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Age and Ethnic Differences in the Onset, Persistence and Recurrence of Alcohol Use Disorder

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Abstract

Aims—To estimate ethnic differences in three components of alcohol use disorder and alcohol dependence course (onset, persistence and recurrence) in a developmental framework.

Design—Longitudinal data from The National Epidemiologic Survey of Alcohol and Related Conditions (NESARC), collected using face-to-face interviews.

Setting—Civilian non-institutionalized US population aged 18 years and older, with oversampling of Hispanics, Blacks and those aged 18–24.

Participants—Individuals who completed both NESARC assessments, were not lifelong abstainers, and were either White (n=17,458), Black (n=4995), US-born Hispanic (n=2810), or Hispanic-born outside the US (n=2389).

Measurements—Alcohol dependence (AD) and alcohol use disorder (AUD; abuse or dependence) onset, persistence and recurrence were examined using the Alcohol Use Disorders and Associated Disabilities Interview Schedule, DSM-IV version.

Findings—Among men: relative to Whites aged 18–29, AUD onset and persistence were elevated only in US-born Hispanics 40 and older; odds were reduced for all non-US born Hispanics, older Whites, most Blacks, and US-born Hispanics aged 30–39. For AD, onset risk was elevated for all younger minority men and only reduced among non-US born Hispanics 40 or older. For women: compared to young Whites, non-US born Hispanics were at decreased AUD and AD onset risk; AUD and AD onset and persistence were increased for older Blacks and US-born Hispanics.

Conclusions—Ethnic differences in alcohol disorder transitions (onset, persistence, and recurrence) vary across age, gender, and whether a broad (alcohol use disorder) or narrow (alcohol dependence) alcohol definition is used. Evidence of increased risk for some transitions in minority groups suggests that attention should be paid to the course of alcohol use disorders, and that differences in prevalence should not be assumed to reflect differences in specific transitions.

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Introduction

Ethnic differences in the prevalence of alcohol use disorder have been well-documented in large psychiatric epidemiologic studies over the last 30 years, with findings indicating that compared to their White counterparts, prevalence is lower among Blacks and Asians, higher among Native Americans, and similar among Hispanics[1–6]. Equally well-established in the literature is the association between age and the prevalence of alcohol use disorders. Findings from studies[2, 7–9] indicate that prevalence of alcohol use disorders is highest in those 18–29 years old and lower among older age groups. In general, ethnicity and age have been studied separately, rather than jointly, with many[10–12] (but not all[13]) failing to consider that age effects may differ across ethnic groups. Thus, the degree to which age associations with the prevalence of alcohol use disorder may differ across ethnic groups has not been consistently investigated.

Although overall prevalence does capture for a given time-point the number of affected individuals, and thus is useful for estimating disease burden and treatment planning, it is a heterogeneous indicator of illness combining new, persistent and recurrent cases that are not distinguished yet have different implications for prevention and treatment. For example, as pointed out by others[14], differences in persistence of disorder, unlike new onset of disorder, may indicate unequal access to, lower retention in, or differential efficacy of, treatment in different ethnic, gender or age sub-groups. Potential policies to impact rates of persistent disorder include removal of barriers to improve access to treatment, or the addition of culturally sensitive elements to treatment regimens to promote retention of ethnic minorities in programs. In contrast, new occurrence of disorder is addressed in universal prevention efforts that typically involve a broader, systemic approach to address the host of factors that precede the disorder, an approach that arguably is less amenable to short-term policy directives. As well, there may be age, ethnic, gender or other socio-demographic differences underlying each type of case that are meaningful from a prevention or services perspective but are obscured when these are subsumed in an overall prevalence rate.

In some alcohol studies, prevalence has been disaggregated into its constituent pieces, most commonly persistence/remission[10–12], less commonly onset and recurrence/relapse[10,11]. In the few studies where ethnic differences in course has been an objective, Blacks and Hispanics were found to be significantly more likely to have persistent DSM-IV alcohol dependence diagnosis compared to Whites (based on cross-sectional data)[3]. Another study discovered significantly greater persistence of mood and anxiety disorders for Blacks and Hispanics compared to Whites, but failed to find similar evidence for alcohol abuse/dependence[14]. (However, the likely under-diagnosis of alcohol abuse in the National Comorbidity Survey data[2], which was observed disproportionately in minority women and men[9] may partly account for the disagreement with prior work.) Remission/recovery studies have for the most part not reported on interactions of age and ethnicity.

