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Progression from First Drink, First Intoxication and Regular Drinking to Alcohol Use Disorder: A Comparison of African American and European American Youth

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Abstract

Background—Differences between African Americans and European Americans in the prevalence and age at onset of alcohol use and alcohol use disorder (AUD) have been documented, but distinctions in the timing of early stage transitions and contribution of various psychiatric and psychosocial risk factors to the progression from initiation to AUD have yet to be investigated. The current study characterized progression from alcohol use initiation - defined alternatively as first drink, first intoxication, and regular drinking onset - to AUD in young African American and European American youth.

Methods—Psychiatric interviews were administered via telephone to 1,461 participants (56% African American, 44% European American) in a high-risk family study (50.3% female, mean age=17.6, SD=3.8). Cox proportional hazards regression analyses were conducted separately for the African American and European American subsamples to predict *DSM-5* AUD as a function of age at alcohol use initiation, with age at first drink, age at first intoxication, and age at regular drinking onset as the point of origin in separate models.

Results—Across race/ethnicity, regardless of how it was measured, early alcohol use initiation predicted AUD, but hazard ratios (HRs) were lowest for first drink. Regular smoking and social anxiety disorder were significant predictors in both racial/ethnic groups but associations with conduct disorder (all 3 models: HR range=2.07–4.15) and major depressive disorder (regular drinking: HR=4.51, CI:1.60–12.69 for AUD onset age ≥ 20) were specific to African Americans. Posttraumatic stress disorder (HR=5.38, CI:1.44–20.08) and generalized anxiety disorder

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(HR=7.35, CI:2.31–23.34 for AUD onset age 17) were strongly associated with progression from regular drinking to AUD exclusively in European Americans.

Conclusions—Early alcohol use initiation is a marker of risk for alcohol use disorder in both African American and European American youth, but the contributions of various psychiatric risk factors to the development of alcohol use disorder are not universal across racial/ethnic groups.

Keywords

alcohol use initiation; African Americans; alcohol use disorder; alcoholism

INTRODUCTION

Considerable research has linked early onset of alcohol use to elevated rates of subsequent alcohol and other substance involvement as well as numerous short- and long-term adverse health outcomes (Dawson et al., 2007; McGue et al., 2001; Stueve and Donnell, 2005; Swahn et al., 2008). Alcohol use during adolescence can also impact psychosocial outcomes by interfering with the development of skills and behaviors necessary to negotiate the transition to adulthood (Schulenberg and Maggs, 2002), which in turn can increase risk for heavy alcohol use. Thus, understanding how the timing of drinking onset and the psychiatric and psychosocial risk factors associated with early alcohol use contribute to the progression to problem drinking is of critical importance.

Characterization of Initiation

Based on the risks associated with early drinking onset, some researchers have advocated for the development of programs targeted at increasing age at first drink, and delaying alcohol use onset is an objective for Healthy People 2020 (U.S. Department of Health and Human Services, 2014), yet there is no precise, shared understanding of what is meant by early initiation. A variety of definitions and wordings are in use to describe the same phenomenon. (See commentary by Kuntsche et al., 2015). Initiation of alcohol use has been operationalized in several ways, ranging from any use/trying of alcohol, to use beyond a sip or a few sips/taste, to most or all of a drink of alcohol. Ages at first intoxication or regular consumption have also been proposed as important milestones in the movement from initial use to heavier and problematic use. However, the lack of consistency in defining age at alcohol use initiation has led researchers to suggest that we identify alternate phenotypes with predictive utility. One such construct is speed of progression through stages of alcohol involvement (Ridenour et al., 2006; Wagner and Anthony, 2002). Several studies have examined progression from alcohol use initiation to alcohol dependence, each defining initiation as consumption of the first drink of alcohol (Behrendt et al., 2009; DeWit et al., 2000; Sartor et al., 2007; Sartor et al., 2008). Jackson (2010) examined transitions through increasingly severe drinking milestones - first drink, first intoxication, weekly drinking, and weekly and daily consumption of 5 or more drinks - in a sample of adolescents in substance use treatment, but did not include measures of alcohol abuse or dependence. No studies to date have investigated the transition to alcohol use disorder (AUD) using an initiation measure other than first consumption of an alcoholic drink.

Racial/ethnic differences in initiation and progression in drinking

Multiple studies indicate that rates of alcohol use are lower for African Americans (AAs) than European Americans (EAs) (Jackson et al., 2002; Johnston et al., 2015; Wallace et al., 2003). Furthermore, compared to EAs, AAs report an older age at first drink of alcohol (Guttmanova et al., 2011; Johnson et al., 2005; Malone et al., 2012; Wagner et al., 2002) and first regular consumption (Guttmanova et al., 2011; Johnson et al., 2005; Swendsen et al., 2012). Findings with respect to changes in alcohol use over drinking course have been mixed. Results from an investigation with a national sample indicated that escalation in frequency of alcohol use was faster for EAs than AAs (Jackson et al., 2002) but the reverse was found in a high-risk sample (Lee et al., 2010).

