HHS Public Access

Author manuscript

Am J Addict. Author manuscript; available in PMC 2017 August 01.

Published in final edited form as:

Am J Addict. 2017 August ; 26(5): 437–445. doi:10.1111/ajad.12462.

Differences Between White and Black Young Women in the Relationship Between Religious Service Attendance and Alcohol Involvement

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Abstract

Background and Objectives—We examined the associations of religious attendance during childhood (C-RA) and adulthood (A-RA) with alcohol involvement (ever drinking, timing of first alcohol use, and alcohol use disorder [AUD]) in White and Black female twins. As genetic and environmental factors influence religious attendance and alcohol involvement, we examined the extent to which they contribute to their association.

Methods—Data on 3,234 White and 553 Black female twins (18–29 years) from the Missouri Adolescent Female twin Study. Significant correlations between C-RA or A-RA and alcohol involvement were parsed into their additive genetic, shared environmental, and individual-specific environmental sources.

Results—C-RA was associated with ever drinking and timing of first alcohol use in Whites. A-RA was associated with ever drinking and AUD in both Whites and Blacks. Shared environmental influences did not contribute to alcohol or religiosity phenotypes in Blacks. In Whites, the association between C-RA and alcohol was due to shared environmental influences, whereas the association between A-RA and alcohol was attributable to additive genetic, shared environmental, and individual-specific environmental sources. Individual-specific environment and genetics contributed to associations between A-RA and ever drinking and AUD, respectively, in Blacks.

Conclusions—Factors other than C-RA contribute to lower rates of alcohol involvement in Blacks. Shared environment does not contribute to links between A-RA and alcohol involvement in Blacks.

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Declaration of Interest

Other authors have no conflicts to report.

Scientific Significance—The protective impact of childhood religiosity on alcohol use and misuse is important in Whites and is due to familial factors shared by religiosity and alcohol involvement.

INTRODUCTION

Involvement in pro-social activities, particularly religious services during adolescence, has been robustly identified as a deterrent to onset of substance use, particularly alcohol.[1–11] The first effects of religiosity on substance use are likely to be embedded in early religious behaviors, typically during childhood and adolescence, and often involving familial practices. However, religious attendance tends to decay into adulthood, becoming more related to personal devotion.[5] The role of religious involvement in the etiology of alcohol use and misuse is particularly intriguing. This influence may involve specific proscriptions against alcohol use in certain religious denominations (eg, Seventh Day Adventists) or it may be generally protective against deviant behavior through prosocial community level involvement. The associations between religiosity, both childhood and adult, and alcohol-related problems are also not well understood, even though self-help groups such as Alcoholics Anonymous incorporate elements of spiritual belief into their therapeutic framework.[12]

In the United States, Black youth, particularly females,[13] are more likely to report increased religiosity, including greater involvement in religious activities, than White youth.[3,6] Seventy-nine percent of Blacks, compared with 56% of the U.S. population, report that religion is very important in their lives.[13] Furthermore, there are several distinct cultural and community practices that distinguish and enhance the prominence of religiosity and religious service attendance in Blacks[14,15] and these behaviors might exert protective influences on other outcomes, such as alcohol use and misuse. Alcohol use and use disorders (AUD) are more common in White versus Black youth.[16] Despite the greater prominence of religiosity in Black youth, the protective effect of religious involvement on alcohol use is markedly more evident in White youth with some studies finding weak to no association in Black youth.[3,6,8] Thus, even though protective influences are at play in Black youth, whether religiosity serves as one such factor is unclear. Furthermore, even though Whites are more likely than Blacks to use alcohol at an early age and to progress to AUD, the negative social and health outcomes associated with alcohol misuse are more pronounced in Blacks and other minority groups,[17] who also tend to be less likely to access alcohol treatment services.[18] Therefore, identifying factors that contribute to differences in onset of drinking and progression to AUD in Whites and Blacks, and elucidating the pathways by which they contribute to their association with alcohol involvement, is essential if bridging ethnic disparities is to be a priority.