Thus, in light of the paucity of research on ethnic-age differences in course of alcohol use disorder, we took advantage of the longitudinal data in a large general population survey of the U.S. household adult population to explore age-related differences in course of alcohol use disorders across ethnicity groups. We consider three transitions– onset, persistence and recurrence of disorder – and investigate these for a broad outcome of Alcohol Use Disorder (AUD), which is a combination of alcohol abuse (AA) and/or dependence, and for a narrow outcome, alcohol dependence (AD; ignoring AA status). AUD may be thought of as an approximation for the proposed DSM-5 definition for substance use disorder, where the separate categories of abuse and dependence will be eliminated and in their place a single diagnosis made based on criteria from both. The large sample available from the National Epidemiologic Survey on Alcohol and Related conditions (NESARC), in which data were

collected at two points in time over a three year interval, permits contrasting course among 4 ethnic groups with sufficient numbers – Whites, Blacks, Hispanics born in the United States (H-US), and Hispanics born outside the United States (H-nonUS). As has been reported[15,16], country of birth is an important factor when comparing prevalence in Hispanics to others. Foreign-born Latinos (Mexican-Americans[15], Puerto Ricans and Cubans[16]), compared to their U.S. born counterparts, are at lower risk of DSM-IV lifetime alcohol abuse and dependence both separately and combined. This makes it possible to disentangle the effects of immigration from those of ethnicity[15–18].

Method

Participants

The base sample for the present analyses was individuals who completed interviews for both assessments of the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC). As described in detail elsewhere[11,19–20], Wave 1 included 43,093 respondents 18 years of age and older who completed an in-person interview in 2001–2002, and Wave 2 included follow-up in-person interviews with 34,653 individuals in 2004–2005. NESARC targeted the civilian non-institutionalized US population, with oversampling of Blacks, Hispanics, and adults aged 18–24. Sampling weights are used in analyses to yield a sample representative of the target population[13].

The present analyses are restricted to the 27,652 Wave 2 participants of White, Black, and Hispanic ancestry. Excluded from the analyses were 4660 lifelong abstainers, as well as non-abstainers who were of Asian (n=690) or Native American (n=500) heritage, or were of White (n=798) or Black (n=351) ancestry but were not U.S. born. These groups of drinkers were excluded due to small cell sizes for the alcohol use disorder transitions. The final weighted sample was 77.5% White (unweighted n=17458), 10.5% Black (unweighted n=4995), 5.9% US-born Hispanic (H-US, unweighted n=2810), and 6.1% foreign-born Hispanics (H-nonUS, unweighted n=2389). The weighted sample was 49.4% female, had a mean age of 44.4 years at Wave 1 and 47.5 years at Wave 2.

Measures

DSM-IV alcohol abuse and dependence—For both Waves, past-year and lifetime DSM-IV alcohol abuse (AA) and dependence (AD) were assessed using the Alcohol Use Disorders and Associated Disabilities Interview Schedule—Version for DSM-IV[21] (AUDADIS-IV), with well-documented reliability[22,23]. Separate analyses were conducted for DSM-IV AD and for alcohol use disorder (AUD) which included both AA and AD. The course definitions were based on Wave 1 status for AUD/AD and on the interval diagnosis of AUD/AD obtained at Wave 2. For the interval diagnosis, disorders that occurred at any time between Wave 1 and Wave 2 were included. At Wave 1, past year and prior-to-past-year assessments were collected; at Wave 2, diagnosis was assessed for the past year and the interval between interviews, not for the entire lifetime.

Alcohol use disorder transitions—Using data from both Waves 1 and 2, three types of AUD and AD transitions were constructed: *onset*, *persistence*, and *recurrence*. Outcomes were calculated as a proportion of those at risk, not as a proportion of the population as a whole. Thus, the proportion reported for each outcome should not be construed as the population-based rate. The fraction of the at-risk group converting to a given outcome could give rise to very different overall prevalence rates in the overall population, depending on the magnitude of the at-risk group itself, and the converse is also true- the same population-based rate of outcome could reflect markedly different underlying conversion rates in the group at risk. For an overall estimate in the population, one must take into account not only

the conversion rate among the at-risk group, but also the proportion of the total population that the at-risk group reflects.

