High-Density Lipoprotein Cholesterol and Alcohol Consumption in US White and Black Adults: Data from NHANES II

ABSTRACT

Objectives. High-density lipoprotein (HDL) cholesterol is known to be positively related to moderate alcohol consumption from studies in selected populations. This study describes the association in a representative sample of the US adult population.

Methods. Stratification and multivariate regression analyses were used to examine HDL cholesterol levels and alcohol consumption.

Results. Fewer women than men reported consumption of alcohol at any frequency. Similar percentages of Whites and Blacks reported alcohol consumption. Age-adjusted mean HDL cholesterol levels were higher among alcohol drinkers than among nondrinkers in all sex–race strata. Mean HDL cholesterol levels of Whites and Blacks of both sexes increased consistently with increased frequency of consumption of beer, wine, and liquor. With age, education, body mass index, smoking, and physical activity controlled for, there were higher age-adjusted HDL cholesterol levels with increasing reported quantities of alcohol consumed. Daily or weekly use of alcohol led to an increase of 5.1 mg/dL in mean HDL cholesterol level, whereas consumption of 1 g of alcohol led to an increase of 0.87 mg/dL.

Conclusion. Even if there is a causal association between alcohol consumption and higher HDL cholesterol levels, it is suggested that efforts to reduce coronary heart disease risks concentrate on the cessation of smoking and weight control. (*Am J Public Health.* 1993;83:811–816)

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Introduction

High-density lipoprotein (HDL) cholesterol is known to be inversely related to coronary heart disease.[1,2] Also, an inverse relationship between moderate alcohol consumption and coronary heart disease has been found in epidemiological studies of diverse design,[3–9] although some studies have reported conflicting results.[10] The Lipid Research Clinics Follow-up Study provided information from a prospective study of more than 7400 men and women in 10 North American populations followed for an average of 8.5 years.[1,5] In that study, moderate alcohol consumption was weakly protective for cardiovascular disease, and the effect of alcohol consumption on cardiovascular disease mortality was partially mediated by the HDL cholesterol increase.

Alcohol consumption has been found to be related to high-density lipoprotein cholesterol in various studies.[6–9,11–22] In the Lipid Research Clinics Prevalence Study,[1,13] the association of alcohol consumption and HDL cholesterol levels was confirmed for Whites. Furthermore, this relationship was consistent for all types of alcoholic beverages, including beer, wine, and liquor. This direct relationship between alcohol consumption and HDL cholesterol has also been discussed in several review articles.[1,4–7,10,23,24]

However, most studies have used data from selected populations, and only a few have provided data on the relationship of HDL cholesterol levels to different types of alcoholic beverages. Moreover, there are few data on the frequency of alcohol use in the United States in recent years, and only a few studies have reported on the effect of alcohol on HDL cholesterol levels in Blacks. The second National Health and Nutrition Examination Survey (NHANES II)[25,26] measured HDL cholesterol levels in a probability sample of the civilian noninstitutionalized population of the United States and thus provides information on the distribution of HDL cholesterol levels for categories of alcohol consumption as well as for selected potential correlates, namely age, sex, and race. The NHANES II also provides data on alcohol use for US adults. The data permit control for other potential confounding factors such as education, body mass index, smoking, and physical activity.

The current report presents data on the relationship between HDL cholesterol and alcohol consumption in this representative sample of the US population.

Population and Methods

The NHANES II, conducted between February 1976 and February 1980 by the National Center for Health Statistics, was a national cross-sectional prob-

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ability survey of the civilian noninstitutionalized population of the United States aged 6 months to 74 years.[25,26] The data and analyses presented here were limited to those examined persons who had HDL cholesterol determinations.

NHANES II consisted of two components: (1) household interviews and (2) physical examinations and interviews in mobile examination centers. Socioeconomic and demographic information for the sample person and the family and a medical history questionnaire were collected in the household interview. The medical examination was conducted according to a standardized protocol and included laboratory tests on whole blood and sera. For each person examined, dietary data, including alcohol consumption, were obtained by means of a 3-month food frequency questionnaire administered by trained dietary interviewers.

In any health examination survey conducted in a manner similar to NHANES, the potential exists for three types of nonresponse: (1) interview nonresponse, (2) examination nonresponse, and (3) item nonresponse (with respect to this research topic, the total cholesterol and HDL cholesterol determinators). Interview nonresponse occurs when the medical history questionnaire is not completed; examination nonresponse occurs when those sample persons who respond to the household questions do not come to the examination center. HDL cholesterol determination nonresponse results when the sample person refuses to have blood drawn, when the venipuncture is unsuccessful, when the vial is lost or destroyed, or when there is insufficient specimen to make the determination. Intense efforts were undertaken during NHANES II to develop and implement procedures that would minimize all types of nonresponse.[26] An analysis of selected health variables of persons who were examined vs those who were not examined (but interviewed) indicated no substantial bias due to non-response.[27] The examined sample has been adjusted to reflect a nationally representative sample.[26] Furthermore, an examination of the characteristics of persons with and without HDL cholesterol determinations such as age, income, education, body mass index, alcohol use, smoking, and physical activity did not detect differences that would suggest a bias in the results.