The pathways that lead to associations between religious attendance and alcohol involvement may involve shared familial factors (eg, genetic attributes or shared environment that predisposes youth to prosocial behaviors) as well as individual-specific environmental factors that influence both religiosity and alcohol involvement (eg, an experience of trauma that leads to decreased religiosity as well as increased alcohol use). Twin studies offer an ideal approach for disentangling the role of genetic and environmental

influences on the covariance between religious service attendance and alcohol involvement. These studies suggest that genetic and shared environmental factors contribute to familial sources of resemblance in earlier and non-problem stages of alcohol use (eg, regular drinking), while genetic factors are more distinctly important for problem drinking.[19] On the other hand, the role of shared environment has been more frequently implicated in studies of religiosity (variously defined, eg, ref.[4,20–22]), even though its role declines from adolescence to adulthood, being replaced by genetic factors.[23,24] Two studies explored whether religiosity modified the extent to which genetic factors influence alcohol use. Koopmans et al.[25] reported that while religiosity was not associated with alcohol initiation, heritable influences on alcohol initiation were absent in adolescent females who were religiously raised and as high as 40% in those who were not religiously raised. Similarly, Button et al.[26] found that the heritability of problem alcohol use at age 12–18 was inversely related to adolescent religious involvement but no such effects on the heritability of problem drinking at age 17–29 were noted. In contrast, four studies have studied the bivariate association between alcohol involvement and religiosity. Harden[27] noted that religious adolescents did not differ from their non-religious siblings on timing of initiation of alcohol use, indicating the role of familial influences that siblings share (genes and environment) on this association. Vance et al.[28] found that associations between alcohol dependence and 6 of 7 dimensions of adult religiosity were attributable to overlapping genetic factors alone (rg −.18 to −.32). Kendler and Myers[23] reported that negative correlations between church attendance and alcohol use during adolescence are modest (r −.22 at ages 12–14) and attributable to familial environment. By adulthood, the correlation was stronger (r −.47) and more significantly attributable to additive genetic (62%) influences. Finally, Maes et al.[29] used an extended kinship design to demonstrate that while genetic factors contributed to the relationship between church attendance and alcohol use, the most critical contributor to the association in females was genotype-environment covariance (ie, correlations between parental and offspring behaviors via genetic and cultural pathways). These studies indicate a complex relationship between dimensions of religiosity measured during childhood and adulthood and stages of alcohol involvement; however, none of these studies were based on ethnically diverse samples. In order to more systematically address potential sources of alcohol-related disparities across Whites and Blacks, a comparison of patterns of genetic and environmental influence on alcohol involvement, religious service attendance, and their correlation is needed in ethnically diverse twin samples.

The goal of the present study was to explore the relationship between religious service attendance, retrospectively reported for childhood (C-RA; for ages 6–13 years) and currently, during adulthood (A-RA; current age 18–29 years) with three aspects of alcohol involvement (ever drinking, timing of first alcohol use and AUD) in 3,234 White and 553 Black female twins. First, we examined whether levels of C-RA and A-RA were associated with alcohol involvement measures across White and Black women. We hypothesized that (a) C-RA would be associated with ever drinking and timing of first alcohol use, but not AUD, and that A-RA would be associated with AUD and that (b) both associations would be stronger in White than Black women. Second, for those measures of alcohol involvement that were significantly correlated with C-RA and A-RA, univariate and bivariate twin models were fitted to raw data to decompose the variance in and covariance between alcohol

involvement and C-RA as well as A-RA into genetic and environmental sources. We hypothesized that family environment would be a major contributor to this association in Whites.

METHODS

Sample

The Missouri Adolescent Female Twin Study (MOAFTS)[30,31] is a population-based longitudinal study of female twin pairs born between July 1, 1975 and June 30, 1985 in Missouri to Missouri-resident parents. The sample was demographically representative of the Missouri population at the time the twins were born, with nearly 15% of twins being Black and the remainder being of White descent. A baseline interview was conducted with 3,258 twins beginning in 1995 (median age = 15 years). Between 2002 and 2005, all twins from the target cohort (excluding those who had withdrawn from the study or whose parents asked that the family not be recontacted) were contacted for Wave 4 interviews (N = 3,787). Wave 5 interviews were conducted between 2005 and 2008 with 91% of the Wave 4 participants.

Because Wave 4 interviews yielded the largest sample size and covered lifetime psychiatric and psychosocial information in young adulthood, we limited the sample to MOAFTS participants who completed Wave 4 interviews. However, data from baseline, Wave 3, and 5, which were available for over 95% of Wave 4 participants, were integrated into coding ever drinking, timing of first alcohol use and AUD diagnosis; in cases where first alcohol consumption or AUD criteria were endorsed prior to Wave 4, we used the age at onset reported in the interview in which it was first endorsed. The Wave 4 sample consisted of 1,038 monozygotic (MZ) twin pairs, 735 dizygotic (DZ) twin pairs, and 241 twins whose co-twins did not participate.

