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Association between Alcoholic Beverage Consumption and Incidence of Coronary Heart Disease in Whites and Blacks

The Atherosclerosis Risk in Communities Study

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The authors evaluated the relation between consumption of alcoholic beverages and incidence of coronary heart disease in White and African-American participants in the Atherosclerosis Risk in Communities Study. The average duration of follow-up was 9.8 years between 1987 and 1998. The association was analyzed by means of Cox proportional hazards regression models. The authors found a positive association between ethanol consumption and incident coronary heart disease for Black men (for a 13-g/day increment in ethanol consumption, adjusted hazard ratio (HR) = 1.13, 95% confidence interval (CI): 1.01, 1.28) and an inverse association for White men (HR = 0.88, 95% CI: 0.79, 0.99). There was an inverse association of coronary heart disease with rare drinking (HR = 0.47, 95% CI: 0.28, 0.80) and with consumption of ≥70 g of ethanol per week (HR = 0.49, 95% CI: 0.24, 0.98) in White women and with consumption of ≥210 g/week (HR = 0.56, 95% CI: 0.33, 0.95) in White men. In Black men, the association was positive for consumption of 140–<210 g/week (HR = 2.61, 95% CI: 1.11, 6.17). The contrasting findings in Whites and Black men in this cohort raise the question of whether the cardioprotective effect of alcohol is real or may be confounded by lifestyle characteristics of drinkers.

alcohol drinking; coronary disease

Abbreviations: ARIC, Atherosclerosis Risk in Communities; CHD, coronary heart disease; HDL, high density lipoprotein.

Consumption of high amounts of alcoholic beverages is a definite risk to health. Consumption of lower amounts, however, may not be harmful or may even be beneficial. Prevention of coronary heart disease (CHD) is the chief potential benefit attributed to light consumption of alcoholic beverages—an association that has been attributed to alcohol itself or to alcohol beverage components. Most (1–9) but not all (10–12) observational studies of the alcohol-CHD relation have shown a lower incidence of CHD among persons drinking low-to-moderate amounts of alcoholic beverages in general, and wine in particular, in comparison with nondrinkers. In the absence of a clinical trial, however, the possibility that this association may be confounded by other physical, socioeconomic, and lifestyle characteristics shared by moderate drinkers cannot be discarded. Meanwhile, it is worth examining the association between alcoholic beverage consumption and CHD incidence in different populations, preferably in studies with detailed information on the amounts and types of alcoholic beverages used and on risk factors for CHD that might act as confounders. In this paper, we present data showing inconsistent patterns of association between consumption of alcohol and different types of alco-

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holic beverages and incidence of CHD in different race-sex strata of the Atherosclerosis Risk in Communities (ARIC) Study cohort.

MATERIALS AND METHODS

The design of the ARIC Study has been described previously (13–16). The overall ARIC cohort consists of 15,792 persons aged 45–64 years at their baseline examination (visit 1). Approximately equal numbers of participants were selected from four communities in the United States: Forsyth County, North Carolina; Jackson, Mississippi; selected suburbs of Minneapolis, Minnesota; and Washington County, Maryland. The latter two samples were predominantly White. Only Blacks were sampled in Jackson, while 12 percent of the study sample from Forsyth County was Black. The response rate was 46 percent of eligible participants in Jackson and approximately 65 percent in the other three communities. Baseline data were collected between 1987 and 1989 (visit 1). Participants underwent reexamination in 1990–1992 (visit 2; 93 percent return rate), 1993–1995 (visit 3; 86 percent return rate), and 1996–1998 (visit 4; 81 percent return rate) and were followed until 1998 for this analysis.

Exclusions included 48 participants who were neither Black nor White; 51 Black participants from Minneapolis or Washington County; 762 persons who had CHD at the baseline evaluation (electrocardiographic evidence of myocardial infarction, reported history of myocardial infarction, or reported angina, coronary bypass, or angioplasty); and 425 persons with missing data on drinking status, ethanol intake, or prevalent CHD at baseline. The final sample contained 14,506 persons with information on incident CHD, with a mean follow-up period of 9.8 years.