Only individuals who were unaffected at Wave 1 (that is, had neither a past-12-month AUD or a prior to past 12 month AUD) were included in the calculation of new cases of AUD at Wave 2. Only individuals at Wave 1 with prior, but not current (i.e., past 12 months), AUD were at risk for "recurrence" at Wave 2; and only those with current AUD at Wave 1 were at risk for "persistence" at Wave 2. Thus, AUD *onset* included individuals who had no AUD diagnosis at Wave 1 but met criteria for AUD during the interval between Waves 1 and 2. *Persistent* AUD was included those who had a diagnosis of AUD in the 12 months preceding Wave 1 and met criteria for AUD in the interval between Waves 1 and 2. *Recurrent* AUD occurred among respondents who had a lifetime, but not current, diagnosis of AUD at Wave 1, and who met criteria for AUD at Wave 2. Cases of AD *onset, persistence*, and *recurrence* (ignoring AA status) were defined in a similar fashion. Transitions are expressed as the fraction of those with the outcome among those at risk for the outcome.

Predictors—The primary set of predictor variables was ethnicity, with Whites being the reference group. Dummy variables were coded for Blacks, H-US, and H-nonUS. Age at baseline was divided into approximate quintiles with the oldest three quintiles later collapsed due to low rates of AUD/AD transitions. Baseline age was included as a predictor in all models, with 18–29 year olds (unweighted n=5492, weighted 22.2%) being the reference group, and 30–39 year olds (unweighted n=6023, weighted 20.6%), and 40 or older (unweighted n=16137, weighted 57.2%) included as dummy variables.

Analyses

Logistic regression analyses were conducted via PROC SURVEYLOGISTIC using SAS® software[24], which allowed adjustments for the NESARC sampling design. Preliminary analyses tested for age by ethnicity interactions, and significant interactions were retained in final models, consistent with our interest in investigating ethnic differences in course within a developmental framework. Because preliminary analyses indicated there were age by gender interactions, all analyses were run separately for men and women. This allowed for examination of age by ethnicity interactions as well as potential gender differences.

Results

Table 1 displays weighted prevalence estimates of unaffected, AA, AD and AUD individuals by ethnicity for both Waves. In Table 2, the number of participants eligible for each AUD and AD transition and the percentage who transitioned are presented by ethnicity, gender, and age. Overall, in the interval between waves 1 and 2, 11.4% of men and 4.6% of women at risk for new AUD became affected, 60.6% of men and 48.3% of women at risk remained affected by AUD, and 16.6% of men and 13.1% of women at risk experienced a recurrence of AUD (all percentages are weighted). Comparable rates for AD transitions were 5.2% and 2.7%% for *onset*, 47.4% and 42.2% for *persistence*, and 12.5% and 10.8% for *recurrence*.

Alcohol use disorder transitions

Results from the logistic regression analyses for AUD transitions are shown separately for males and females in Tables 3 and 4. Relative to White men aged 18–29 years, only H-US men 40 or older had significantly elevated odds of AUD *onset* and *persistence* between Waves 1 and 2. In contrast, AUD *onset* and *persistence* odds were significantly lower among White men in other age groups, H-nonUS men in each age group, and H-US men

30–39 years of age. Compared to White men aged 18–29, the odds of AUD *onset* were similar in H-US men aged 18–29, and for Black men of all ages. However, Black men of all ages were at significantly reduced risk of having *persistent* AUD relative to 18–29 year old White men.

Results for *recurrent* AUD indicated that Black men aged 18–29 were more likely than their similarly aged White counterparts to have an AUD *recur* between Waves, but Black men 30–39 years did not differ from the younger White men, and Black men 40 or older were at reduced risk of an AUD *recurrence*. Although there was no evidence of age differences among Hispanic men regardless of country of nativity, those who were not US-born had reduced odds of AUD *recurrence*, while those who were US-born had similar odds of AUD *recurrence*, compared to White men 18–29. Among White men, decreased odds for AUD *recurrence* were observed for each older age group compared to their younger counterparts.

The patterns were different among women. Although all 18–29 year-old non-White groups had significantly reduced odds of AUD *onset* relative to White women aged 18–29 years, this pattern was reversed in the older age groups, with Black and H-US women 40 years and older, and Black women 30–39 years old, at increased risk of AUD *onset* compared to young White women. H-nonUS women 40 years and older did not differ from the young White women on risk of AUD *onset*.

AUD *persistence* showed a similar pattern to AUD *onset* for both the Black and H-US women, with reduced odds of *persistence* among women aged 18–29, and increased odds among the women in the older groups. In contrast, among H-nonUS women, odds of AUD *persistence* were elevated among those 18–29, compared to White women 18–29.