Measures

Religious Service Attendance—Childhood religious service attendance (C-RA) was assessed (in Wave 4 only) by asking, “How often did you attend religious services between the ages of 6 and 13?” Participants endorsed 1 of 6 possible responses, “more than once a week,” “once a week,” “a few times a month,” “once or twice a month,” “less than once a month,” or “never.” Religious service in adulthood (A-RA) was assessed (in Wave 4 only) by asking, “In the past 12 months, how often did you attend religious services?” using the same list of possible responses. A-RA and C-RA were modeled as weekly or more and greater than weekly religious service attendance, respectively. The different cut-offs reflected differences in the distribution of RA during childhood and adulthood.

Ever Drinking—Ever drinking was defined as consumption of at least one full standard alcoholic drink (with definition provided by the interviewer) at any point during the lifetime.

Timing of First Alcohol Use—Timing of first alcohol use was defined as age at consumption of first full standard alcoholic drink. Ages at onset were structured as a categorical variable of 0 = onset prior to age 16, 1 = onset age 16–17, and 2 = onset

subsequent to age 17. These age cutoffs resulted in roughly equivalently sized groups for White (but not Black) twins and satisfied assumptions of multivariate normality ($p > .05$).

Alcohol Use Disorder (AUD)—AUD was determined using a modified DSM-5 diagnosis: ≥2 of 10 criteria (seven DSM-IV dependence and three DSM-IV abuse symptoms, no craving), occurring in the same 12-month period (self-reported or based on ages of onset and recency of individual criteria).

Data Analysis

Data were processed in SAS v9. Binary and multinomial logistic regression was conducted in STATA to examine the association between each alcohol measure (dependent variables: ever drinking, timing of first alcohol use, and AUD) and religious service attendance (independent variables: C-RA and A-RA). A robust variance estimator was used to account for familial clustering.

Univariate twin models were fitted to raw data on ever drinking, timing of first alcohol use, AUD, C-RA, and A-RA to attribute variance in these measures to additive genetic (A), shared/familial environmental (C), and individual-specific environmental (E) sources.[32] When the alcohol and religious attendance variables were significantly correlated, this correlation was also decomposed into its genetic and environmental sources using bivariate twin models. Additive genetic (A) influences reflect the additive effect of all genetic loci shared identical-by-descent. Shared environment (C) reflects those factors that make members of twin pairs similar to each other (eg, similar family practices), while individual-specific environment (E) refers to those factors that are unique to each member of the twin pair (eg, individual trauma exposure) as well as measurement error. Evidence for A is derived from the MZ correlation (rMZ) being larger than the DZ (rDZ) correlation. Evidence for C arises from rDZ > ½ rMZ, while support for E is based on rMZ < 1. All our results are based on formal model-fitting procedures where raw data were fitted using full information maximum likelihood estimation in the statistical software package, Mx.[33] Thresholds were adjusted for age at Wave 4 (binary, age ≥22; median split). Analyses were conducted in White and Black twins separately; where there were effects in both White and Black twins, parameters across racial/ethnic groups were compared using multi-group post hoc tests that examined whether constraining parameters across racial/ethnic groups resulted in a change in model fit.

RESULTS

Sample Characteristics

The distribution of C-RA, A-RA, and alcohol involvement, across race/ethnicity, is presented in Table 1. Overall, 62.1% and 71.3% of Whites and Blacks reported weekly or greater C-RA, respectively. The frequency of RA declined into adulthood with only 26.2% of Whites and 34.9% of Blacks reporting similar levels of A-RA as C-RA. The most commonly endorsed religious affiliations were Baptist, Roman Catholic, Other Protestant, and None and most participants reported the same affiliation during childhood and adulthood. Of the Whites, 5.5% had never had a drink of alcohol. Of those who had ever had

a drink, 41.0% reported an age at first drink prior to age 16 and 31.1% met criteria for modified DSM-5 AUD. Of the Blacks, 13.2% reported no lifetime history of ever drinking. Of those who had ever had a drink, 26.6% had started drinking prior to age 16 and 16.8% met criteria for a lifetime history of modified DSM-5 AUD. C-RA and A-RA were moderately correlated in White (r =.49 [.45, .53]) and Black (r = .35 [.25, .46]) females.