Determination of Serum Lipids

The National Center for Health Statistics and the National Heart, Lung and Blood Institute agreed to the joint collection, chemical analysis, and processing of NHANES II serum specimens for total cholesterol, HDL cholesterol, and triglycerides. Chemical analyses were provided by the Lipid Research Clinics Laboratory at George Washington University.[28]

Definitions

Frequency of alcohol consumption. The frequency of consumption of beer, wine, and liquor was obtained from a food frequency questionnaire. Participants were asked about their usual consumption of alcoholic beverages during the 3 months that preceded the interview. Consumption was classified as follows: never, seldom (less than once per week), weekly (one to six times per week), or daily (one or more times per day). The data for all types of alcohol—beer, wine, and liquor—were compared. The data were then examined, in regard to each type of alcoholic beverage, for those who reported that they never or seldom consumed alcohol and for those who reported weekly or daily use. This was done to avoid small numbers in cells created by multiple classification for the four sex–race groups. The greatest frequency of reported alcohol use was calculated as a composite measure of habitual (chronic) frequency of alcohol use of any kind.

Alcohol quantity. The number of grams of pure alcohol reported was calculated for all types of alcoholic beverages reported on the food frequency questionnaire. The following conversions were used: The consumption of beer was multiplied by 0.48, which represents the average amount of alcohol in a 12-oz glass of beer. The consumption of wine was multiplied by 0.6, which represents the average amount of alcohol in a 4-oz glass of wine. The consumption of liquor was multiplied by 0.45, which represents the average amount of alcohol in a 1-oz glass of liquor. The total amount of alcohol reported was then defined as the sum of the amount of beer, wine, and liquor reported.

Race. The observed race was recorded as White, Black, or other. Only Whites and Blacks are included in this report.

Educational level. The educational level was determined by the highest grade attended in a public or a private school.

Body mass index (Quetelet index). Body mass index (weight in kg/height in m^2) was calculated as a measure of ponderosity.

Smoking. Examinees were asked (1) whether they had smoked at least 100 cigarettes during their entire life, (2) whether they currently smoked, and (3) the daily number of cigarettes smoked. Individuals who responded positively to questions 1 and 2 were defined as current smokers. Those who responded positively to question 1 but negatively to question 2 were defined as past smokers (of at least 100 cigarettes), and those who responded negatively to question 1 were defined as nonsmokers. For current smokers, the data were further explored in two categories: those who smoked fewer than 21 cigarettes (up to one pack) per day and those who smoked 21 or more cigarettes per day (more than a pack).

Physical activity. Two questions dealt with physical activity. Participants were asked (1) whether they were getting "much exercise," "moderate exercise," or "little or no exercise" for recreation and (2) whether, in their usual day, aside from recreation, they were "very active," "moderately active," or "quite inactive." The higher category of activity in the two questions was then calculated as a composite of activity of any kind.

Statistical Methods

All statistical analyses incorporated both the weights and the complex sample design of the NHANES II. The SURREGR[29] and GENCAT[30,31] programs were used to calculate age-specific and age-adjusted mean HDL cholesterol levels of men and women by race and by reported frequency of alcohol consumption. The age-adjusted means were standardized by the direct method to the 1980 US census population. The first level of analysis consisted of testing the equality of age-specific and age-adjusted mean HDL cholesterol by frequency of any type of alcohol reported for the four sex–race groups. A χ^2 statistic with one degree of freedom was used to test hypotheses of no difference in mean HDL cholesterol at the .05 level of significance. For this report, the use of the word "significant" implies "statistically significant."

Second, Pearson correlation coefficients were estimated to investigate the relationship between HDL cholesterol and calculated daily quantity (in grams) of alcohol. The OSIRIS[32] program was used to estimate correlation coefficients and standard errors. Since the sampling distribution of the correlation coefficients is not normal, Fisher's Z transformation was used to test the hypothesis of zero correlation.

Third, the SURREGR program was used in weighted least squares multiple re-

gression analyses,[29] with HDL cholesterol as the dependent variable, to control for potential confounding. All correlates were included simultaneously in the equation.

