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Relationship of Alcohol Consumption and Type of Alcoholic Beverage Consumed With Plasma Lipid Levels: Differences Between Whites and African Americans of the ARIC Study

KELLY A. VOLCIK, PhD, CHRISTIE M. BALLANTYNE, MD, FLAVIO D. FUCHS, MD, A. RICHEY SHARRETT, MD, and ERIC BOERWINKLE, PhD

Human Genetics Center, University of Texas Health Science Center, Houston, TX (K.A.V., E.B.); Department of Medicine, Baylor College of Medicine, Houston, TX (C.M.B.); Division of Cardiology, Clinical Hospital of Porto Alegre, Federal University of Rio Grande do Sul, Porto Alegre, Brazil (F.D.F.); and Department of Epidemiology, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD (R.S.).

Abstract

PURPOSE: Alcohol consumption has been shown to contribute to a favorable lipid profile, and most studies have reported a reduction in coronary heart disease risk with low-to-moderate consumption of alcohol that is generally attributed to the beneficial effects of alcohol on lipids. The influence of different types of alcoholic beverages on plasma lipid levels has been investigated to a lesser extent and in limited populations.

METHODS: We investigated the effect of overall alcohol consumption, as well as the type of alcoholic beverage consumed, on multiple lipid measures in the large bi-ethnic population of the Atherosclerosis Risk in Communities study.

RESULTS: We found both low-to-moderate and heavy alcohol consumption, regardless of the type of alcoholic beverage consumed, to result in significantly greater levels of high-density lipoprotein (HDL) cholesterol, HDL₃ cholesterol, and apolipoprotein A-I in both white and African-American males and females. Associations with other lipid measures contrasted between whites and African Americans, with greater levels of alcohol consumption resulting in significantly greater triglyceride levels in African Americans.

CONCLUSIONS: Our results confirm previous studies associating alcohol consumption, regardless of beverage type, with greater HDL cholesterol levels, with additional consistent associations detected for the major HDL cholesterol density subfraction, HDL₃ cholesterol, and the major HDL cholesterol structural apolipoprotein, apolipoprotein A-I.

Keywords

Alcohol; Lipid; HDL; Triglycerides; ARIC

INTRODUCTION

Prospective studies have consistently reported a reduction in coronary heart disease (CHD) risk with low-to-moderate steady (as opposed to binge) consumption of alcohol (1-7). The

reduction in risk of CHD associated with low-to-moderate alcohol consumption is generally attributed to the beneficial effects of alcohol on lipids, namely high-density lipoprotein (HDL) cholesterol (8-12). Approximately half of the protective effects of alcohol against CHD have been suggested to be mediated by an increase in HDL cholesterol (13-15). A decrease in low-density lipoprotein (LDL) cholesterol with increased alcohol consumption has also been reported, but this effect is less consistent (9). In addition to HDL and LDL cholesterol, alcohol has been shown to affect levels of lipoprotein(a), apolipoprotein A-I, apolipoprotein A-II, apolipoprotein B, and triglycerides (13,15-19).

The majority of studies evaluating the different effects of the type of alcoholic beverage consumed have focused on disease end points. The influence of different types of alcoholic beverages on plasma lipid levels has been investigated to a lesser extent and in limited populations. Both cross-sectional and experimental studies suggest that the beneficial effects of low-to-moderate alcohol consumption on HDL cholesterol are not dependent upon the type of alcoholic beverage consumed (i.e., beer, wine, or spirits), but instead the result of alcohol itself rather than the nonalcoholic components of some alcoholic beverages (i.e. antioxidant polyphenols in red wine) (20-25). For the current study, we have investigated the effect of overall alcohol consumption, as well as the type of alcoholic beverage consumed, on multiple lipid measures in the large bi-ethnic population of the Atherosclerosis Risk in Communities (ARIC) study.