Association Between Religious Service Attendance and Alcohol Involvement

Whites—Increasing levels of religious service attendance was associated with a decreased likelihood of alcohol involvement (Table 2). Those who attended religious services weekly or more often during childhood (C-RA) were at .16–.46 lower odds of ever drinking alcohol and, if they did drink, were also less likely to start drinking prior to age 18. Likewise, A-RA was associated with .04–.19 lower odds of ever drinking, and with a reduced likelihood of first alcohol use prior to age 18. In addition A-RA was associated with .29–.43 lower odds of DSM-5 AUD.

Blacks—Despite the higher rates of attendance and lower rates of alcohol involvement, associations were less pronounced. There was no relationship between C-RA and any measure of alcohol involvement in Black females. However, A-RA was associated with lower likelihood of ever drinking and of meeting criteria for DSM-5 AUD.

Genetic and Environmental Influences

MZ and DZ pair correlations in Table 3 indicated the role of individual-specific environment (E) on all measures (rMZ < 1.0). MZ correlations were greater than DZ correlations suggesting the role of additive genetic (A) influences. However, shared environmental factors (C) featured prominently as well, as DZ correlations were consistently greater than half the MZ correlation, particularly in Whites.

Estimates for A, C, and E from the full univariate models are shown in Fig. 1 and in Table 3 (fit indices are available upon request).

Whites—Variance in C-RA was attributable to A (23%), C (55%), and E (22%). A (28%), C (54%), and E (18%) contributed to variance in A-RA. A (32–55%), C (17–55%), and E (13–41%) contributed to variance in alcohol involvement (Fig. 1).

Blacks—For C-RA, A-RA and alcohol involvement, A (44–71%) and E (25–45%) made the most substantial contributions to variance, with only modest evidence for C (Fig. 1).

Sources of Covariance Between Religious Attendance and Alcohol Involvement

Based on significant phenotypic relationships that were identified in Table 2, the following bivariate relationships between C-RA, A-RA, and alcohol involvement were explored (see Table 4 for cross-trait correlations due to A, C, and E; additional details regarding fit indices are available upon request):

a) C-RA and ever drinking in Whites: this relationship was solely attributable to C, which were negatively and strongly correlated (r_C = −.72; Table 4).

b) C-RA and timing of first alcohol use in Whites: this relationship was solely attributable to C, with higher C-RA associated with later first use of alcohol (r_C = .42; Table 4). The upper confidence limit on the correlation of C was 1.0, indicating that all shared environmental influences on timing were correlated with those influencing C-RA.

c) A-RA and ever drinking in Whites and Blacks: in Whites, C factors were potentially completely overlapping and responsible for 83% of the covariance. E factors explained 17% of the covariance in Whites and 100% of the covariance in Blacks.

d) A-RA and timing of first alcohol use in Whites: this correlation was primarily attributable to A factors (r_A = .75) with little evidence for specific genetic influences on timing.

e) A-RA and DSM5-AUD in Whites and Blacks: variance components in this model could be completely equated across ethnicity, and the relationship was attributable to A factors that were negatively correlated (r_A = −.61).

DISCUSSION

Our study confirms prior research that has documented higher rates of religious service attendance during childhood (C-RA) and adulthood (A-RA)[5,34] and lower rates of alcohol involvement in Blacks compared to Whites.[16,35] Across racial/ethnic groups, we noted a decline in overall RA and this change was more precipitous for Blacks (ie, dropping from 35% to 15% for RA on a greater than weekly basis). Consistent with the literature, we also noted that ever drinking and AUD were less common in Blacks who also were less likely than Whites to start drinking prior to age 16.[17] Despite these trends, and also consistent with other studies and our initial hypotheses,[6] while C-RA was associated with lower likelihood of alcohol involvement in Whites, it was not correlated with any level of alcohol involvement in Blacks. Thus, other childhood protective influences are likely to act as deterrents to onset of drinking, particularly at an early age, in Black youth. However, more large-scale studies that gather data on childhood protective factors for alcohol use that might be of particular salience in Blacks are needed.

On the other hand, increased A-RA was associated, to a similar degree, with lower odds of DSM-5 AUD in Blacks and Whites. As our data are cross-sectional, we cannot be certain that this association reflects a causal effect of increased A-RA on reduced progression to AUD, however, our results suggest that adult religious involvement, which might more closely approximate personal religious devotion rather than adherence to family norms during childhood, may be better a indicator of the protective influence of religion on alcohol problems in Blacks.