Only a small number of studies have examined racial/ethnic differences in stage-specific transitions. Among the few are Swendsen et al.'s investigation (2012), which revealed significantly reduced risk for AA adolescents to transition to regular use, and Malone et al.'s study (2012), which showed decreased likelihood for AA youth to continue drinking the year after initiation. Similarly, Chung and colleagues (2013) found that alcohol use in the years following initiation was more intermittent and experimental in AA than EA girls, suggesting more rapid progression among EA girls. In contrast, Johnson et al. (2005) demonstrated a greater speed of transition from regular use of alcohol to regular drinking to intoxication among AAs, but only in adults 30 years of age and older. In a study of adolescents by Jackson (2010), transition times between milestones, particularly from weekly to daily heavy episodic drinking, were shorter for AAs than EAs. The inconsistency in findings and lack of studies examining racial/ethnic differences in the pathway from initiation to AUD – for example, in risk conferred by psychiatric or psychosocial factors associated with increased alcohol involvement - is notable, particularly in light of the evidence for racial/ethnic differences in the prevalence of risk factors such as regular smoking (Garrett et al., 2013) and depression (Hasin et al., 2005) that are associated with AUD (Grant et al., 2015).

Overview

The present study was guided by two aims: (1) to determine which measure of initiation - timing of first drink, first intoxication, or onset of regular drinking - is the most robust indicator of risk for progressing to AUD in AA vs. EA youth; and (2) to identify common and race/ethnicity-specific psychiatric and psychosocial predictors of the transition from initiation to AUD.

MATERIALS AND METHODS

Sample

The Missouri Family Study is a high-risk family study of alcoholism and related conditions in which AA families were oversampled. From 2003 to 2009, Missouri state birth records were used to identify families with at least one child aged 13, 15, 17 or 19 years and at least 1 additional full sibling. Biological mothers completed brief telephone screening interviews to determine the level of familial risk for alcoholism. Families in which the mother reported that the biological father had a history of excessive drinking were classified as "high risk."

All others were classified as “low risk.” A second sample ascertainment strategy was used to recruit additional high-risk families. Data from birth records were merged with state driving records to identify children in the targeted age range born to men with 2 or more drunk driving convictions. This group of families was classified as “very high risk.” (Risk group status was reevaluated in participating families initially categorized as “high risk” or “low risk” following the completion of full interviews with mothers, which included maternal report of the father’s DSM-IV alcohol abuse and dependence symptoms. This resulted in the reassignment of a small number of families from low to high or high to low risk, as seen in Table 1).

Mothers were interviewed first. Biological fathers were then solicited for interview and mothers were asked permission to contact the index child and up to 2 full siblings. Ninety-two percent of targeted families (450 AA families and 317 of non-AA (primarily EA) descent) were enrolled in the study. Data for the current study were drawn from the baseline interview, which was completed by 1,461 offspring (50.3% female, 56.0% AA), including 659 index children and 802 siblings. Mean ages at the time of interview were 16.7 (SD=2.7, range=13–27) and 18.4 (SD=4.3, range=12–38), for index children and siblings, respectively.

Procedure and Assessment Battery

Data were collected via telephone interview by trained interviewers using a modified version of the Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA) (Bucholz et al., 1994; Hesselbrock et al., 1999). The SSAGA was designed to assess *Diagnostic and Statistical Manual of Mental Disorders (DSM)-IV* psychiatric disorders, lifetime substance use history, related psychosocial domains (e.g., childhood abuse and neglect), and demographic characteristics. The study protocol was approved by the Washington University School of Medicine Human Research Protections Office and by the Ethics Board of the State Department of Health and Senior Services, in accordance with regulations governing the use of vital records in research. Informed consent (parental consent and offspring assent if under age 18) was obtained prior to participation.

Alcohol Milestones

Three indicators of age at alcohol use initiation were examined: *first drink*: first consumption of a full standard alcoholic drink; *first intoxication*: first “got drunk” (defined as “you couldn’t talk clearly or you were unsteady on your feet or you found it was hard to keep your balance”); and *onset of regular drinking*: first started drinking at least once a month for 6 consecutive months or at least once a week for 8 consecutive weeks. Intoxication and regular drinking were only assessed in individuals who endorsed consuming at least one full standard alcoholic drink. *Alcohol use disorder (AUD)* was determined using a proxy for the *DSM-5* diagnosis: 2 or more of the 7 *DSM-IV* dependence and 3 *DSM-IV* abuse symptoms included in the *DSM-5* diagnosis occurring in the same 12 month period. (Craving was not queried, as it was added in *DSM-5*). Age at AUD onset was defined as the age at which the 2 or more symptoms were experienced in the same 12 month period.