Regarding AUD *recurrence*, compared to White women aged 18–29, Black women 18–29 and H-nonUS born women aged 30–39 had reduced odds, but Black and H-US women aged 30–39, H-US women aged 30 or older, and H-nonUS women aged 40 or older had increased odds of AUD *recurrence*. For White women 30–39, and 40 or older, risk of AUD *recurrence* was significantly reduced compared to their 18–29 counterparts.

Alcohol dependence transitions

Results from logistic regression analyses of AD transitions are shown in Tables 5 (men) and 6 (women). For men, results for AD transitions differed from those observed for AUD. Black men had increased risk for AD *onset*, compared to White men aged 18–29, with no evidence of age differences. H-US men 18–29 and 40 or older also had increased risk of AD onset, as did H-nonUS born men under age 40. Only White men over age 29 and H-nonUS men who were aged 40 or older had significantly reduced risk of AD *onset* compared to young White males. Black, H-US and H-nonUS men aged 18–29 had lower odds of AD *persistence*, as did H-US men aged 30–39 and all older H-nonUS men. However, White males 30 years or older had significantly increased risk of AD *persistence* compared to their 18–29 year old counterparts. Relative to young White men, H-nonUS men had increased odds of AD *recurrence* (with no evidence of age differences), whereas White men over age 29 had reduced risk of *recurrence*.

In women, 18–29 year-old Black and H-US women, and H-nonUS women of all ages, had reduced odds of AD *onset* relative to White women aged 18–29 years. However, this pattern was reversed in the older age groups among Black and H-US women 30 and older, where odds of AD *onset* were increased. Compared to young white women, AD *persistence* risk was significantly reduced in Black women 18–29 years old, but significantly increased in Black women 40 and older, H-US women aged 30 or older, and H-nonUS women aged 18–29. Risk of AD *persistence* did not differ by age among White women. Among White

women, AD *recurrence* was significantly lower in older women compared to women aged 18–29. Only H-US women aged 30–39 years of age had significantly elevated odds of AD *recurrence*.

Discussion

Our analyses indicate substantial ethnic differences in AUD and AD transitions across age groups for men and women, and further, that results vary based on whether a broad (AUD) or narrow (AD) transition is used. Consistent with a prior NESARC report that focused on Mexican-Americans only[15], we find that H-nonUS men are at lower risk compared to young White men for all AUD course outcomes. This is consistent with the healthy immigrant hypothesis: compared to their US-born counterparts, immigrants are at lower risk for the development of AUD[15–18] (although the current findings do not provide a strong test of the healthy immigrant hypothesis since there is no sample of Hispanics not living in the US to compare to the non-US-born immigrant sample[25–28]). Interestingly, the pattern of reduced risk among H-nonUS men does not hold in our sample when examining AD *onset* or *recurrence* (i.e., relative to young Whites, H-nonUS men under the age of 40 have increased risk of AD *onset*, as do all H-nonUS men for AD *recurrence*). This is in line with recent studies of Mexican migrants and non-migrants, where excess rates of substance use disorders were observed in migrants and in families of migrants, compared to non-migrant Mexicans[25,26], suggesting possible selection in who migrates, such as those more vulnerable to develop (or experience recurrence of) disorder in the context of immigration-associated stress, or perhaps differences in genetic risk for AUD/AD for migrants vs. non-migrants. Other possible explanations for this effect include cohort effects and differences between migrant and non-migrant Hispanics in nature of employment, disposable income, and the drinking cultures encountered. Further, the combination of reduced risk for AUD transitions and increased risk for AD transitions in men suggests that risk for alcohol abuse and alcohol dependence are not always parallel, a finding that has implications for DSM-5, in which the disorders will be combined.

In contrast, for women our findings are parallel for AUD and AD transitions, with the odds of both AUD and AD *onset* and *persistence* reduced among young women, and elevated among older Black and H-US women (in comparison to young White women). The pattern appears particularly pronounced for AD transitions in Black women but is evident in H-US women 30 and older, and is consistent with previous literature suggesting that the peak for alcohol use disorders occurs later in life for Blacks compared to their White counterparts[7].

Although a detailed examination is beyond the scope of this manuscript, one possible explanation for the higher *onset* and *persistence* risk found in older H-US men and women is acculturation stress[29], high levels of which have been linked to substance use disorders in other studies[30]. A model of acculturation[31] suggests that H-US individuals may be at higher risk of losing the connection with the original culture yet may not be completely acculturated to the new one, and thus are at risk of becoming marginalized, a situation that has been found to be associated with high levels of distress[32].