The extent to which genetic and environmental factors contribute to these correlations between religious service attendance and alcohol involvement can have varying implications for etiology, prevention, and, importantly, for identifying potential pathways that result in ethnic disparities. For instance, in Whites, the association between C-RA and ever drinking as well as timing of first alcohol use was primarily attributable to shared environmental

factors. This finding is consistent with other studies[23,27] and suggests that potential religiosity-related interventions that delay the onset of alcohol use should be targeted at the family-level. In contrast, the association between A-RA and AUD was attributable to additive genetic influences (as in another study[28]) indicating that predispositions that result in prosocial behaviors, such as recent religious service attendance may also influence reduced progression to AUD, and could also include genetic liability to personality traits, such as conservatism.[36] The prominent role of shared environment in the associations between C-RA and ever drinking as well as timing of first alcohol use should also be viewed in light of findings by Maes et al.[29] that identified cultural transmission and genotype-environment covariance to be the major contributor to this relationship in females. In other words, cultural values regarding religious service attendance and alcohol initiation are co-transmitted via correlated genetic and environmental pathways from parents to offspring. For instance, parents who do not drink alcohol are likely to pass this genetic predisposition on to their offspring. In addition, these parents may be more likely to attend religious services and to encourage their offspring to attend, thus producing a further protective environment in which inherited protections against drinking can be manifested.

The present study is among the first to examine the extent to which genetic and environmental factors contribute to religious service attendance and alcohol involvement in Blacks. Two findings are noteworthy. First, unlike in the White twins, shared environment contributed minimally to individual differences in C-RA, A-RA, ever drinking, timing of first alcohol use, and AUD in Blacks. This supports the idea that adult indices of religious service attendance might reflect the more heritable aspects of religiosity, such as personal devotion,[5,20] and might relate to alcohol use via shared inherited attributes, such as personality. Second, only A-RA was associated with alcohol involvement in Blacks. Similar to the White twins, the relationship with AUD was due to genetic factors, but the relationship between A-RA and ever drinking was entirely due to individual-specific factors (in contrast to shared and individual-specific environmental factors in White twins). Unlike the negative correlations between C-RA and ever drinking, which likely reflect the role of religious involvement as a deterrent to drinking onset, the correlation between A-RA and ever drinking might be indicative of the higher prevalence of lifetime alcohol abstinence in religiously involved adults. Therefore, despite both A-RA and ever drinking being highly heritable in Blacks, their correlation is likely due to individual-specific factors that contribute to both outcomes, such as affiliation with non-deviant peers involved in prosocial activities. Some caution is required in the interpretation of the role of individual-specific environment, however, as it does include estimates of measurement error.

A few limitations of the current study are noteworthy. First, we assessed only one facet of religiosity—religious service attendance—which represents a combination of community level involvement and strength of religious beliefs. While we did have data on other aspects of personal spirituality, these measures were not retrospectively reported during childhood, a period critical to the onset of alcohol use. Second, as our measures of C-RA, A-RA, and alcohol involvement were collected contemporaneously, these cross-sectional data do not allow us to make causal assertions. Third, we did not have data on male twins and, given that findings differ in males,[29] our findings may not generalize across gender. Fourth, we did not examine whether RA moderated the extent of heritable influences on alcohol involvement

(similar to[25,26]), so we cannot draw conclusions regarding whether the heritability of alcohol involvement varied as a function of C-RA or A-RA. Fifth, our relatively small sample size of Black twins may have restricted our power to significantly detect certain correlations and variance components. For instance, C-RA is associated with a lower likelihood of ever drinking as well as AUD in Blacks (OR = .61–.69) but these estimates do not achieve statistical significance. Nonetheless, we note that this is currently the largest cohort of Black twins available. Finally, as Black females tend to have a later age at onset of AUD,[37] they may not have been past the age of vulnerability during the course of the study.

Our findings suggest that etiologically distinct mechanisms underlie the association between religious service attendance and alcohol involvement in Black and White women. In Blacks, the association with ever drinking is due to individual-specific factors that might be modifiable. On the other hand, the association between A-RA and AUD, in both Blacks and Whites was due to common genetic influences, which are less amenable to prevention and intervention efforts. Given noted ethnic disparities in the consequences of AUD in Blacks, isolating modifiable risk and protective factors that robustly relate to drinking milestones and afford protection against the development of problem drinking is needed, particularly in genetically informative datasets.

Acknowledgments

This study was funded by grants DA032573 (AA) from the National Institute on Drug Abuse, grants AA021235 (AA), AA017921 (CS), AA023549 (CS), AA12640 (KKB), AA017688 (ACH), and AA016383 (JRH) from the National Institute on Alcohol Abuse and Alcoholism, grant HD049024 (ACH) from the National Institute of Child Health and Human Development, and a grant from the Robert E. Leet and Clara Guthrie Patterson Trust.