Predictors

Child maltreatment, a well-documented risk factor for AUD (Afifi et al., 2012), along with a wide range of psychiatric conditions associated with AUD (Grant et al., 2015) were included in models. Diagnoses of cannabis dependence, conduct disorder, major depressive disorder, generalized anxiety disorder, social anxiety disorder, panic disorder, and posttraumatic stress disorder (PTSD) were assessed according to *DSM-IV* criteria. Regular smoking was defined as smoking 100 or more cigarettes over the lifetime and at least weekly for 2 or more consecutive months. Childhood sexual abuse was queried in two sections of the interview and coded positive if either rape or sexual molestation (trauma section) or forced sexual intercourse (health habits section) were endorsed and first occurred before age 16. Childhood physical abuse and neglect were assessed in two sections of the interview and coded as positive if respondents reported physical abuse or serious neglect before age 16 (trauma section) or that between ages 6 and 13, they were often punched or punished so hard they still hurt the next day, they were hurt on purpose by an adult, or their usual punishment was “harsh non-physical” or “harsh physical” (parental discipline section). Participants reported age at onset of *DSM-IV* diagnoses, regular smoking, childhood sexual abuse, and childhood physical abuse or neglect. Self-reported maternal education level was dummy coded as less than high school and greater than high school, with high school as the comparison group. Maternal problem drinking was defined as the mother’s endorsement of one or more symptoms of *DSM-IV* alcohol abuse or dependence. (Paternal problem drinking was not entered into the models as a predictor variable, as it was the basis for ascertaining the sample and thus embedded in the study design. As noted below, models were adjusted for ascertainment strategy). The prevalence of demographic factors, psychiatric disorders, and psychosocial risk factors are shown by race/ethnicity in Table 1.

Data Analysis

Analyses were stratified by racial/ethnic group, as a pooled analysis would be statistically compromised, given the substantial sociocultural and socioeconomic separation between AAs and EAs in our sample and its impact on relevant risk factors (see Imbens and Rubin, 2015).

Timing of transitions between indicators of initiation and AUD

The timing of transitions between drinking milestones (i.e., first drink, first intoxication, regular drinking, and AUD) was calculated as the difference between the reported ages at onset of each milestone. Statistical significance of differences between AAs and EAs in the transition times was assessed with independent sample t-tests.

Prediction of AUD

Cox proportional hazards (PH) regression analyses were conducted to predict AUD as a function of age at alcohol use initiation, with age at first drink, age at first intoxication and age at onset of regular drinking each used as the point of origin in separate models. This survival analysis approach accounts for the possibility that participants who have not yet developed AUD may do so in the future and thus is particularly well suited for studies such as ours that include participants who have not passed through the peak period of risk for

AUD. It also involves testing the PH assumption that risk remains constant over time (using the Grambsch and Therneau test of the Schoenfeld residuals (Grambsch and Therneau, 1994)), thus revealing the extent to which risk associated with age at alcohol use initiation and each of the predictors varies over the period of risk. To ensure that only risk factors preceding AUD diagnosis were treated as predictors, all variables other than maternal education and maternal alcohol problems were modeled as time-varying by creating a data set with each line of data representing a single year in the participant's life. Predictors were coded as absent in the years prior to their onset and present from the year of first occurrence onward to represent the continued risk conferred by that disorder or condition (e.g., the potential impact of childhood sexual abuse beyond the years that the abuse occurred). Analyses were conducted in Stata (Statacorp, 2007), using the clustered sandwich estimator to adjust for the non-independence of observations in siblings. Age at initiation was entered into the models as two dummy variables representing the highest and lowest thirds of the distribution, i.e., early and late: 14 and 17 for first drink, 15 and 18 for first intoxication, and 16 and 19 for regular drinking. This approach allowed us to address both the degree to which early initiation elevates risk and the degree to which late initiation protects against progression to AUD. (It also produced results that can easily be compared to the existing literature on alcohol use initiation, in which individuals are typically categorized based on whether they initiated use at an early age.) Models were adjusted for ascertainment (by including dummy variables representing 4 of the 5 risk groups, with the group that consistently identified as low risk as the comparison), age, and gender. Violations of the PH assumption were resolved by breaking up the risk period and estimating hazard ratios (HRs) for each age range.