Alcohol consumption

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. Former drinkers were asked to give the number of drinks of hard liquor, bottles of beer, and glasses of wine that they used to drink per week. In calculating the amount of ethanol consumed (in g/week), it was assumed that 4 ounces (118 ml) of wine contain 10.8 g of ethanol, 12 ounces (355 ml) of beer contain 13.2 g, and 1.5 ounces (44 ml) of liquor contain 15.1 g. When asked how many glasses, bottles, or drinks they usually consumed per week, a large proportion of persons who classified themselves as current drinkers reported none. These people were classified as rare drinkers in our analysis, with the other categories being current drinkers, never drinkers, and former drinkers. To determine whether the association between alcohol use and incident CHD varied by amount consumed, the category of current drinkers was stratified into 70-g/week intervals of alcohol consumption.

Among current drinkers, a particular type of alcoholic beverage was defined as predominant if consumption of that type of beverage (wine, beer, or liquor) accounted for two thirds or more of the total amount of ethanol consumed (14). Other drinkers were classified as drinkers with no preference.

Incident CHD

Incident CHD events included CHD death, hospitalized myocardial infarction, silent infarction detected by electrocardiogram, coronary bypass, and angioplasty occurring before December 31, 1998. The overall response rate at that time was 96.1 percent of the participants who were still alive. Possible clinical events were ascertained via annual follow-up telephone calls and surveillance of vital records and community hospitals. A death was classified as being due to CHD in the absence of a probable non-CHD cause among persons with either a recent myocardial infarction, chest pain within 72 hours of death, or a history of CHD. A more complete description of event ascertainment has been published elsewhere (17).

Measurement of other baseline covariates

Home and clinic interviews included assessment of educational level (highest level completed), total family income, cigarette smoking, and the presence of diabetes mellitus. The average of the second and third of three measurements of sitting blood pressure, measured with a Hawksley random-zero sphygmomanometer after 5 minutes of rest, was used in the analyses. An index of physical activity in sports (sport index) was derived using the Baecke physical activity questionnaire (18). Levels of total cholesterol, high density lipoprotein (HDL) cholesterol, and triglycerides were measured enzymatically, and the concentration of low density lipoprotein cholesterol was calculated. Anthropometric measurements for the calculation of body mass index (weight (kg)/height $(m)^2$) were carried out with participants wearing light clothing and no shoes. Diabetes was defined as a self-reported physician diagnosis, a fasting glucose level above 125 mg/dl, a nonfasting glucose level above 199 mg/dl, or self-reported pharmacologic treatment for diabetes.

Statistical analysis

Analyses were conducted within four race- and sex-specific strata (White and Black, men and women). Piecewise cubic (spline) models (19) were used to test for linearity of the association between alcohol consumption and incidence of CHD in the four race-sex strata. The association between incidence of CHD and baseline use of ethanol was analyzed by means of Cox proportional hazards regression models (20), controlling for several risk factors for CHD, including age, cigarette-years of smoking (number of cigarettes per year × years of smoking), body mass index, low density lipoprotein cholesterol, waist:hip ratio, educational level, income level, sport index, and diabetes. In a supplemental model, we also adjusted for systolic blood pressure, use of antihypertensive medication, and HDL cholesterol level—potential intermediate factors for the effects of alcohol consumption on CHD incidence. Inclusion of a variety of nutritional variables assessed by food frequency

TABLE 1. Selected characteristics of the study sample, by race and sex, Atherosclerosis Risk in Communities Study, 1987–1998*