There are no contractual constraints on publishing the research being reported. Funding sources were not involved in any aspect of this submission.

Unrelated to this study, AA previously received peer-reviewed funding from ABMRF/Foundation for Alcohol Research (completed 12/2012) which receives funding from the brewing industry.

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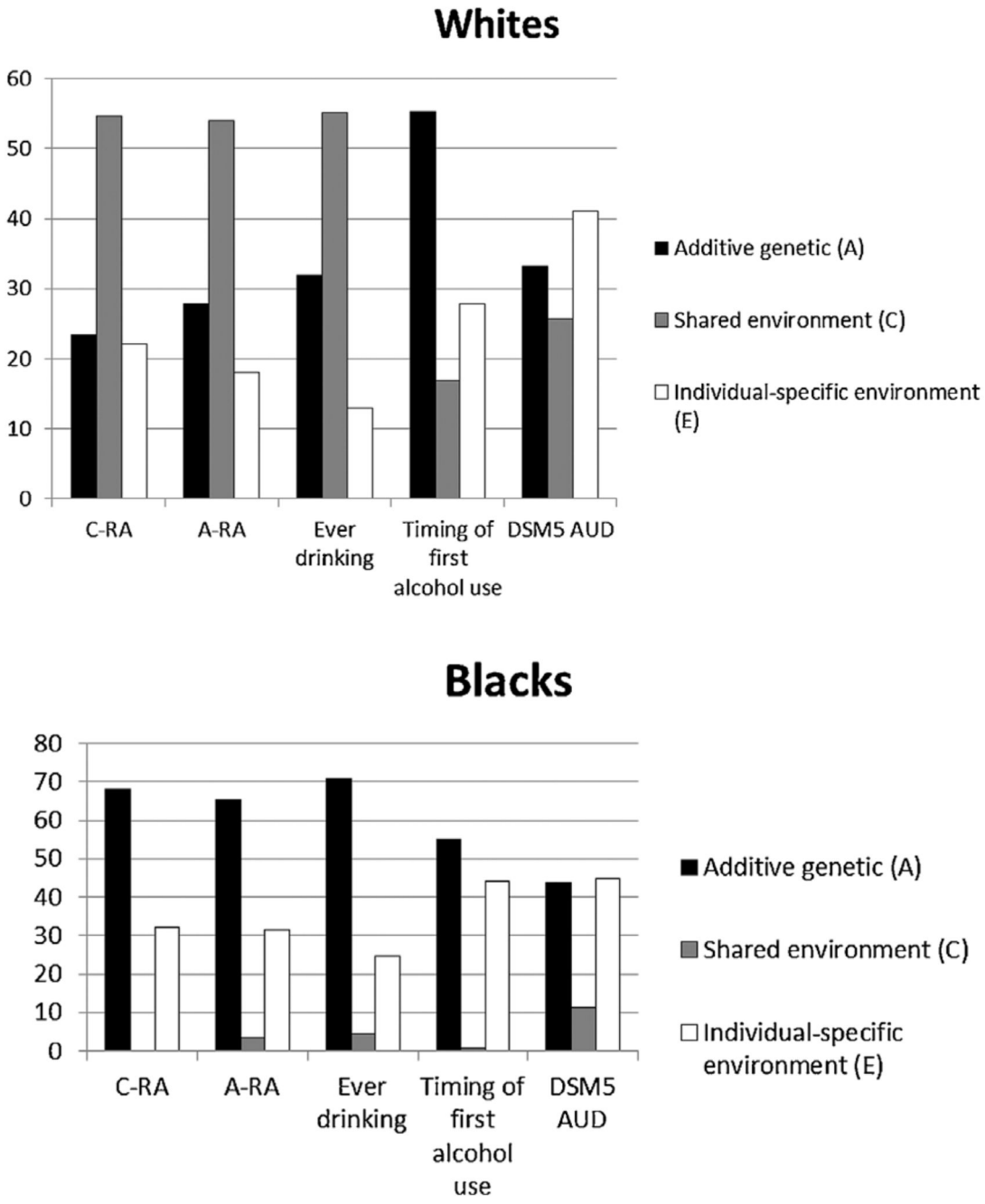

FIGURE 1.