RESULTS

Prevalence and Timing of Initiation of Alcohol Use and AUD Onset by Race/ethnicity

As seen in Table 2, AAs were less likely than EAs to report ever consuming a full drink, and among those who did, AAs were less likely to report intoxication or regular drinking or to meet AUD criteria. Among participants who met AUD criteria, AAs were more likely than EAs to report never having been intoxicated (13.9% vs. 4.4%, $\chi^2_{(1)}=6.70$, $p=0.01$). Ages at onset of each indicator of initiation as well as AUD were older for AAs than EAs. The timing of transitions from initiation to AUD did not differ by race/ethnicity for any of the three indicators of initiation nor did the proportion of participants reporting becoming intoxicated the first time they drank, but compared to EAs, AAs progressed more slowly from first drink to first intoxication and more rapidly from first intoxication to regular drinking.

Age at Initiation and Progression to AUD

Results of Cox PH regression analyses predicting AUD onset as a function of age at first drink, age at first intoxication, and age at onset of regular drinking in the AA and EA subsamples are shown with 95% confidence intervals (CIs) in Tables 3 and 4, respectively.

African Americans—All early age at initiation indicators were associated with elevated risk for AUD, but risk conferred by early onset regular drinking was highest and specific to

AUD onset age 17 (HR=5.51, CI:1.79–16.99, compared to HR=1.90, CI:1.07–3.35 for first drink and HR=2.42, CI:1.15–5.12 for first intoxication). Late age at initiation was protective against AUD onset across indicators, with a similar magnitude of effect observed for first drink (HR=0.34, CI:0.20–0.59), first intoxication (HR=0.43, CI:0.25–0.75) and regular drinking (HR=0.28, CI: 0.16–0.50). Conduct disorder and social anxiety disorder emerged as significant predictors of AUD in all three alcohol use initiation models, with HRs ranging from 2.07–4.15 and 2.11–3.03, respectively. The only other common significant predictor was female gender, which was associated with lower risk for AUD in both the first intoxication (HR=0.54, CI:0.33–0.91) and regular drinking (HR=0.54, CI:0.33–0.91) models. No indicator-specific effects were observed in the first drink or first intoxication models, but three emerged in the regular drinking model. Major depressive disorder was associated with elevated risk for AUD age 20 (HR=4.51, CI:1.60–12.69), regular smoking was associated with elevated risk for AUD age 17 (HR=3.54, CI:1.30–9.67), and generalized anxiety disorder was protective against AUD (HR=0.35, CI:0.13–0.93).

European Americans—Early age at initiation was significant across indicators, with higher HRs for first intoxication (specific to AUD onset age 17, HR=3.43, CI:2.10–5.60) and regular drinking (HR=3.29, CI:2.14–5.07) than first drink (HR=1.69, CI:1.07–2.66). No other predictors were significant across all three models. However, in both first drink and first intoxication models, late initiation and female gender were associated with reduced AUD risk (first drink: HR=0.38, CI:0.23–0.62 and HR=0.62, CI:0.40–0.95; first intoxication: HR=0.39, CI:0.23–0.65 and HR=0.63, CI:0.41–0.96). Maternal alcohol problems were associated with elevated AUD risk in first drink (HR=1.56, CI:1.01–2.40) and first intoxication (HR=1.56, CI:1.02–2.37) models. Regular smoking was associated with elevated AUD risk in both first intoxication (HR=1.60, CI:1.09–2.37) and regular drinking models (HR=1.86, CI:1.19–2.89), and social anxiety disorder was associated with elevated AUD risk in both first drink (HR=1.64, CI:1.02–2.64) and regular drinking (HR=1.93, CI:1.20–3.10) models. No significant associations specific to either first drink or first intoxication were observed. However, several specific to regular drinking were found: maternal education (less than high school and greater than high school, both protective), PTSD (HR=5.38, CI:1.44–20.08) and generalized anxiety disorder for AUD onset age 17 (HR=7.35, CI:2.31–23.34).

DISCUSSION

Building on the documented differences in alcohol use between AAs and EAs and the evidence that early alcohol use initiation is a marker of heightened risk for AUD, we characterized the progression from three different indicators of initiation to AUD in AA and EA youth. Similarities were found across race/ethnicity in the magnitude of association between indicators of age at initiation and AUD but substantial differences between AAs and EAs were observed in the contribution of correlated psychiatric conditions to the progression from regular drinking to AUD, highlighting the importance of considering race/ethnicity in etiological models of AUD development.