METHODS

The ARIC Study

Study participants were selected from the ARIC study, a prospective investigation of atherosclerosis and its clinical sequelae involving 15,792 individuals ages 45 to 64 years at recruitment (1987–1989). Institutional review boards approved the ARIC study, and all participants provided their written informed consent. A detailed description of the ARIC study design and methods has been published elsewhere (26). In brief, subjects were selected by probability sampling from four communities: Forsyth County, North Carolina; Jackson, Mississippi; northwestern suburbs of Minneapolis, Minnesota; and Washington County, Maryland. Participants were excluded from these analyses ($n = 6860$) if they 1) had an ethnic background other than white or African-American ($n = 48$), 2) took cholesterol-lowering medications within 2 weeks of the baseline clinical examination ($n = 446$), 3) were classified as a former drinker ($n = 2863$), 4) were classified as a current drinker but reported zero grams/week of alcohol consumed ($n = 2609$), or 5) had missing information for ethanol consumption or any of the lipid measures or covariates included in the analyses ($n = 894$). After exclusions, a total of 3270 white females, 3140 white males, 1634 African-American females and 888 African-American males were available for analysis.

Baseline Examination and Laboratory Measures

Home and clinic interviews and questionnaires included assessment of education level (highest level completed), use of cholesterol lowering medications, dietary intake, and cigarette smoking. Cigarette smoking status was analyzed by comparing current smokers to individuals who had formerly or never smoked. Cigarette years of smoking were calculated as the average number of cigarettes smoked per day times the number of years smoked for both current and former smokers. Body mass index (BMI, kg/m^2) was calculated from height and weight measurements. An index of physical activity in sports (sport index) was derived using the Baecke physical activity questionnaire (27). Keys score was calculated from the assessment of usual dietary intake (28). Blood glucose levels were measured with a hexokinase method. Plasma levels of total cholesterol and triglycerides were measured enzymatically, and the concentration of LDL cholesterol was calculated (29). HDL cholesterol was measured after

dextran-magnesium precipitation of non-HDL lipoproteins (30). Apolipoprotein A-I and apolipoprotein B were determined by radioimmunoassay (31-33).

Self-reported alcohol consumption was ascertained at baseline by means of an interviewer-administered dietary questionnaire. Subjects were asked whether they currently drank alcoholic beverages, and, if not, whether they had done so in the past. The amount of ethanol consumed (in grams per week) was calculated assuming the following alcohol content: 4 oz. of wine, 10.8 g; 12 oz. of beer, 13.2 g; and 1.5 oz. of hard liquor, 15.1 g. For a drinker who reported less than one drink per week, the alcohol consumption was recorded as zero grams per week.

Statistical Analysis

All statistical analyses were conducted using STATA version 8.0 (College Station, TX). Because of different drinking patterns between males and females and to variations in lipid measures between whites and African Americans, all analyses were conducted separately by race- and sex-specific strata. Additionally, race-sex strata by alcohol interaction terms were tested and were significant for all lipids studied, further justifying our stratified analyses. Multiple linear regression models were used to assess the relation between baseline alcohol consumption and lipid measures. Regression model coefficients were determined to be significant using the standard *t* test. For all analyses, the covariates included age, field center, smoking status, years of cigarette smoking, BMI, education level, physical activity, glucose levels, Keys score, and use of medications that secondarily lower cholesterol. BMI and physical activity values were log-transformed to achieve an approximately normal distribution.

Overall alcohol consumption was considered as a categorical variable (never/low-to-moderate/heavy). Categories of low-to-moderate and heavy were defined differently by sex using standard guidelines set forth by the U.S. Department of Health and Human Services/U.S. Department of Agriculture Dietary Guidelines 2005: for men, low-moderate ≤ 210 grams/week, heavy > 210 grams/week; for women, low-moderate ≤ 105 grams/week, heavy > 105 grams/week. The reference group only included never drinkers (former drinkers were excluded), thus avoiding the potential problem of including former drinkers which may include persons who have abstained from alcohol due to poor health (the "sick quitter effect") (34,35).

When considering the specific type of alcoholic beverage consumed, we took into consideration that persons were not likely to consume only one type of alcoholic beverage but rather consume different quantities of wine, beer, and/or spirits. To account for this, we defined a particular type of alcoholic beverage as predominant if consumption of that type of beverage (wine, beer, or spirits) accounted for two-thirds or more of the total amount of ethanol consumed, with other drinkers classified as 'no preference' drinkers (the reference group was never drinkers). This classification system has been reported in previous publications (34,36).