Our data highlight ethnic differences in persistent disorder, which is elevated in middle aged Black and H-US women compared to young White women, and may extend to young H-nonUS women. This may reflect reduced access to care, testable with NESARC data, but beyond the scope of the present report. Recent evidence indicates there are ethnic differences in alcohol-based treatment utilization, particularly among those more severely affected[33]: relative to Whites, more severely affected Blacks and Hispanics are less likely to seek alcohol treatment, more severely affected Blacks are less likely to have used mental health services, and more severely affected Hispanics are less likely to use mutual aid. In

addition, Blacks and Hispanics are less likely to see a non-specialist health professional regarding alcohol services[33]. These differences may be associated with differential barriers to care, such as disposable income or insurance coverage. They may also stem from ethnic differences in the perceived stigma of alcoholism, which could be associated with perceived acceptability of treatment. However, although Smith et al. (2010) found that the stigma for alcoholism was lowest among Whites and Native Americans, higher among Blacks, and highest among Asians and Hispanics, they found no evidence that perceived stigma was associated with treatment utilization[34].

Among Whites, for both women and men, the age-specific odds for AUD *onset* and *recurrence* are lower with increasing age, a result that does not hold when examining AD *persistence* (where the odds are increased for men, and are not different for women, across age category). It may be that AA, which is included in AUD but not AD transitions, is more strongly related to age than is AD. The removal of a distinct abuse designation that is currently proposed for the upcoming DSM-5 system may help sort out age-related differences that are driven by a single construct.

Some limitations must be acknowledged. These are self-report data, and thus are vulnerable to bias associated with recall and insight. This is a generic problem associated with survey data, and we would not expect NESARC to be more vulnerable to reporting bias than other survey-based reports. The follow-up rates, although excellent, were lower for H-nonUS (73%) than for other groups (80–82%), which may affect the results. However, follow-up rates were similar across lifetime alcohol abuse/dependence status within ethnicity, including H-nonUS, suggesting that affected individuals were not disproportionately lost to follow-up, a reassuring finding. The available sample is too small to support examination of potential distinctions between Hispanics by area/country of origin, despite reports of subgroup differences in alcohol use and disorder[16, 17].

Our findings suggest that ethnic differences in AUDs are not limited to differences in prevalence, but also extend to the transitions involving alcohol abuse and dependence. Moreover, the discrepancies between AUD and AD transitions, particularly for men, suggest that the risks for alcohol abuse and alcohol dependence are not always parallel, and that caution should be used in combining the disorders when examining alcohol transitions. Furthermore the discrepancies between this study and others, with our report of higher risks for Blacks and US-born Hispanics for some transitions, suggest that more attention should be paid to the course of AUDs, and that differences in prevalence should not be assumed to be equivalent to differences in course.

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Table 1

Prevalence at Waves 1 and 2 of DSM-IV alcohol abuse, alcohol dependence and alcohol use disorder by ethnicity. (weighted percentages with actual samples sizes)

Ethnicity	Unaffected	Alcohol Abuse	Alcohol Dependence	AUD [b]
			Wave 1 12-month diagnosis	
White	90.4% (n=15774)	5.6% (n=1007)	4.0%(n=677)	9.6% (n=1684)
Black	91.2% (n=4611)	4.3% (n=195)	4.5% (n=189)	8.8% (n=384)
Hispanic US-born	85.7% (n=2463)	6.9% (n=176)	7.4% (n=171)	14.3% (n=347)
Hispanic non-US-born	93.7% (n=2266)	3.1% (n=63)	3.2% (n=60)	6.3% (n=123)
			Wave 1 lifetime diagnosis	
White	61.7% (n=10727)	23.0% (n=4079)	15.3% (n=2652)	38.3% (n=6731)
Black	73.2% (n=3724)	15.9% (n=766)	10.9% (n=505)	26.8% (n=1271)
Hispanic US-born	63.5% (n=1834)	19.5% (n=559)	17.0% (n=417)	36.5% (n=976)
Hispanic non-US-born	81.9% (n=1999)	10.7% (n=249)	7.4% (n=141)	18.1% (n=390)
			Wave 2 interval diagnosis	
White	85.5% (n=14996)	8.4% (n=1443)	6.1% (n=1019)	14.5% (n=2462)
Black	86.3% (n=4405)	6.2% (n=275)	7.5% (n=315)	13.7% (n=590)
Hispanic US-born	82.0% (n=2334)	7.8% (n=217)	10.2% (n=259)	18.0% (n=476)
Hispanic non-US-born	90.7% (n=2204)	3.4% (n=78)	5.9% (n=107)	9.3% (n= 185)
			Wave 2 lifetime diagnosis [a]	
White	57.2% (n=9991)	24.6% (n=4338)	18.2% (n=3129)	42.8% (n=7467)
Black	66.8% (n=3448)	17.3% (n=838)	15.9% (n=709)	33.2% (n=1547)
Hispanic US-born	57.4% (n=1663)	19.8% (n=586)	22.8% (n=561)	42.6% (n=1147)
Hispanic non-US-born	77.4% (n=1904)	11.2% (n=269)	11.4% (n=216)	22.6% (n=485)