The Pathway to AUD in African Americans and European Americans

AAs were less likely than EAs to endorse ever consuming a full alcoholic drink, becoming intoxicated, drinking regularly, or meeting AUD criteria, and across gender, the mean age at onset for each of these drinking milestones was older in AAs, consistent with a growing literature on racial/ethnic differences in alcohol use (Dauber et al., 2009; Wagner et al., 2002; Wu et al., 2011) and problem drinking (Dauber et al., 2009; Wu et al., 2011; Gruzca et al., 2008). AAs also progressed more slowly from first drink to first intoxication but faster from first intoxication to regular drinking. The proportion of (ever) drinkers who reported becoming intoxicated the first time they drank was the same across race/ethnicity, but AAs who developed AUD were less likely than their EA counterparts to report ever being intoxicated. Given the more conservative drinking norms (Herd et al., 1997) and evidence for development of AUD at lower levels of consumption in AAs vs. EAs (Zapolski et al., 2014), it is possible that AUD diagnoses in AAs were derived to a greater extent from endorsement of symptoms reflecting interference with social functioning than heaviness of consumption. Overall, findings suggest that transitions between first intoxication and other early drinking milestones cannot be treated as equivalent in AAs and EAs. However, of greatest relevance to the aims of the present study, across indicators of initiation, the rate of progression from initiation to AUD did not differ by race/ethnicity.

Age at initiation of alcohol use predicted AUD across indicators of initiation and race/ethnicity: early initiators were at elevated risk and late initiators were at reduced risk (at the trend level for regular drinking in EAs, significant in all other models), after adjusting for a range of correlates of AUD. Regular smoking and social anxiety disorder emerged as significant predictors of progression from alcohol use initiation to AUD in both the AA and EA models, but distinctions by race/ethnicity were also observed. Most notably, PTSD conferred a greater than 5-fold increase in risk for progression from regular drinking to AUD in EAs but was less than half of the magnitude and non-significant in AAs, generalized anxiety disorder conferred a greater than 7-fold increase in risk for progressing from regular drinking to AUD at age 17 or younger in EAs but was protective in AAs. Finally, whereas conduct disorder was associated with significantly elevated risk for AUD across measures of initiation in AAs, it was not significant for any initiation indicators in EAs.

The absence of association between conduct disorder and AUD in EAs was unexpected, given the extensive documentation of their link in the larger literature (Regier et al., 1990; Weinberg et al., 1998). This finding could potentially be accounted for by high comorbidity of conduct disorder with internalizing disorders (Goodwin and Hamilton, 2003; Morcillo et al., 2012), which were robust predictors in the regular drinking model. To address this possibility, we reran all three models for EAs after dropping major depressive disorder and all anxiety disorders. Conduct disorder remained non-significant, thus ruling out this potential explanation, although overlap with other significant predictors (e.g., regular smoking) could also account for this pattern of findings. (High and very high risk group variables were non-significant in all EA models and thus did not appear to act as proxies for conduct disorder.) The consistency of our results with Chung et al.'s (2013) finding that conduct problems were highly predictive of drinking onset in AA girls but not EA girls

suggests the merit of further investigating racial/ethnic differences in the relationship between externalizing problems and alcohol outcomes.

The association of internalizing disorders with progression to AUD in both racial/ethnic groups is noteworthy as well, given the lesser attention in the larger literature to the role of internalizing than externalizing problems on the development of AUD. Social anxiety disorder, which, with one exception, predicted AUD across indicators and racial/ethnic groups, has been linked to alcohol related problems in numerous studies (e.g., Buckner et al., 2008; Grant et al., 2004), including a recent study of adolescents, in which risk for severe AUD was doubled in adolescents who met criteria for social anxiety disorder (Black et al., 2015). The contribution of anxiety disorders to AUD across the adolescent to early adult years is also well documented. For example, in a longitudinal study by Goodwin et al. (2004), a greater than 2-fold increase in odds of developing alcohol dependence was observed among individuals with anxiety disorders in both the developmental periods that were assessed: ages 16 to 18 and ages 18 to 21. Interpretation of the elevated risk for progression from regular drinking to AUD associated with generalized anxiety disorder in EAs should be made with caution, given the wide confidence intervals and overall trend in point estimates for other initiation indicators, but co-occurrence of AUD with the other internalizing disorders that emerged as significant predictors, PTSD (Kessler et al., 1995) and major depressive disorder, have consistently been found in the larger literature (Grant et al., 2004; Hasin et al., 2007).

Utility of Various Indicators of Age at Initiation for Predicting AUD

The magnitude of association between initiation of alcohol use and AUD was highest for age at onset of regular drinking in AAs, equally high for regular drinking and first intoxication in EAs, and lowest for first drink in both racial/ethnic groups. The finding that the timing of a drinking milestone reached later in drinking course is a stronger predictor of AUD than timing of the earliest milestone is not surprising, particularly given that under half of ever drinkers in our sample met the definition for regular drinking. The regular drinking models also had the largest number of significant predictors; thus, the pathway from regular drinking to AUD was better characterized than the pathways from either first drink or first intoxication to AUD. Although potentially more informative for developing interventions in terms of identifying risk and protective factors, on an individual level, the utility of age at regular drinking onset as a marker of risk is limited. The average transition time from regular drinking to AUD onset in our sample was only 6 months - with over 40% reporting regular drinking and AUD onset in the same year and 18.1% reporting onset of AUD prior to regular drinking - which translates into a very brief, if any, window for intervention. Similarly, regular drinking typically starts at 17 to 18 years of age, so age at onset of regular drinking would not be a useful marker of risk in an early adolescent population. In addition, 1 in 10 EAs and nearly 1 in 5 AAs who met AUD criteria never endorsed regular drinking, so assessing AUD risk in terms of age at onset of regular drinking would result in a substantial proportion of at-risk drinkers - particularly AA at-risk drinkers - going undetected. By contrast, age at first drink, which can be assessed in every individual who has consumed alcohol and preceded AUD by nearly 3 years in this sample, was more modestly associated