Results

Table 1 shows the percentage of examined men and women who reported use of any type of alcohol, by frequency categories of alcohol consumption. Fewer women than men reported the consumption of beer, wine, or liquor. Generally, similar percentages of Whites and Blacks of both sexes reported weekly or daily alcohol consumption. About 24% of the men, 40% of the White women, and 46% of the Black women reported that they had never consumed alcohol. About 17% of White men and about 15% of Black men reported that they seldom used alcohol, compared with 24% to 25% of women. Forty percent to 44% of the men, compared with about 25% to 28% of the women, reported weekly use of alcohol, and about 16% to 20% of the men, compared with 7% of the White women and 5% of the Black women, reported daily use of alcohol. The data for beer, wine, and liquor consumption show similar trends. However, more Black than White women reported beer use, while more White than Black women reported use of wine or liquor.

We have previously reported that the age-adjusted mean HDL cholesterol levels of Whites and Blacks of both sexes increased consistently with increased reported frequency of alcohol consumption.[21] The increases in levels for White and Black men and for White and Black women were 8.3 mg/dL, 9.7 mg/dL, 13.0 mg/dL, and 7.1 mg/dL, respectively. Table 2 shows similar trends for the combined categories of reported never–seldom and weekly–daily consumption of beer, wine, and liquor. The increases in mean HDL cholesterol levels for White men who reported consumption of beer, wine, and liquor were 4.2 mg/dL, 3.7 mg/dL, and 4.0 mg/dL, respectively. The corresponding increases were 5.9 mg/dL, 4.3 mg/dL, and 4.0 mg/dL for Black men; 5.6 mg/dL, 5.5 mg/dL, and 6.3 mg/dL for White women; and 6.1 mg/dL, 8.2 mg/dL, and 0.8 mg/dL for Black women. Similarly, for each of the four sex–race groups, age-adjusted HDL cholesterol levels generally increased with increased reported quantities of alcohol consumed (Table 3), with increases of 6.7 mg/dL, 9.8 mg/dL, 9.4 mg/dL, and 7.0 mg/dL for White and Black men and White and Black women, respectively. The correlation coefficients of HDL cholesterol and grams of alcohol reported were statistically significant for men and White women. They were highest for White women and lowest for Black women.

White men had the lowest age-ad-

TABLE 1—Percentage of Persons 20 through 74 Years of Age by Reported Consumption of Alcohol: US Population, 1976 through 1980

	White Men (n = 3998)	Black Men (n = 457)	White Women (n = 4543)	Black Women (n = 576)
Any alcohol				
Never	23.6	24.8	40.6	46.3
Seldom	16.9	14.7	24.9	23.6
Weekly	39.7	44.1	27.9	24.7
Daily	19.7	16.4	6.6	5.4
Beer				
Never	33.2	33.0	66.7	63.7
Seldom	19.8	18.1	18.0	16.4
Weekly	33.2	36.3	13.2	16.1
Daily	13.9	12.6	2.1	3.8
Wine				
Never	55.8	62.1	59.9	69.4
Seldom	27.1	21.9	24.4	19.7
Weekly	15.0	14.5	13.6	10.1
Daily	2.1	1.5	2.1	0.8
Liquor				
Never	45.8	47.2	57.7	69.5
Seldom	28.0	23.4	24.1	15.8
Weekly	20.2	24.9	15.3	13.8
Daily	6.0	4.6	2.8	0.9

TABLE 2—Age-Adjusted Mean High-Density Lipoprotein Cholesterol Levels (mg/dL) by Frequency of Alcohol Consumption: US Population, 1976 through 1980

	White Men	Black Men	White Women	Black Women
Beer				
Never/seldom	42.5	49.8	52.5	55.1
Weekly/daily	46.7	55.7	58.1	61.2
Wine				
Never/seldom	43.8	51.4	52.6	56.0
Weekly/daily	47.5	55.7	58.1	64.2
Liquor				
Never/seldom	43.4	51.0	52.3	56.2
Weekly/daily	47.4	55.0	58.6	57.0

TABLE 3—Age-Adjusted Mean High-Density Lipoprotein Cholesterol Levels (mg/dL) of Persons Aged 20 through 74 Years Who Reported Alcohol Consumption: US Population, 1976 through 1980

Quartile of Consumption	White Men (n = 2818)	Black Men (n = 318)	White Women (n = 2368)	Black Women (n = 278)
1st	42.8	49.1	51.8	54.0
2nd	44.0	51.7	54.5	57.8
3rd	46.8	54.1	54.3	59.6
4th	49.5	58.9	61.2	61.0
Pearson correlation	.162*	.189*	.224*	.063

Note. Means were age adjusted by the direct method to the 1980 US census population. Pure alcohol consumption in grams was derived from a food frequency questionnaire.
*$P < .001$.