RESULTS

Mean lipid measures for each race- and sex-specific stratum, according to alcohol intake group and type of alcoholic beverage consumed, are presented in Tables 1a-d. The reference group for all analyses was never drinkers. For all race- and sex-specific strata, overall low-to-moderate and heavy drinkers had significantly greater measures of HDL cholesterol, HDL₃ cholesterol, and apolipoprotein A-I compared with never drinkers. The highest measures of HDL cholesterol, HDL₃ cholesterol, and apolipoprotein A-I were observed in overall heavy drinkers for all race-sex strata.

In white males and females (Tables 1a and b), all types of alcoholic beverages (for both low-to-moderate and heavy drinkers) were significantly associated with greater levels of HDL cholesterol, HDL₂ cholesterol, HDL₃ cholesterol, and apolipoprotein A-I, compared with never

drinkers. For all of these measures, the largest effects were observed in heavy drinkers for both white males and females. In white females only, both low-to-moderate and heavy drinkers had significantly lower LDL cholesterol levels, whereas only low-to-moderate drinkers had significantly lower apolipoprotein B and triglyceride levels. With regards to the type of alcoholic beverage consumed in white females, significantly lower levels of LDL cholesterol were observed for both wine and beer drinkers, while significantly lower levels of apolipoprotein B and triglycerides were observed only in wine drinkers.

For African-American males and females (Tables 1c and d), the number of wine drinkers was too small for interpretation. The number of no preference African-American female drinkers was also small. In both African-American males and females, all types of alcoholic beverages consumed (for both low-to-moderate and heavy drinkers) were associated with significantly higher levels of HDL cholesterol, HDL₃ cholesterol, and apolipoprotein A-I, compared with never drinkers. Also, in both African-American males and females, overall heavy drinkers had significantly higher triglyceride measures. Although greater triglyceride levels were observed for all types of alcoholic beverages consumed by both African-American male and female heavy drinkers, these findings were only significant for African-American male beer and spirits drinkers. Significant findings may not have been observed for triglycerides in the other heavy drinker groups and in African-American females due to the smaller number of persons in these groups.

DISCUSSION

In the current study, we demonstrated both low-to-moderate and heavy alcohol consumption, regardless of the type of alcoholic beverage consumed, to result in significantly greater levels of HDL cholesterol, HDL₃ cholesterol, and apolipoprotein A-I in both white and African-American males and females of the large ARIC study. Associations with other lipid measures contrasted between whites and African Americans, as well as between males and females. HDL₂ cholesterol levels were significantly associated with low-moderate and heavy drinking in both white males and females, but not in African Americans. Significantly lower levels of LDL cholesterol, apolipoprotein B, and triglycerides were observed only in white females, whereas significantly higher triglyceride levels were observed only in African-Americans. The small number of African-American wine drinkers precluded any conclusions about the association between lipid measures and wine consumption for this racial group. Overall, our results confirm previous studies associating alcohol consumption, regardless of beverage type, with higher HDL cholesterol levels, with additional consistent associations detected for the major HDL cholesterol density subfraction, HDL₃ cholesterol, and the major HDL cholesterol structural apolipoprotein, apolipoprotein A-I.

The effects of alcohol consumption on lipid profiles have been primarily investigated in men, or when women have been included, the data were generally not analyzed by sex. The few studies evaluating this relationship in separate populations of men and women have indicated that the association of alcohol consumption with higher levels of HDL cholesterol occurs at lower intakes of alcohol in women than in men (23,37-39). Results from the current study corroborate these previous findings. When comparing never drinkers to low-to-moderate drinkers, as well as never drinkers to heavy drinkers, the percent raise in HDL cholesterol levels was similar in both white and African-American men and women. However, these findings were observed for consuming ≤ 105 grams of alcohol per week for women (low-moderate; >105 grams/week for heavy) and for consuming ≤ 210 grams of alcohol per week for men (low-moderate; >210 grams/week for heavy). Women have been shown to be more sensitive to the effects of alcohol than men, especially with respect to liver function (37,40,41). Greater blood alcohol concentrations have also been observed in women compared with men after ingestion of the same amount of alcohol, with this difference attributed to decreased gastric metabolism