with AUD, but it was statistically significant in both AA and EA subsamples, and thus would be well suited as a marker of risk in either AA or EA adolescent populations.

Limitations

Study findings should be interpreted with several limitations in mind. First, the average age of the sample was between 17 and 18 years and, as in other studies examining AUD in this age range, many participants had not yet passed through the peak period of risk for AUD. However, since some participants were much older, results may differ from studies recruiting exclusively adolescents, even after adjustment for age, given the greater heterogeneity in our sample and the differences between adolescents and adults in reliability of assessments of certain AUD symptoms (Chung and Martin, 2005). Second, although the overrepresentation of low-risk and high-risk families in the current sample would not be expected to substantially impact estimated associations of initiation or psychosocial risk factors with progression to AUD, the rate of endorsement of psychiatric disorders, alcohol milestones and AUD are somewhat higher than those in the general population. Third, although most of the sample was ascertained in the early to peak years of alcohol use initiation and analyses were adjusted for age, retrospective assessments are inherently vulnerable to retrospective reporting bias. Fourth, participants reported their age rather than dates at the time drinking milestones were reached, so we were unable to determine timing of transitions more precisely than yearly intervals. Finally, the sample size likely limited statistical power to detect associations between AUD and psychiatric disorders of low prevalence.

Conclusions and Future Directions

Despite lower rates of initiation and AUD and older age at onset for all stages of alcohol use in AAs compared to EAs, regardless of how it was measured, early initiation was a robust predictor of AUD in both racial/ethnic groups. Racial/ethnic differences in the association of various psychiatric conditions to the progression to AUD, the endorsement of intoxication and regular drinking prior to AUD onset, and the timing of initiation of alcohol use indicate that the most useful indicator of alcohol use initiation for gauging AUD risk depends on the age and racial/ethnic composition of the population. However, the observed consistencies in the association between social anxiety disorder and AUD risk across racial/ethnic groups and between conduct disorder and AUD in AAs suggest some common sources of psychosocial risk, that is, adolescents who are isolated from peers either because of anxiety or behavioral problems are more vulnerable to rapid escalation to problem drinking.

The observed differences between AAs and EAs in the current study underscore the importance of including members of other racial/ethnic groups in future studies to identify common as well as race/ethnicity-specific markers of AUD risk and influences on AUD development. Consideration of sociocultural risk factors for alcohol related problems that disproportionately impact racial/ethnic minorities, including poverty and discrimination (Hunte et al., 2012) - and may differentially influence alcohol outcomes (Zapolski et al., 2014) and their correlates (e.g., depression) - is critical to the development of more nuanced models of the pathways from initiation to AUD.

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Table 1

Sample characteristics and prevalence of psychiatric and psychosocial risk factors

	Full sample (n=1,461)	African Americans (n=818)	European Americans (n=643)
Female	50.3%	49.8%	51.0%
Age: Mean (SD) ^a	17.6 (3.8)	17.8 (3.9)	17.4 (3.5)
Risk group ^a			
Low - consistently identified as low	31.7%	36.0%	26.1%
Low - initially identified as high	6.6%	8.7%	3.9%
High - consistently identified as high	21.6%	22.9%	20.1%
High - initially identified as low	4.6%	4.8%	4.5%
Very high	35.5%	27.6%	45.4%
Mother's level of education ^a			
< high school	8.7%	10.6%	6.1%
high school	30.7%	31.1%	30.3%
> high school	60.6%	58.3%	63.6%
Maternal alcohol problems ^a	24.2%	18.3%	31.7%
Childhood sexual abuse	7.6%	9.3%	5.6%
Childhood physical abuse or neglect ^a	36.5%	49.8%	19.6%
Regular smoking ^a	18.3%	13.5%	24.4%
Cannabis dependence	3.9%	3.7%	4.1%
Conduct disorder	7.2%	7.7%	6.5%
Major depressive disorder	10.9%	10.4%	11.6%
Generalized anxiety disorder	4.0%	4.2%	3.7%
Posttraumatic stress disorder	2.8%	3.2%	2.3%
Panic disorder	2.2%	1.6%	3.0%
Social anxiety disorder	20.3%	20.2%	20.4%

