's and father's lifetime history of alcohol-related problems;

† p <0.1 (trend),

* p<0.05,

** p<0.01,

*** p<0.001

Table 4

Results of Cox proportional hazards regression analysis predicting rate of transition from alcohol initiation to AUD diagnosis

	African American	European American
	HR (95% CI)	**HR (95% CI)**
Sexual Abuse	0.74 (0.43–1.26)	**1.21 (1.01–1.44)** *
Physical Abuse	1.14 (0.73–1.77)	**1.30 (1.12–1.50)** **
Witness	1.39 (0.43–4.46)	**2.14 (1.27–3.59)** **
Accident	0.96 (0.60–1.53)	1.05 (0.87–1.26)
Disaster	0.92 (0.51–1.66)	1.13 (0.95–1.34)
Tobacco Use	1.49 (0.83–2.67)	**1.53 (1.26–1.86)** ***
Cannabis Use	**2.81 (1.74–4.54)** ***	**1.91 (1.67–2.19)** ***
PTSD	1.50 (0.61–3.67)	1.21 (0.90–1.63)

Note: Models adjusted for age, early alcohol use, conduct disorder, lifetime major depression, familial household income, mothers and fathers education, and mother's and father's lifetime history of alcohol-related problems;

* $p<0.05$,

** $p<0.01$,

*** $p<0.001$