Characteristic	White men (*n* = 4,820)		White women (*n* = 5,786)		Black men (*n* = 1,456)		Black women (*n* = 2,444)	
	No. or mean	%	No. or mean	%	No. or mean	%	No. or mean	%
Mean age (years)	54.6 (5.7)†		53.9 (5.7)		53.7 (6.0)		53.3 (5.7)	
Highest level of education								
Grade school	308	6.4	252	4.4	363	25.0	393	16.1
High school (no degree)	493	10.2	674	11.7	265	18.3	574	23.5
High school graduation/vocational school	1,889	39.2	2,942	50.9	378	26.1	726	29.8
College or higher	2,123	44.1	1,913	33.1	445	30.7	746	30.6
Annual income								
<$12,000	178	3.8	504	9.2	397	30.5	1,014	45.9
$12,000–$15,999	199	4.3	355	6.5	155	11.9	269	12.2
$16,000–$34,999	1,438	30.9	1,944	35.4	443	34.0	640	29.0
≥$35,000	2,833	61.0	2,696	49.0	308	23.6	286	12.9
Mean body mass index‡	27.4 (4.0)		26.6 (5.5)		27.6 (5.0)		30.8 (6.5)	
Body mass index category								
<25	1,309	27.2	2,696	46.6	449	30.9	418	17.1
25–29	2,470	51.3	1,755	30.4	608	41.8	838	34.3
≥30	1,039	21.6	1,330	23.0	397	27.3	1,187	48.6
Mean blood pressure (mmHg)								
Systolic	120.1 (16.0)		117.0 (17.7)		130.1 (21.0)		127.9 (21.2)	
Diastolic	73.7 (9.9)		69.8 (9.8)		82.5 (12)		78.0 (11.4)	
Current drinker	2,645	54.9	1,953	33.8	655	45.0	372	15.2
Rare drinker§	735	15.2	1,576	27.2	73	5.0	137	5.6
Current smoker	1,177	24.4	1,435	24.8	555	38.1	596	24.4
Parental history of hypertension	2,245	47.3	3,251	56.9	736	52.2	1,521	63.5
Poor self-perceived health	46	1.0	99	1.7	109	7.7	151	6.3
Diabetes mellitus	445	9.3	452	7.8	249	17.3	475	20.0

* The average duration of follow-up was 9.8 years. Some frequencies do not sum to the total number of participants because of missing data.
† Numbers in parentheses, standard deviation.
‡ Weight (kg)/height (m)^2.
§ Rare drinkers were defined as self-reported current drinkers who consumed less than one drink per week, on average.

questionnaire (21), including dietary sodium, potassium, calcium, magnesium, fiber, carbohydrates, saturated and unsaturated fatty acids, and serum levels of sodium, potassium, calcium, and magnesium, did not change the estimates substantially; thus, these variables were excluded from the final model. In a model including all Black and White men, we tested for the significance (Wald test) of the hazard ratios for the interaction terms between race and the four categories of drinkers. We additionally allowed for interactions between race and independent covariables in order to explain potential racial differences in the association between alcohol consumption and CHD incidence. The association between type of beverage predominantly consumed and incidence of CHD was examined in similar models, with additional adjustment for the amount of ethanol consumed. We investigated models containing terms for the interaction between beverage type and ethanol level to see whether the association between CHD and drink preference varied by the amount of ethanol consumed. All analyses were conducted with SAS, version 8 (22).

RESULTS

Race- and sex-specific distributions for several baseline characteristics are presented in table 1. Black participants had fewer years of formal education, a lower income, a higher prevalence of obesity (as defined by a body mass index of ≥30), higher blood pressure, and a higher prevalence of diabetes. For both sexes, the percentage of current drinkers was higher in Whites than in Blacks. More than one fourth of White women identified themselves as current drinkers but reported consuming less than one drink per week, and thus were classified as rare drinkers. More Blacks than Whites perceived their health as poor, and more reported a family history of hypertension.

During an average of 9.8 years of follow-up, 707 persons (4.9 percent) had a CHD event, of which 146 were fatal.