of alcohol by women (42). Thus, the question has been raised as to whether alcohol consumption also affects lipid metabolism differently in men and women, but biochemical studies of lipid and alcohol metabolism have not addressed this issue (37). In the current study, we observed similar changes in lipid levels at lower intakes of alcohol in women compared to men, but a mechanistic interpretation of these findings is difficult because of the lack of detailed metabolic and biochemical measures on these same individuals.

To our knowledge, this is the first study to investigate the association between alcoholic beverage consumption and lipid measures in a large sample of African Americans. The major HDL cholesterol density subfraction, HDL₂ cholesterol, while significantly associated with drinking in whites, was not associated with alcohol consumption in African Americans. In addition, significantly higher triglyceride levels were associated with heavy drinking in African Americans, but not in whites. Previous research in the ARIC study has demonstrated differences in whites and African Americans with regards to HDL and triglyceride levels (43-45). Although the mechanism(s) for these differences is not fully understood, it may partially be due to differences in body composition, glucose and insulin metabolism, and genetic factors. In never drinkers, triglyceride levels were lower in African Americans compared with whites, and HDL₂ cholesterol levels were greater in African Americans compared with whites. However, in drinkers of alcohol (most notably heavy drinkers), triglyceride and HDL₂ cholesterol levels were less disparate between African Americans and whites (Fig. 1). These findings are intriguing in light of the fact that the mechanism(s) by which alcohol affects lipid measures remains largely unknown (13,46,47). The differences observed in whites and African Americans with respect to the effects of alcohol intake on lipid measures (never drinkers have different levels; heavy drinkers have similar levels) could potentially be explained by genetic differences and thus warrant further study.

Limitations to the current study include the limited sample sizes obtained when classifying the race-sex strata by type of alcoholic beverage consumed, particularly in African Americans. Another limitation is that alcohol consumption data was obtained based on self-report. The validity of self-reported data on alcohol consumption has been debated, but there is evidence that the questionnaire administered to the ARIC participants adequately captured their alcohol consumption (34,36). With regards to self-reported alcohol data, another concern is that different patterns of alcohol consumption in the ARIC cohort, such as binge drinking, could not be assessed. We were unable to determine whether an individual's pattern of drinking included regular drinking (i.e., daily drinking) or binge drinking (i.e., predominantly weekend drinking), which may differentially affect lipid levels. However, a previous controlled trial determined that the favorable effects of alcohol on lipid measures were independent of drinking patterns (18).

Overall, the current study determined that both low-moderate and heavy alcohol consumption, regardless of the type of alcoholic beverage consumed, results in significantly greater levels of HDL cholesterol, HDL₃ cholesterol, and apolipoprotein A-I in both white and African-American males and females. Alcohol consumption has been shown to contribute to a favorable lipid profile, and studies have consistently reported a reduction in CHD risk with low-moderate consumption of alcohol that is generally attributed to the beneficial effects of alcohol on lipids (1-12). Alcohol consumption has been shown to have potential harmful effects on health as well, including increased risk for hypertension and hemorrhagic stroke, diseases that are more common in African Americans (36,48,49). Previous research in the ARIC study showed an inverse association between alcohol consumption and incidence of CHD in white men and women, but found a positive association between alcohol consumption and CHD in African-American men (34). Our observations of an absence of a beneficial effect of alcohol consumption on HDL₂ cholesterol and greater triglyceride levels in African Americans may be one factor of several contributing to the absence of protection against CHD in African

Americans of the ARIC cohort. Therefore, the beneficial effects of alcohol on cholesterol measures must be weighed against the potential harmful effects on health, with considerations given to one's sex, cardiovascular disease risk, family history, racial/ethnic background and drinking behavior.