^a difference between African Americans and European Americans significant at p<0.05

Table 2

Prevalence and timing of initiation of alcohol use and AUD onset

	Full sample (n=1,461)	African Americans (n=818)	European Americans (n=643)
Rates of Initiation and AUD			
Consumed full alcoholic drink ^a	54.8%	51.2%	59.5%
Became intoxicated (among drinkers) ^a	61.9%	51.5%	73.3%
Intoxicated first time drank ^b	28.5%	28.9%	28.2%
Drank regularly (among drinkers) ^{a,c}	44.5%	38.5%	50.9%
AUD (among drinkers) ^d	31.8%	26.0%	38.2%
Age at Initiation and AUD onset: Mean (SD)			
First drink ^a	15.7 (2.6)	16.2 (2.8)	15.0 (2.3)
First intoxication ^a	16.5 (2.6)	17.3 (2.7)	15.9 (2.2)
Regular drinking ^a	17.4 (2.6)	18.0 (2.6)	17.0 (2.5)
AUD ^a	17.6 (2.6)	18.3 (2.6)	17.1 (2.6)
Timing of early stage transitions (years): Mean (SD)			
First drink to first intoxication ^a	1.1 (1.6)	1.3 (2.0)	0.9 (1.3)
First drink to regular drinking	2.1 (2.1)	2.0 (2.3)	2.2 (2.1)
First intoxication to regular drinking ^a	1.1 (1.9)	0.8 (2.0)	1.3 (1.8)
Timing of transitions to AUD (years): Mean (SD)			
First drink to AUD	2.7 (2.6)	2.9 (3.0)	2.6 (2.3)
First intoxication to AUD	1.6 (2.3)	1.5 (2.8)	1.7 (1.9)
Regular drinking to AUD [*]	0.5 (1.8)	0.6 (1.9)	0.4 (1.8)

^a difference between African Americans and European Americans significant at p<0.05;^b among individuals reporting ever becoming intoxicated;^c monthly for at least 6 consecutive months or weekly for at least 8 consecutive weeks; drinker=individual who endorsed consumption of at least one standard alcoholic drink;^{*} 18.1% of participants reported an earlier age at onset of AUD than regular drinking, 40.2% reported the same age at onset for regular drinking and AUD, and 41.7% reported an earlier age at onset of regular drinking than AUD; prevalence of AUD in the full sample=12.8% for African Americans and 22.0% for European Americans.

Table 3

Results of Cox proportional hazards regression analyses predicting AUD onset from age at first drink, age first intoxicated and age at onset of regular drinking in African Americans^a

	First drink	First intoxication	Onset of regular drinking
	HR (95% CI)	HR (95% CI)	HR (95% CI)
<i>Timing of alcohol use initiation</i>			
<i>Early age at initiation^b</i>			
AUD onset age 17	1.90 (1.07-3.35)	2.42 (1.15-5.12)	5.51 (1.79-16.99)
AUD onset age 18			0.34 (0.11-1.08)
<i>Late age at initiation^c</i>			
	0.34 (0.20-0.59)	0.43 (0.25-0.75)	0.28 (0.16-0.50)
<i>Demographic, psychosocial, and psychiatric factors</i>			
Female	0.78 (0.50-1.23)	0.54 (0.33-0.91)	0.46 (0.28-0.77)
<i>Mother's level of education^d</i>			
< high school	0.38 (0.12-1.22)	0.46 (0.12-1.76)	0.78 (0.18-3.28)
> high school	0.92 (0.55-1.52)	0.81 (0.51-1.28)	0.99 (0.59-1.68)
Maternal alcohol problems	1.22 (0.66-2.27)	1.14 (0.64-2.03)	1.29 (0.73-2.28)
Childhood sexual abuse	0.48 (0.21-1.11)	0.63 (0.24-1.63)	0.42 (0.17-1.02)
<i>Childhood physical abuse or neglect</i>			
AUD onset age 17	1.03 (0.63-1.70)	1.32 (0.54-3.23)	0.75 (0.42-1.33)
AUD onset age 18		0.97 (0.54-1.75)	
<i>Regular smoking</i>			
AUD onset age 17	1.32 (0.71-2.46)	1.04 (0.54-2.00)	3.54 (1.30-9.67)
AUD onset age 18			1.40 (0.75-2.63)
<i>Cannabis dependence</i>			
	1.51 (0.51-4.46)	1.09 (0.37-3.26)	1.32 (0.55-3.15)
<i>Conduct disorder</i>			
	2.07 (1.10-3.90)	2.23 (1.25-4.00)	4.15 (2.41-7.15)
<i>Major depressive disorder</i>			