In models including a linear term for ethanol intake (g/day), controlling for age, cigarette smoking, body mass index, physical activity, low density lipoprotein cholesterol, waist:hip ratio, and diabetes, the consumption of ethanol was inversely associated with the incidence of CHD among Whites and positively associated with the incidence of CHD among Black men. The adjusted hazard ratio for a 13-g/day increment in ethanol consumption (the approximate amount of ethanol in one drink) was 1.13 (95 percent confidence interval: 1.01, 1.28) in Black men and 0.88 (95 percent confidence interval: 0.79, 0.99) in White men. In models also adjusted for HDL cholesterol level, systolic blood pressure, and use of blood-pressure-lowering medication (putative mediators of the effects of alcohol on the cardiovascular system), the estimates were almost identical but no longer significant. With the use of a restricted cubic spline for the CHD-ethanol association (piecewise cubic, linear in the tails), only Black males exhibited a statistically significant deviation from a linear model ($p = 0.04$).

Table 2 shows hazard ratios for incident CHD in the four race-sex strata according to category of alcohol consumption. In Whites, there was an overall trend of an inverse association between alcohol consumption and incidence of CHD. The estimates for rare drinking were similar to those observed for drinking of higher amounts of alcohol in both men and women. The association between alcohol consumption and CHD incidence among Black men was positive in all strata of alcohol consumption and was statistically significant in the 140–<210 g/week category (table 2). Associations for Black women had large confidence intervals because of the small numbers of persons in some strata and the lower incidence of CHD. In models also adjusted for HDL cholesterol level, systolic blood pressure, and use of blood-pressure-lowering medication, the estimates and confidence limits changed only slightly. The hazard ratio for the interaction between race (Black men vs. White men) and the categories of alcohol consumption was not significant ($p = 0.15$). The p value for the test of the interactions between the alcohol dummy variables and race was 0.27.

Figure 1 presents these findings, with the several strata of current drinkers collapsed into one category. Again, the hazard ratios were on the protective side for Whites and Black women and on the riskier side for Black men, but in most cases the 95 percent confidence intervals included the null hypothesis. Current drinking and rare drinking had very similar associations in all race-sex strata.

The hazard ratios for incident CHD among current drinkers by predominant type of beverage consumed are presented in table 3. The hazard ratios were less than 1 (inverse association) for all beverage types for White women and higher than 1 (positive association) in Black men but were statistically significant only for liquor in Black men. In White men, the association was positive for beer and wine and inverse for liquor and no preference (not statistically significant). The estimates changed only slightly in models containing systolic blood pressure, blood pressure medication, and HDL cholesterol. The small number of Black women who were classified as drinkers did not allow calculation of meaningful estimates in this stratum. No statistically significant interaction between beverage type and level of ethanol was observed.

DISCUSSION

In this analysis, we demonstrated an inverse association between alcohol consumption and incidence of CHD among Whites in a large cohort of free-living US citizens. The association between alcohol consumption and incident CHD in Black men contrasted with the findings in Whites. The very infrequent drinking among Black women precluded any conclusions about the association between alcoholic beverage consumption and incident CHD in this race-sex stratum. Overall, these findings are in agreement with the absence of any consistent cross-sectional association between current alcohol intake and carotid atherosclerosis in the baseline data of the ARIC Study (23).

To our knowledge, this is the first study to explore the longitudinal association between alcohol consumption and incidence of CHD in African Americans. The risk of alcohol consumption at lower amounts was previously described in Black men in a cross-sectional US national survey, wherein Black men reported having CHD more frequently at lower levels of alcohol consumption than White men (24). The positive association between alcohol consumption and CHD in Black male participants in the ARIC cohort has no apparent explanation. The consumption of alcohol at low amounts was a risk factor for hypertension in Black males in this cohort (14). However, this and other differential effects of alcohol on risk factors for CHD could hardly explain the association observed in Blacks, since the risk was independent of several potentially confounding factors controlled in the multivariate analysis. Models that included hypertension and HDL cholesterol, which are putative mediators of the effects of alcohol on the cardiovascular system, did not substantially modify the association in Black men.