Univariate estimates (from the full model) of the contributions of additive genetic (A), shared environmental (C), and individual-specific environmental (E) influences on religious service attendance during childhood (C-RA) and adulthood/past-year (A-RA) as well as alcohol involvement in 1515 White and 254 Black twin pairs.

TABLE 1

Comparison of White and Black young adult women on childhood and past year church attendance and lifetime alcohol involvement in 3,787 twin females

	White	Black
Age at Wave 4 interview	*M* = 21.6 years [2.7]; 18–29	*M* = 22.0 year [2.8]; 18–28
Religious service attendance		
Childhood (C-RA): (between ages 6 and 13)		
Never *	5.6 [4.8, 6.4]	3.3 [1.8, 4.8]
Occasionally, less than weekly **	32.3 [30.7, 33.9]	25.4 [21.8, 29.0]
Weekly ns	40.4 ns [38.7, 42.0]	36.3 [32.3, 40.3]
More than weekly ***	21.7 [20.3, 23.1]	35.0 [31.0, 39.0]
Adulthood (A-RA): past 12 months, age 18–29)		
Never **	22.7 [21.3, 24.2]	17.3 [14.1, 20.4]
Occasionally, less than weekly ns	51.1 ns [49.4, 52.8]	47.8 [43.6, 52.0]
Weekly *	16.1 [14.8, 17.4]	20.0 [16.7, 23.3]
More than weekly **	10.1 [9.1, 11.1]	14.9 [11.9, 17.9]
Alcohol use and disorder		
Ever drank alcohol ***	94.5 [93.7, 95.3]	86.8 [84.0, 89.6]
Age at first drink	*M* = 16.1 years [2.6]; 5–27	*M* = 17.2 years [3.1]; 5–28
Timing of first alcohol use		
Before age 16 ***	41.0 [39.3, 42.8]	26.6 [22.7, 30.6]
Age 16–17 **	32.6 [30.9, 34.3]	26.0 [22.1, 30.0]
Age 18 or older ***	26.4 [24.8, 28.0]	47.4 [42.9, 51.9]
DSM5 alcohol use disorder *** among those who ever drank	31.1 [29.5–32.8]	16.8 [13.5, 20.2]

Significant differences between Whites and Blacks are indicated by:

ns = $p > .05$, not significant.

* $p < .05$;

** $p < .01$;

*** $p < .001$.

TABLE 2

Logistic and multinomial logistic regression analyses between alcohol involvement from childhood (C-RA) and adult past year (A-RA) religious attendance in White and Black young adult women

	Outcomes			
		Timing of first alcohol use[1,2] relative risk ratios		
Predictors	**Ever drinking odds ratios**	**15 or younger**	**16–17 years old[2]**	**DSM5 AUD[1] odds ratios**
Whites				
Childhood-religious service attendance (C-RA; ages 6–13)[3]				
Weekly	.46*,a [.28, .75]	.66*,a [.52, .82]	.85c [.67, 1.07]	.98 [.82, 1.18]
Greater than weekly	.16*,b [.10, .27]	.47*,b [.36, .62]	.64*,a [.49, .85]	.92 [.73, 1.16]
Adult-religious service attendance (A-RA; past 12 months, ages 18–29)[3]				
Weekly	.19*,a [.12, .31]	.28*,a [.21, .37]	.53*,c [.41, .68]	.43* [.34, .56]
Greater than weekly	.04*,b [.03, .06]	.16*,b [.10, .24]	.27*,a [.19, .38]	.29* [.19, .43]
Blacks				
Childhood-religious service attendance (C-RA; ages 6–13)[3]				
Weekly	.93 [.31, 1.19]	1.26 [.71, 2.24]	1.10 [.63, 1.92]	.69 [.35, 1.36]
Greater than weekly	.61 [.31, 1.86]	.99 [.56, 1.75]	.97 [.54, 1.73]	1.31 [.70, 2.46]
Adult-religious service attendance (A-RA; past 12 months, ages 18–29)[3]				
Weekly	.70 [.37, 1.31]	1.39 [.75, 2.57]	1.34 [.73, 2.48]	.48* [.24, .95]
Greater than weekly	.39* [.21, .74]	.89 [.45, 1.79]	.78 [.39, 1.56]	.43 [.18, 1.03]

a–d Within a model, ratios with different superscripts differ significantly from each other.