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Selected Abbreviations and Acronyms

CHD	coronary heart disease
HDL	high-density lipoprotein
LDL	low-density lipoprotein
ARIC	Atherosclerosis Risk in Communities
BMI	body mass index

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FIGURE 1.

Mean triglyceride levels in male ARIC participants according to alcohol intake and race. Values are adjusted for age, center, smoking status, cigarette years of smoking, BMI, education, physical activity, glucose, keys score, and cholesterol medication use. Means marked with “a” differ from those marked “b” ($p < 0.05$) and “c” ($p < 0.001$).

TABLE 1A

Adjusted means of lipid measures by alcohol intake group and type of beverage consumed for white females

Alcohol intake	n	HDL (mg/dL)	HDL2 (mg/dL)	HDL3 (mg/dL)	LDL (mg/dL)	Apo A-I (mg/dL)	Apo B (mg/dL)	Triglycerides (mg/dL)
Never drinker ^a	1370	55.4 (0.5)	15.8 (0.3)	39.6 (0.3)	136.9 (1.1)	138.3 (0.9)	92.6 (0.8)	124.9 (1.7)
Overall								
Low-Mod	1481	61.0 (0.4) ***	18.4 (0.3) ***	42.6 (0.3) ***	131.9 (1.0) **	146.6 (0.8) ***	88.5 (0.7) **	116.7 (1.6) **
Heavy	419	68.5 (0.8) ***	21.6 (0.5) ***	46.8 (0.5) ***	130.0 (1.9) **	159.7 (1.5) ***	89.3 (1.4)	120.0 (2.9)
Wine								
Low-Mod	484	61.5 (0.8) ***	18.8 (0.4) ***	42.7 (0.5) ***	130.7 (1.9) *	148.8 (1.5) ***	88.4 (1.4) *	118.2 (2.9) *
Heavy	54	71.5 (2.1) ***	24.5 (1.2) ***	47.0 (1.3) ***	127.8 (5.1)	171.0 (4.1) ***	86.1 (3.7)	124.5 (7.9)
Beer								
Low-Mod	196	59.4 (1.1) ***	17.2 (0.6) *	42.2 (0.7) ***	134.1 (2.9)	143.2 (2.3) *	90.6 (2.1)	122.4 (4.6)
Heavy	82	66.3 (1.7) ***	20.0 (1.0) ***	46.3 (1.1) ***	124.3 (4.4) **	157.6 (3.4) ***	91.5 (3.2)	137.8 (7.0)
Spirits								
Low-Mod	444	61.0 (0.8) ***	18.4 (0.5) ***	42.6 (0.5) ***	133.0 (2.1)	147.5 (1.6) ***	89.9 (1.5)	120.8 (3.1)
Heavy	195	67.0 (1.2) ***	20.6 (0.7) ***	46.4 (0.7) ***	134.9 (3.0)	155.9 (2.3) ***	90.4 (2.1)	120.8 (4.5)
No Pref								
Low-Mod	357	60.6 (0.9) ***	18.4 (0.5) ***	42.1 (0.5) ***	136.0 (2.3)	147.3 (1.7) ***	90.5 (1.7)	121.5 (3.5)
Heavy	88	70.9 (1.7) ***	23.3 (1.0) ***	47.6 (1.0) ***	130.5 (4.2)	166.2 (3.2) ***	90.9 (3.1)	116.9 (6.4)

Values are presented as mean (standard error of the mean) and adjusted for age, center, smoking status, cigarette years of smoking, BMI, education, physical activity, glucose, keys score and cholesterol medication use.

^aReference category for each alcohol intake group.

*
p < 0.05.

**
p < 0.005.

p < 0.001.