In White men and women, the estimates of association between alcohol consumption and incident CHD were similar to those described in most previous studies that investigated the association between alcohol and CHD. The persistence of an inverse association in Whites who consumed higher amounts of ethanol is similar to the observations of some other studies, but most of them demonstrated that alcohol may protect against CHD if consumed at lower amounts (25). The most noticeable finding in Whites, however, was the reduced hazard ratios observed in rare drinkers, since it is not biologically plausible that either alcohol or other components of alcoholic beverages could have a protective effect when taken in very low amounts. This inverse association cannot be attributed to bias related to the presence of former drinkers in the reference group, because our reference group comprised only never drinkers. This has been a potential problem in early studies that combined never and past drinkers as the reference group. Past drinkers may include people who abstain from alcohol because of poor health. Inclusion of past drinkers within the reference population could contribute to the observation of a protective association between alcohol consumption and incident cardiovascular disease (26). However, our study, like some other studies (1), included potentially sick quitters

TABLE 2. Hazard ratio for incident coronary heart disease in different strata of alcohol consumption, by race and sex, Atherosclerosis Risk in Communities Study, 1987–1998*

Race, sex, and level of exposure	No. exposed	Incident coronary heart disease		Hazard ratio†	95% confidence interval	Hazard ratio‡	95% confidence interval
		No.	%				
White men							
Never drinker	490	41	8.4	1.0		1.0	
Former drinker	950	80	8.4	0.84	0.56, 1.27	0.90	0.59, 1.37
Rare drinker§	735	48	6.5	0.77	0.49, 1.22	0.79	0.50, 1.25
Current drinker (g/week)							
1–<70	1,141	87	7.6	0.93	0.62, 1.40	1.05	0.70, 1.58
70–<140	633	40	6.3	0.72	0.44, 1.16	0.81	0.50, 1.32
140–<210	368	21	5.7	0.67	0.38, 1.18	0.81	0.45, 1.44
≥210	503	27	5.4	0.56	0.33, 0.95	0.69	0.40, 1.18
White women							
Never drinker	1,448	55	3.8	1.0		1.0	
Former drinker	809	38	4.7	0.91	0.58, 1.42	0.93	0.59, 1.47
Rare drinker	1,576	24	1.5	0.47	0.28, 0.80	0.49	0.29, 0.84
Current drinker (g/week)							
1–<70	1,250	24	1.9	0.60	0.34, 1.04	0.64	0.36, 1.12
≥70	703	12	1.7	0.49	0.24, 0.98	0.55	0.27, 1.13
Black men							
Never drinker	328	17	5.2	1.0		1.0	
Former drinker	400	36	9.0	1.73	0.91, 3.30	1.68	0.88, 3.21
Rare drinker	73	5	6.8	1.32	0.43, 4.05	1.39	0.45, 2.84
Current drinker (g/week)							
1–<70	247	18	7.3	1.57	0.75, 3.26	1.80	0.86, 3.76
70–<140	171	10	5.8	1.09	0.44, 2.72	1.13	0.45, 2.84
140–<210	90	11	12.2	2.61	1.11, 6.17	2.67	1.12, 6.34
≥210	147	8	5.4	1.30	0.51, 3.32	1.10	0.40, 2.98
Black women							
Never drinker	1,449	62	4.3	1.0		1.0	
Former drinker	486	32	6.6	1.33	0.82, 2.15	1.45	0.89, 2.38
Rare drinker	137	1	0.7	0.28	0.04, 2.06	0.37	0.05, 2.71
Current drinker (g/week)							
≥1	372	10	2.7	0.51	0.21, 1.21	0.49	0.20, 1.18

* The average duration of follow-up was 9.8 years. The reference category for each race-sex group was never drinkers.

† Adjusted for age, cigarette-years of smoking, body mass index, low density lipoprotein cholesterol level, waist: hip ratio, educational level, income, sport index, and diabetes mellitus.