* Indicates significant at $p < .05$

1 Restricted to those who had ever consumed at least one full drink of alcohol (ie, alcohol initiation).

2 Risk relative to those who began drinking at age 18 or older.

3 Comparison group is those who did not attend church at least weekly.

TABLE 3

Twin pair correlations and standardized proportions of variance [95% Confidence Intervals] attributable to additive genetic (A), shared environmental (C), and non-shared environmental (E) influences for the full and final univariate models of religious service attendance during childhood (C-RA) and adulthood (A-RA) and alcohol involvement in 3,787 White and Black twins

	Twin pair correlations		Full model			Final model	
	r_{MZ}	r_{DZ}	Additive genetic (A)	Shared environment (C)	Person-specific environment (E)	A	E
Whites							
C-RA (more than weekly)	.78* [.72, .84]; N = 911 pairs	.66* [.56, .66]; N = 607 pairs	.23 [.01, .48]	.55 [.32, .74]	.22 [.16, .29]	ACE is best-fitting model	
A-RA (weekly or more)	.82* [.77, .87]; N = 911 pairs	.68* [.59, .77]; N = 607 pairs	.28 [.22, .50]	.54 [.33, .72]	.18 [.14, .24]	ACE is best-fitting model	
Ever drinking	.87* [.80, .94]; N = 910 pairs	.72* [.58, .87]; N = 606 pairs	.32 [.02, .69]	.55 [.20, .81]	.13 [.07, .22]	ACE is best-fitting model	
Timing of first alcohol use	.72* [.67, .77]; N = 833 pairs	.44* [.35, .53]; N = 549 pairs	.55 [.35, .75]	.17 [.00, .35]	.28 [.23, .33]	.73 [.68, .77]	.27 [.23, .32]
DSM5 AUD	.59* [.50, .67]; N = 823 pairs	.42* [.30, .55]; N = 542 pairs	.33 [.04, .64]	.26 [.00, .51]	.41 [.33, .50]	.61 [.53, .69]	.39 [.31, .47]
Blacks							
C-RA (more than weekly)	.68* [.50, .87]; N = 127 pairs	.33* [.07, .59]; N = 127 pairs	.68 [.06, .83]	.00 [.00, .50]	.32 [.17, .54]	.68 [.48, .83]	.32 [.17, .52]
A-RA (weekly or more)	.69* [.50, .87]; N = 127 pairs	.36* [.10, .62]; N = 127 pairs	.65 [.08, .84]	.03 [.00, .54]	.31 [.17, .53]	.69 [.50, .84]	.31 [.27, .51]
Ever drinking	.75* [.54, .97]; N = 127 pairs	.43* [.09, .76]; N = 128 pairs	.71 [.00, .91]	.04 [.00, .67]	.25 [.09, .52]	.76 [.51, .91]	.24 [.09, .49]
Timing of first alcohol use	.57* [.38, .76]; N = 99 pairs	.29* [.04, .53]; N = 96 pairs	.55 [.00, .72]	.01 [.00, .50]	.44 [.28, .66]	.56 [.36, .72]	.44 [.28, .64]
DSM5 AUD	.56* [.26, .86]; N = 102 pairs	.36 [−.03, .74]; N = 96 pairs	.44 [.00, .79]	.11 [.00, .65]	.45 [.21, .78]	.57 [.25, .79]	.43 [.21, .75]

* Indicates significant at $p < .05$.

TABLE 4

Additive genetic (r_A), family environmental (r_C), and non-shared environmental (r_E) correlations between religious service attendance during childhood (C-RA) and adulthood/past-year (A-RA) and alcohol involvement in 3,787 White and Black female twins

	Genetic correlation (r_A)	Shared environment correlation (r_C)	Person-specific environment correlation (r_E)	% of overlap attributable to A, C, and E[a]
Childhood-religious service attendance (C-RA; ages 6–13)				
Ever drinking				
Whites	–	−.72* [−1.00, −.59]	–	100% C
Timing of first alcohol use				
Whites	–	.42* [.21, 1.00]	–	100% C
Adult-religious service attendance (A-RA; past 12 months, ages 18–29)				
Ever drinking				
Whites	–	−.80* [−1.00, −.64]	−.40*,[b] [−.64, −.15]	83% C, 17% E
Blacks	–	–	−.40*,[b] [−.64, −.15]	100% E
Timing of first alcohol use				
Whites	.75* [.64, 1.00]	–	–	100% A
DSM5 Alcohol Use				
Disorder (AUD) Whites = Blacks	−.61* [−.91, −.44]	–	–	100% A

*Indicates $p < .05$

[a]A, additive genetic; C, shared environment, E, individual-specific environment;

[b]E component equated across race/ethnicity.