[a] Wave 2 lifetime diagnosis calculated by adding Wave 2 interval onsets to the Wave 1 lifetime diagnosis;

[b] AUD=Alcohol Abuse + Alcohol Dependence

Table 2

AUD and AD alcohol transitions by ethnicity, age, and gender: actual number of eligible respondents and weighted percentage of eligible respondents with AUD/AD at Wave 2.

		MEN			WOMEN		
Ethnicity	**Age Group**	**AUD onset**	**AUD persistence**	**AUD recurrence**	**AUD onset**	**AUD persistence**	**AUD recurrence**
White	18–29	23.6% n=685	67.3% n=363	29.8% n=295	12.8% n=1142	52.3% n=233	27.6% n=331
	30–39	12.6% n=656	58.5% n=257	20.4% n=613	5.8% n=1205	50.0% n=164	14.3% n=565
	40+	6.8% n=2568	59.1% n=471	13.3% n=2099	1.9% n=4471	42.2% n=196	7.8% n=1144
Black	18–29	21.1% n=250	63.0% n=62	37.8% n=35	10.5% n=559	33.0% n=49	15.4% n=41
	30–39	12.4% n=226	58.6% n=54	20.8% n=109	7.1% n=599	46.5% n=50	18.9% n=77
	40+	7.2% n=641	51.4% n=115	11.5% n=412	3.4% n=1449	53.3% n=54	8.2% n=213
Hispanic US-born	18–29	23.3% n=242	61.0% n=113	35.1% n=50	7.3% n=391	41.0% n=69	25.5% n=58
	30–39	10.3% n=128	46.0% n=57	16.7% n=107	4.9% n=328	75.7% n=25	23.4% n=89
	40+	10.7% n=283	64.3% n=60	10.9% n=213	2.5% n=462	52.3% n=23	9.1% n=112
Hispanic non-US-born	18–29	17.4% n=215	45.8% n=38	21.5% n=32	2.4% n=218	68.4% n=15	0% n=6
	30–39	6.9% n=258	54.8% n=32	18.3% n=69	0.9% n=334	44.6% n=3[a]	4.0% n=18
	40+	1.9% n=463	42.4% n=30	10.1% n=122	1.8% n=511	16.3% n=5[a]	9.7% n=20
		AD onset	**AD persistence**	**AD recurrence**	**AD onset**	**AD persistence**	**AD recurrence**
White	18–29	10.4% n=957	48.3% n=212	22.0% n=174	7.5% n=1405	42.8% n=109	16.2% n=192
	30–39	4.8% n=1142	55.5% n=91	11.0% n=293	2.4% n=1623	40.3% n=61	12.2% n=250
	40+	2.4% n=4297	53.9% n=133	9.2% n=708	1.2% n=5382	40.4% n=71	6.6% n=358
Black	18–29	15.0% n=299	31.4% n=34	26.7% n=14	6.2% n=605	25.3% n=29	3.3% n=15
	30–39	7.3% n=322	61.5% n=20	12.1% n=47	5.0% n=669	41.1% n=31	10.0% n=26
	40+	4.3% n=985	42.9% n=48	9.7% n=135	2.1% n=1610	64.9% n=27	5.8% n=79
Hispanic US-born	18–29	18.9% n=313	36.4% n=72	37.8% n=20	4.3% n=442	40.5% n=37	19.7% n=39
	30–39	4.3% n=237	41.8% n=16	8.5% n=39	5.3% n=393	50.9% n=13	25.4% n=36
	40+	5.4% n=471	56.2% n=19	8.9% n=66	2.5% n=537	47.9% n=14	1.3% n=46
Hispanic non-US-born	18–29	13.3% n=254	8.4% n=16	24.5% n=15	0.6% n=225	53.5% n=11	0% n=3 [a]
	30–39	6.9% n=317	28.4% n=20	14.3% n=22	0.4% n=349	0% n=1 [a]	0% n=5 [a]
	40+	1.5% n=572	38.1% n=11	25.3% n=32	0.5% n=531	100% n=1 [a]	49.3% n=4 [a]