‡ Additionally adjusted for systolic blood pressure, use of antihypertensive medication, and high density lipoprotein cholesterol level.

§ Rare drinkers were defined as self-reported current drinkers who consumed less than one drink per week, on average.

in the category of former drinkers, not in the reference population.

The category of very infrequent drinking is not commonly included in longitudinal studies of the association between alcohol consumption and CHD. In most studies, the category into which very infrequent drinkers were placed is not clear, since categories of alcohol consumption are often stratified in ranges of numbers of drinks or equivalents (0, 1–<3, 3–7, etc.) consumed per day or per week. In the few studies in which data on infrequent drinkers have been presented, an inconsistent association of this pattern of drinking with incident CHD and with other risk factors for CHD has been described. In the Kaiser Permanente study, very infrequent drinkers had a risk of coronary artery disease hospitalization

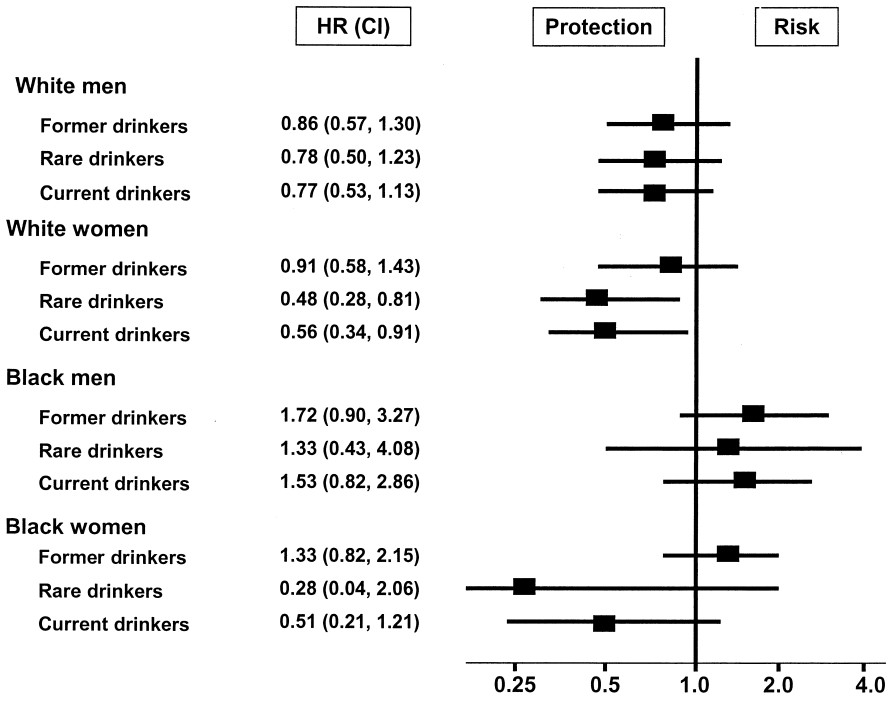

FIGURE 1. Hazard ratio (HR) for incident coronary heart disease in different strata of alcohol consumption, by race and sex, Atherosclerosis Risk in Communities Study, 1987–1998. The reference category for each race-sex group was never drinkers. Hazard ratios were adjusted for age, cigarette-years of smoking, body mass index, low density lipoprotein cholesterol level, waist:hip ratio, educational level, income, sport index, and diabetes mellitus. Horizontal bars, 95% confidence interval (CI).

similar to that of lifelong abstainers (27). An inverse, though nonsignificant, association between the consumption of less than 1.5 g of alcohol per day and the incidence of CHD in women has been described in the Nurses' Health Study (4). In the Physicians' Health Study, persons classified as rare drinkers had concentrations of endogenous tissue type plasminogen activator antigen similar to those of never drinkers (28).