	First drink	First intoxication	Onset of regular drinking
	HR (95% CI)	HR (95% CI)	HR (95% CI)
AUD onset age 19	1.02 (0.51-2.02)	1.02 (0.52-2.00)	0.49 (0.13-1.85)
AUD onset age 20	1.02 (0.51-2.02)	1.02 (0.52-2.00)	4.51 (1.60-12.69)
Generalized anxiety disorder	0.53 (0.20-1.42)	0.59 (0.17-2.07)	0.35 (0.13-0.93)
Posttraumatic stress disorder	0.93 (0.27-3.15)	0.59 (0.12-2.82)	2.46 (0.95-6.36)
Panic disorder	1.59 (0.47-5.35)	1.72 (0.52-5.68)	1.28 (0.23-7.17)
Social anxiety disorder	2.11 (1.17-3.81)	3.03 (1.68-5.45)	2.14 (1.08-4.24)

AUD=alcohol use disorder; HR=hazards ratio; CI=confidence intervals; significance at $p<0.05$ shown in bold;

^a adjusted for age and risk group, using low risk consistently defined as low risk as the comparison group;

^b early: 14 for first drink, 15 for first intoxication, 16 for onset of regular drinking;

^c late: 17 for first drink, 18 for first intoxication, 19 for regular drinking;

^d high school as comparison group. Bars indicate that the hazard ratio is for the full period of risk rather than the subdivisions listed in the far left column.

Table 4

Results of Cox proportional hazards regression analyses predicting AUD onset from age at first drink, age first intoxicated and age at onset of regular drinking in European Americans^a

	First drink	First intoxication	Onset of regular drinking
	HR (95% CI)	HR (95% CI)	HR (95% CI)
<i>Timing of alcohol use initiation</i>			
Early age at initiation ^b			
AUD onset age 17	1.69 (1.07-2.66)	3.43 (2.10-5.60)	3.29 (2.14-5.07)
AUD onset age 18		1.00 (0.48-2.09)	
Late age at initiation ^c			
AUD onset age 20	0.38 (0.23-0.62)	0.39 (0.23-0.65)	0.38 (0.14-1.00)
AUD onset age 20			0.78 (0.29-2.11)
<i>Demographic, psychosocial, and psychiatric factors</i>			
Female	0.62 (0.40-0.95)	0.63 (0.41-0.96)	0.84 (0.54-1.32)
Mother's level of education ^d			
< high school	0.41 (0.13-1.33)	0.80 (0.31-2.06)	0.46 (0.22-0.95)
> high school	1.11 (0.75-1.64)	0.96 (0.65-1.42)	0.63 (0.43-0.91)
Maternal alcohol problems	1.56 (1.01-2.40)	1.56 (1.02-2.37)	1.30 (0.86-1.96)
Childhood sexual abuse	1.99 (0.87-4.56)	1.83 (0.79-4.21)	0.86 (0.36-2.07)
Childhood physical abuse or neglect	0.69 (0.39-1.25)	0.56 (0.31-1.01)	1.13 (0.73-1.75)
Regular smoking	1.46 (0.94-2.27)	1.60 (1.09-2.37)	1.86 (1.19-2.89)
Cannabis dependence	1.69 (0.67-4.28)	1.85 (0.81-4.22)	0.96 (0.49-1.87)
Conduct disorder	1.73 (0.80-3.72)	1.34 (0.69-2.61)	1.37 (0.76-2.48)
Major depressive disorder	1.55 (0.89-2.70)	1.58 (0.92-2.71)	1.10 (0.65-1.89)
Generalized anxiety disorder			
AUD onset age 17	0.56 (0.15-2.05)	0.93 (0.22-3.86)	7.35 (2.31-23.34)
AUD onset age 18			0.49 (0.07-3.60)

	First drink	First intoxication	Onset of regular drinking
	HR (95% CI)	HR (95% CI)	HR (95% CI)
Posttraumatic stress disorder	1.32 (0.38–4.54)	1.60 (0.45–5.74)	5.38 (1.44–20.08)
Panic disorder	1.07 (0.33–3.47)	1.06 (0.37–3.07)	0.66 (0.32–1.37)
Social anxiety disorder	1.64 (1.02–2.64)	1.49 (0.94–2.37)	1.93 (1.20–3.10)

AUD=alcohol use disorder; HR=hazards ratio; CI=confidence intervals; significance at p<0.05 shown in bold;

^a adjusted for age and risk group, using low risk consistently defined as low risk as the comparison group;

^b early: 14 for first drink, 15 for first intoxication, 16 for onset of regular drinking;

^c late: 17 for first drink, 18 for first intoxication, 19 for regular drinking;

^d high school as comparison group. Bars indicate that the hazard ratio is for the full period of risk rather than the subdivisions listed in the far left column.