Note. The base sample for these analyses was n=27652, which included all White, Black, and Hispanic respondents with Wave 2 data who were not lifetime abstainers at Wave 1; AUD=alcohol use disorder; AD=alcohol dependence. Participants were eligible for AUD onset if they had no lifetime AA or AD diagnosis at Wave 1, for AUD persistence if they had AA or AD in the 12-months preceding Wave 1, for AUD recurrence if they had no 12-month diagnosis at Wave 1 but had a prior AA or AD diagnosis, for AD onset if they had no lifetime AD diagnosis at Wave 1, AD persistence if they had AD in the 12-months preceding Wave 1, and AD recurrence if they had no 12-month AD diagnosis but had a prior AD diagnosis.

[a] indicates actual sample sizes that were deemed too small for inclusion in logistic regression analyses (n≤5)

Table 3

Odds ratios from logistic regression analyses testing *ethnic and age differences in AUD transition risk among NESARC men*

Variable	Onset	Persistence	Recurrence
Age			
30–39 years[a]	0.48* (0.41 – 0.56)	0.70* (0.61 – 0.81)	0.60* (0.49 – 0.72)
40 plus years[a]	0.24* (0.21 – 0.27)	0.69* (0.60 – 0.81)	0.36* (0.30 – 0.42)
Black [b]	0.95 (0.79 – 1.13)	0.82* (0.73 – 0.93)	n/a
Black × 18–29 yrs	n/a	n/a	1.41* (1.10 – 1.81)
Black × 30–39 yrs	n/a	n/a	1.02 (0.74 – 1.41)
Black × 40 plus yrs	n/a	n/a	0.85* (0.72 – 0.99)
Hispanic US-born [b]	n/a	n/a	0.89 (0.73 – 1.09)
Hisp US × 18–29 yrs	1.00 (0.87 – 1.16)	0.76* (0.62 – 0.93)	n/a
Hisp US × 30–39 yrs	0.79* (0.65 – 0.96)	0.59* (0.44 – 0.79)	n/a
Hisp US × 40 plus yrs	1.64* (1.18 – 2.28)	1.26* (1.03 – 1.55)	n/a
Hispanic non-US-born [b]	n/a	n/a	0.74* (0.58 – 0.95)
Hisp non-US × 18–29 yrs	0.69* (0.62 – 0.78)	0.41* (0.33 – 0.52)	n/a
Hisp non-US × 30–39 yrs	0.51* (0.40 – 0.65)	0.84* (0.71 – 0.99)	n/a
Hisp non-US × 40 plus yrs	0.27* (0.24 – 0.31)	0.52* (0.45 – 0.59)	n/a

Note.

[a] comparison group is White 18–29 year-olds;

[b] for onset and persistence there was no age × Black interaction; for recurrence there was no age by ethnicity interaction for Hispanic US-born and Hispanic non-US-born; other age × ethnicity interactions were significant

* $p < .05$

Table 4

Odds ratios from logistic regression analyses testing *ethnic and age differences in AUD transition risk among NESARC women*

Variable	Onset	Persistence	Recurrence
Age			
30–39 years [a]	0.43* (0.36 – 0.51)	0.91 (0.75 – 1.11)	0.44* (0.36 – 0.53)
40–49 years [a]	0.13* (0.12 – 0.15)	0.67* (0.56 – 0.79)	0.22* (0.19 – 0.26)
Black × 18–29 yrs	0.81* (0.71 – 0.91)	0.45* (0.38 – 0.54)	0.48* (0.27 – 0.86)
Black × 30–39 yrs	1.22* (1.04 – 1.43)	0.87 (0.75 – 1.01)	1.39* (1.04 – 1.86)
Black × 40 plus yrs	1.83* (1.61 – 2.09)	1.57* (1.24 – 1.98)	1.06 (0.81 – 1.39)
Hisp US × 18–29 yrs	0.54* (0.45 – 0.64)	0.63* (0.55 – 0.73)	0.90 (0.54 – 1.48)
Hisp US × 30–39 yrs	0.84 (0.64 – 1.10)	3.12* (2.47 – 3.93)	1.83* (1.34 – 2.51)
Hisp US × 40 plus yrs	1.31* (1.13 – 1.51)	1.50* (1.22 – 1.85)	1.19* (1.00 – 1.41)
Hisp non-US × 18–29 yrs	0.17* (0.15 – 0.18)	1.97* (1.39 – 2.79)	---[b]
Hisp non-US × 30–39 yrs	0.15* (0.13 – 0.17)	---[c]	0.25* (0.22 – 0.28)
Hisp non-US × 40 plus yrs	0.94 (0.87 – 1.02)	---[c]	1.27* (1.13 – 1.44)

Note.