The associations between type of alcoholic beverage consumed and incidence of CHD were inconsistent, since none were significantly associated with lower incidence of CHD in any race-sex strata. The estimates tended to be positive for all beverages in Black men, negative for all beverages in White women, and close to the null in White men. However, statistical power for this analysis was low. There was no statistically significant interaction between preference and the amount of alcohol consumed. Wine has been implicated most commonly among alcoholic beverages in offering protection against cardiovascular disease, but virtually all types of alcoholic beverages seem to be inversely associated with the incidence of CHD in some studies (3, 29, 30).

The lack of a consistent direction for the associations between alcohol consumption and incident CHD in Black men and Whites, particularly the inverse association observed in Whites who declared themselves rare drinkers, suggests that these levels of alcohol consumption may be proxies for the real causes of CHD in these race-sex strata or that they are merely chance associations. Many physical and behavioral characteristics implicated as CHD risk factors were controlled in this study, such as physical activity, smoking, blood lipid levels, blood pressure, income, education, and nutritional habits. Therefore, the real causes might be among unmeasured risk factors, such as mental health (31), socioeconomic position in early life (32), psychosocial characteristics (33–36), social networks (37), sources of emotional support (38), or other unknown confounders, which may vary by sex and ethnic background. The opposite patterns that we observed in Black men and Whites, particularly between the Black drinkers of substantial amounts and White persons who were very infrequent drinkers, may be secondary to differences in health behavior between ethnic groups. Interactions of social class with race/ethnicity and with drinking problems have been described in the United States (39, 40). Less affluent Black men reported greater numbers of drinking consequences and drinking problems than less affluent White men, while more affluent Black men had fewer alcohol-related problems than more affluent Whites (39). Black persons who reported higher involvement with social networks and higher political awareness reported drinking at lower levels than other Blacks (40). The worse outcome in Black men who consumed any amount of alcohol is emphasized by the fact that Black participants in the ARIC cohort were from the Bible Belt. Alcohol drinkers in this conservative zone may be more stressed by their drinking, given the lack of social support for alcohol use in their communities. This finding may not apply to Black people living in other regions in the United States, where

TABLE 3. Hazard ratio for incident coronary heart disease according to predominant type of beverage consumed among current drinkers, by race and sex, Atherosclerosis Risk in Communities Study, 1987–1998*,†

Race, sex, and predominant type of beverage consumed	No. of coronary heart disease events	No. of participants exposed	Hazard ratio‡	95% confidence interval	Hazard ratio§	95% confidence interval
White men						
Beer	76	1,051	1.02	0.66, 1.60	1.15	0.74, 1.81
Wine	13	210	1.03	0.54, 1.97	1.21	0.63, 2.32
Liquor	48	713	0.83	0.51, 1.36	0.92	0.56, 1.51
No preference	38	671	0.83	0.50, 1.39	0.93	0.55, 1.56
White women						
Beer	5	286	0.37	0.12, 1.09	0.30	0.09, 1.03
Wine	7	552	0.58	0.24, 1.39	0.67	0.28, 1.63
Liquor	14	651	0.66	0.31, 1.39	0.68	0.32, 1.45
No preference	10	464	0.76	0.34, 1.70	0.87	0.38, 1.99
Black men						
Beer	17	254	1.40	0.60, 3.26	1.47	0.62, 3.47
Wine/no preference	9	174	0.97	0.34, 2.77	1.30	0.46, 3.71
Liquor	21	227	2.18	0.97, 4.90	2.56	1.13, 5.81

* The average duration of follow-up was 9.8 years. The reference category for each race-sex group was never drinkers.

† Black women were excluded because of the small numbers of subjects exposed by category and the small number of events.

‡ Adjusted for age, cigarette-years of smoking, ethanol intake, body mass index, low density lipoprotein cholesterol level, waist:hip ratio, educational level, income, sport index, and diabetes mellitus.

§ Additionally adjusted for systolic blood pressure, use of blood-pressure-lowering medication, and high density lipoprotein cholesterol level.

social norms against drinking may be not so tight as in the South.