TABLE 1B
Adjusted means of lipid measures by alcohol intake group and type of beverage consumed for white males

Alcohol intake	n	HDL (mg/dL)	HDL2 (mg/dL)	HDL3 (mg/dL)	LDL (mg/dL)	Apo A-I (mg/dL)	Apo B (mg/dL)	Triglycerides (mg/dL)
Never drinker ^a	485	40.5 (0.6)	9.3 (0.3)	31.2 (0.4)	140.4 (1.7)	114.4 (1.2)	95.7 (1.3)	132.8 (3.1)
Overall								
Low-Mod	2142	44.3 (0.3)***	10.5 (0.1)**	33.8 (0.2)***	140.3 (0.8)	121.4 (0.5)***	96.0 (0.6)	137.3 (1.4)
Heavy	513	51.8 (0.5)***	12.2 (0.3)***	39.6 (0.4)***	137.3 (1.6)	134.9 (1.1)***	95.4 (1.2)	137.1 (2.9)
Wine								
Low-Mod	207	45.1 (0.8)***	10.8 (0.4)*	34.3 (0.6)***	141.5 (2.6)	124.0 (1.9)***	95.2 (1.9)	135.3 (4.7)
Heavy	8	60.6 (3.9)***	16.2 (1.9)**	44.5 (3.0)***	120.0 (11.7)	165.8 (8.6)***	92.3 (8.9)	139.0 (21.6)
Beer								
Low-Mod	839	43.8 (0.4)***	10.4 (0.2)**	33.4 (0.3)***	139.1 (1.3)	119.9 (0.9)**	95.1 (0.9)	139.7 (2.2)
Heavy	221	52.3 (0.8)***	12.3 (0.4)***	40.0 (0.6)***	137.1 (2.5)	136.6 (1.7)***	95.1 (1.8)	136.7 (4.4)
Spirits								
Low-Mod	552	45.4 (0.5)***	10.9 (0.2)***	34.5 (0.4)***	141.1 (1.5)	123.7 (1.1)***	97.1 (1.2)	138.1 (2.9)
Heavy	178	50.8 (0.9)***	12.3 (0.4)***	38.6 (0.7)***	136.3 (2.7)	133.2 (2.0)***	97.1 (2.1)	145.2 (5.2)
No Pref								
Low-Mod	544	44.5 (0.5)***	10.2 (0.3)*	34.3 (0.4)***	140.4 (1.6)	122.9 (1.1)***	96.6 (1.2)	134.1 (2.7)
Heavy	106	52.0 (1.1)***	11.9 (0.6)***	40.0 (0.9)***	139.4 (3.6)	134.1 (2.4)***	94.6 (2.7)	125.0 (6.0)

Values are presented as mean (standard error of the mean) and adjusted for age, center, smoking status, cigarette years of smoking, BMI, education, physical activity, glucose, keys score and cholesterol medication use.

^aReference category for each alcohol intake group.

*
p < 0.05.

**
p < 0.005.

p < 0.001.

TABLE 1C
Adjusted means of lipid measures by alcohol intake group and type of beverage consumed for African-American females

Alcohol intake	N	HDL (mg/dL)	HDL2 (mg/dL)	HDL3 (mg/dL)	LDL (mg/dL)	Apo A-I (mg/dL)	Apo B (mg/dL)	Triglycerides (mg/dL)
Never drinker ^a	1314	57.4 (0.4)	17.7 (0.3)	39.7 (0.3)	137.2 (1.2)	142.8 (0.9)	90.8 (0.8)	105.4 (1.3)
Overall								
Low-Mod	262	61.4 (1.0)**	18.1 (0.6)	43.2 (0.7)***	138.6 (2.8)	150.7 (2.0)***	91.7 (1.9)	106.5 (3.0)
Heavy	58	67.6 (2.2)***	19.3 (1.2)	48.4 (1.4)***	132.8 (5.8)	163.7 (4.2)***	91.3 (4.0)	118.9 (6.4)*
Wine								
Low-Mod	30	60.1 (2.9)	17.0 (1.6)	43.1 (1.9)	147.1 (8.0)	146.3 (5.7)	97.3 (5.6)	112.3 (8.6)
Heavy	1	—	—	—	—	—	—	—
Beer								
Low-Mod	123	61.0 (1.5)*	17.4 (0.8)	43.6 (1.0)***	139.0 (4.0)	149.6 (2.9)*	93.4 (2.8)	109.4 (4.4)
Heavy	32	66.2 (2.9)**	18.2 (1.6)	47.9 (1.9)***	120.6 (7.8)*	161.2 (5.7)**	85.7 (5.4)	117.1 (8.6)
Spirits								
Low-Mod	63	63.8 (2.0)**	19.5 (1.2)	44.2 (1.3)**	141.0 (5.5)	155.9 (4.0)**	90.6 (3.9)	102.6 (5.9)
Heavy	18	73.6 (3.8)***	21.9 (2.2)	51.8 (2.5)***	147.5 (10.3)	169.4 (7.4)***	102.6 (7.2)	109.8 (11.1)
No Pref								
Low-Mod	46	61.0 (2.4)	19.4 (1.3)	41.6 (1.6)	131.0 (6.5)	150.8 (4.7)	88.3 (4.5)	100.0 (7.1)
Heavy	7	59.6 (5.9)	16.4 (3.4)	43.2 (3.9)	159.9 (16.3)	153.0 (11.7)	94.3 (11.4)	141.7 (7.7)*