[a] comparison group is 18–29 year-old Whites;

[b] inestimable;

[c] not modeled due to low n (see Table 2)

* $p < .05$

Table 5

Odds ratios from logistic regression analyses testing *ethnic and age differences in AD transition risk among NESARC men*

Variable	Onset	Persistence	Recurrence
Age			
30–39 years [a]	0.44* (0.37 – 0.52)	1.33* (1.04 – 1.71)	0.44* (0.35 – 0.57)
40 plus years [a]	0.22* (0.19 – 0.26)	1.25* (1.05 – 1.48)	0.38* (0.30 – 0.47)
Black [b]	1.61* (1.40 – 1.84)	n/a	1.11 (0.92 – 1.35)
Black × 18–29 yrs	n/a	0.49* (0.42 – 0.57)	n/a
Black × 30–39 yrs	n/a	1.28 (0.56 – 2.96)	n/a
Black × 40 plus yrs	n/a	0.64* (0.50 – 0.83)	n/a
Hisp US × 18–29 yrs	2.03* (1.74 – 2.36)	0.61* (0.48 – 0.77)	2.21 (0.98 – 5.02)
Hisp US × 30–39 yrs	0.89 (0.74 – 1.07)	0.58* (0.38 – 0.87)	0.76* (0.61 – 0.96)
Hisp US × 40 plus yrs	2.27* (1.66 – 3.12)	1.10 (0.79 – 1.52)	0.94 (0.38 – 2.37)
Hispanic non-US-born [b]	n/a	n/a	1.80* (1.16 – 2.77)
Hisp non-US × 18–29 yrs	1.33* (1.17 – 1.52)	0.10* (0.09 – 0.11)	n/a
Hisp non-US × 30–39 yrs	1.46* (1.13 – 1.89)	0.32* (0.24 – 0.42)	n/a
Hisp non-US × 40 plus yrs	0.58* (0.50 – 0.68)	0.53* (0.43 – 0.64)	n/a

Note.

[a] comparison group is White 18–29 year-olds;

[b] for onset and recurrence there was no age × Black interaction; for recurrence there was no age × Hispanic non-US-born interaction; other age × ethnicity interactions were significant

* $p < .05$

Table 6

Odds ratios from logistic regression analyses testing *ethnic and age differences in AD transition risk among NESARC women*

Variable	Onset	Persistence	Recurrence
Age			
30–39 years [a]	0.30* (0.24 – 0.37)	0.90 (0.74 – 1.10)	0.74* (0.60 – 0.91)
40 plus years [a]	0.15* (0.13 – 0.17)	0.91 (0.70 – 1.17)	0.38* (0.32 – 0.45)
Black [b]	n/a	n/a	0.67 (0.44 – 1.04)
Black × 18–29 yrs	0.81* (0.68 – 0.97)	0.45* (0.40 – 0.52)	n/a
Black × 30–39 yrs	2.16* (1.76 – 2.65)	1.03 (0.91 – 1.17)	n/a
Black × 40 plus years	1.71* (1.47 – 1.98)	2.73* (1.78 – 4.18)	n/a
Hisp US × 18–29 yrs	0.55* (0.47 – 0.65)	0.91 (0.80 – 1.03)	1.29 (0.44 – 3.84)
Hisp US × 30–39 yrs	2.32* (1.90 – 2.83)	1.53* (1.35 – 1.74)	2.43* (1.84 – 3.22)
Hisp US × 40 plus yrs	2.08* (1.63 – 2.66)	1.36* (1.10 – 1.68)	0.18* (0.04 – 0.76)
Hisp non-US × 18–29 yrs	0.08* (0.07 – 0.09)	1.54* (1.08 – 2.20)	--- [c]
Hisp non-US × 30–39 yrs	0.17* (0.14 – 0.19)	--- [c]	--- [c]
Hisp non-US × 40 plus yrs	0.45* (0.38 – 0.52)	--- [c]	--- [c]

Note.

[a] comparison group is 18–29 year-old Whites;

[b] for recurrence there was no age × ethnicity interaction for Blacks; other age × ethnicity interactions were significant;

[c] not modeled due to low n (see Table 2)

* $p < .05$