At this point in time, it seems difficult to challenge the consensus concerning the cardioprotective effects of ethanol or some alcoholic beverages, particularly wine. Even investigators who have explored other potential benefits of alcohol consumption contend that light-to-moderate alcohol consumption reduces the risk of CHD (41). Not only is this cardioprotection generally accepted but many investigators have described mechanisms that could lead to such protection. A beneficial effect of ethanol on risk factors for cardiovascular disease, such as blood lipoprotein levels (42, 43), clotting and fibrinolytic factors (28, 44), and insulin sensitivity (45), is one of the mechanisms that could confer protection against cardiovascular disease. A beneficial effect on insulin and triglyceride concentrations from consumption of two drinks per day was recently demonstrated in a clinical trial with nondiabetic postmenopausal women (46). An interaction between moderate alcohol consumption and a genetic polymorphism has been suggested as a cause for higher HDL cholesterol levels and lower myocardial infarction risk among drinkers (47). Components of wine, such as antioxidants (48, 49), inhibitors of endothelin-1 synthesis (50), and polyphenols that increase the release of nitric oxide from endothelial cells (51), are other potential mechanisms of coronary protection. However, in the absence of an unequivocal effect demonstrated in a clinical trial, the large volume of epidemiologic, experimental, and clinical effects of alcohol on surrogate endpoints should be interpreted as hypothesis-generating in terms of cardiovascular disease protection. An analogous situation that shares many similarities with the postulated protection conferred by wine and other beverages is the case of postmenopausal hormone replacement therapy. The large amount of favorable evidence arising from observational studies, experimental studies, and clinical effects on surrogate endpoints was not confirmed in a clinical trial designed to investigate the efficacy of hormone replacement therapy in the primary prevention of CHD and stroke (52).

The findings among Black and White participants in the ARIC cohort could be secondary to measurement bias or the inability to test for an association with various patterns of alcohol consumption, such as binge drinking, or to the ethnic characteristics of the people investigated in the ARIC cohort. The validity of self-reported data on alcohol consumption has been debated. Underestimation of alcohol intake in the entire cohort or selective underestimation in heavy users could have resulted in underestimation of the level of alcohol consumption associated with incidence of CHD in Black males. Misclassification of light drinkers as rare drinkers because of underestimation of alcohol intake by White women could have resulted in the observed association

between rare consumption of alcohol beverages and lower incidence of CHD. However, there is evidence that the questionnaire administered to the ARIC participants captured their pattern of alcohol consumption reasonably well. For example, there was a consistent association between HDL cholesterol level, a proxy for alcohol use, and category of alcohol intake. In addition, there was overall agreement between classification of participants' alcohol intake at visit 1 and the average of intakes reported at visits 1 and 2 (14). While substantial changes occurred in the levels of drinking, more than 80 percent of participants reported the same drinking status (drinker or nondrinker) at visit 1 and visit 3 (53).

During baseline data collection in the ARIC Study, no information was obtained on the frequency of binge drinking, a pattern associated with higher incidence of CHD (54–57) and total mortality (58). If there were more binge drinkers among Black men, this could explain the higher incidence of CHD in Black men. Another potential explanation for the differential association between alcohol and CHD is the ethnic characteristics and overall pattern of drinking of US citizens, which are very distinct from those of other populations in which the cardioprotective effect of alcohol is relatively more consistent (5, 6). This is also unlikely, since some of the cornerstone descriptions of the cardiovascular protection afforded by alcoholic beverages were obtained in US populations (4, 7, 9).

In conclusion, we have demonstrated inconsistent associations of alcohol consumption and consumption of different types of alcoholic beverages with the incidence of CHD in White and Black participants in the ARIC Study. The inverse association observed in White rare drinkers and the positive association observed in Black men raise the question as to how well we can, in observational studies, control for the confounding effects of lifestyle in the investigation of the association between alcohol consumption and cardiovascular disease, and whether the putative cardioprotective effect of alcohol is real.

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