Values are presented as mean (standard error of the mean) and adjusted for age, center, smoking status, cigarette years of smoking, BMI, education, physical activity, glucose, keys score and cholesterol medication use.

^aReference category for each alcohol intake group.

* p < 0.05.

** p < 0.005.

*** p < 0.001.

TABLE 1D
Adjusted means of lipid measures by alcohol intake group and type of beverage consumed for African-American males

Alcohol intake	N	HDL (mg/dL)	HDL2 (mg/dL)	HDL3 (mg/dL)	LDL (mg/dL)	Apo A-I (mg/dL)	Apo B (mg/dL)	Triglycerides (mg/dL)
Never drinker ^a	296	49.0 (1.0)	13.6 (0.5)	35.4 (0.7)	137.3 (2.6)	127.8 (1.8)	92.2 (1.9)	107.1 (3.5)
Overall								
Low-Mod	464	53.3 (0.8)**	14.2 (0.4)	39.1 (0.5)***	135.7 (2.0)	135.7 (1.4)**	93.6 (1.4)	115.9 (2.6)*
Heavy	128	58.1 (1.5)***	15.3 (0.8)	42.7 (1.0)***	128.2 (3.9)	147.0 (2.8)***	92.8 (2.8)	133.8 (5.3)***
Wine								
Low-Mod	16	44.6 (3.4)	9.9 (1.7)*	34.7 (2.4)	144.6 (11.0)	128.9 (6.9)	105.2 (7.9)	112.3 (13.3)
Heavy	2	—	—	—	—	—	—	—
Beer								
Low-Mod	193	53.7 (1.2)**	14.1 (0.6)	39.6 (0.8)***	141.1 (3.1)	136.3 (2.2)**	95.6 (2.2)	112.2 (4.0)
Heavy	38	61.8 (2.7)***	16.9 (1.4)*	44.9 (1.9)***	128.6 (7.2)	156.0 (5.1)***	91.1 (5.0)	135.4 (9.1)*
Spirits								
Low-Mod	153	52.4 (1.3)*	14.2 (0.7)	38.2 (0.9)*	133.4 (3.5)	133.8 (2.4)*	93.6 (2.6)	124.1 (4.8)*
Heavy	52	58.9 (2.3)***	16.1 (1.2)*	42.8 (1.5)***	130.1 (6.2)	149.9 (4.2)***	94.6 (4.6)	139.9 (8.5)**
No Pref								
Low-Mod	102	55.8 (1.5)***	14.7 (0.7)	41.0 (1.1)***	137.2 (4.4)	140.1 (2.9)***	94.4 (3.1)	114.1 (5.5)
Heavy	36	55.1 (2.7)*	13.2 (1.3)	41.9 (1.9)**	124.1 (7.9)	136.3 (5.1)	93.8 (5.6)	125.4 (9.8)

Values are presented as mean (standard error of the mean) and adjusted for age, center, smoking status, cigarette years of smoking, BMI, education, physical activity, glucose, keys score and cholesterol medication use.

^aReference category for each alcohol intake group.

* p <0.05.

** p <0.005.

*** p <0.001.