TABLE 4—Multiple Linear Regression Model: Frequency of Consumption

	Estimated Regression Coefficient	95% Confidence Interval	Partial R^2, %
Sex	9.42	8.76, 10.10	9.09
Body mass index	−0.65	−0.72, −0.59	4.18
Alcohol consumption (daily, weekly vs seldom, never)	5.06	4.43, 5.70	2.16
Current smoker	−3.48	−4.04, −2.90	1.57
Race	6.43	5.00, 7.90	1.43
Age, y	0.09	0.08, 0.09	0.61
Education, y	0.14	0.02, 0.26	0.07
Physical activity (very active or moderate vs inactive)	0.65	0.22, 1.09	0.05
Overall R^2 = 19.2% Intercept = 53.8			

Note. High-density lipoprotein cholesterol was a dependent variable; frequency of alcohol consumption and all correlates were included.

TABLE 5—Multiple Linear Regression Model: Quantity of Consumption

	Estimated Regression Coefficient	95% Confidence Interval	Partial R^2, %
Sex	9.40	8.60, 10.20	10.0
Body mass index	−0.75	−0.82, −0.67	3.8
Alcohol use, g	0.87	0.56, 1.19	2.4
Race	6.29	4.81, 7.77	1.3
Current smoker	−3.34	−3.98, −2.69	1.3
Age, y	0.09	0.06, 0.12	0.7
Physical activity (very active vs moderate or inactive)	0.71	0.08, 1.34	0.0
Education, y	0.14	0.01, 0.27	0.0
Overall R^2 = 19.5% Intercept = 42.1			

Note. High-density lipoprotein cholesterol was a dependent variable; quantity of pure alcohol consumption (grams) and all correlates were included.

justed mean HDL cholesterol levels within all categories of frequency and quantity of alcohol consumption. Age-adjusted levels for White women were consistently and significantly higher than those of White men. Blacks had consistently higher age-adjusted levels than Whites, for both men and women.

Multivariate Analyses

Multivariate models were examined next (Table 4).[21] A model with a variable for oral contraceptive use yielded similar results to a model excluding this variable, and the coefficient for oral contraceptive use was not statistically significant. Therefore, the findings for a parsimonious model excluding oral contraceptive use are reported here. Reported weekly or daily consumption of alcohol was positively related to HDL cholesterol levels, independent of the effects of race, sex, body mass index, age, current smoking, and physical activity. In this model, education was not strongly related to HDL cholesterol. Daily or weekly reported use of alcohol led to an increase in mean HDL cholesterol level of about 5.1 mg/dL. The results were similar when a variable comparing those who ever used alcohol with those who never used alcohol was introduced as a measure of alcohol consumption. Similar results were obtained when the quantity of alcohol use (in grams) was substituted for the 3-month frequency data for a subset of alcohol users (Table 5). Alcohol was directly related to HDL cholesterol levels. The regression analyses indicated that consuming 1 g of alcohol led to an increase in mean HDL cholesterol level of 0.87 mg/dL (95% CI = 0.56, 1.19).

Discussion

The present report provides estimates of the relationships of HDL cholesterol and reported beer, wine, and liquor consumption for a representative sample of the US population. HDL cholesterol levels were found to increase with alcohol consumption of any kind in men and women of both races with a dose–response pattern. The consumption of 1 g of alcohol (in any type of alcoholic beverage) led to an increase of 0.87 mg/dL in mean HDL cholesterol level. This relationship was independent of the relationships of HDL cholesterol and age, education, body mass index, cigarette smoking, or physical activity. Furthermore, the distribution of HDL cholesterol in the four sex–race groups confirms previous reports by Tyroler et al.[33] and Heiss et al.[1] and is consistent with previous analyses of the NHANES II data,[21] even after stratification by frequency and quantity of alcohol consumption.

Some limitations of our study must be acknowledged. The data reported here are cross-sectional and do not enable the assessment of long-term effects of alcohol consumption or abstinence on HDL cholesterol. Previous reports have found that increased alcohol consumption is associated with an increase in HDL_3 levels but not HDL_2 levels.[34–37] However, other reports have shown that both are related to alcohol consumption.[38–41] NHANES II did not have information on the types of HDL cholesterol. Similarly, NHANES II did not collect detailed hormone use among women, therefore, gonadal hormones for contraception or other medical purposes could not be completely evaluated in the analyses.

Self-reported alcohol consumption may tend to underestimate actual consumption. However, the findings reported here are consistent for the various alcohol strata and with previous studies. This indicates the lack of differential random misclassification that would tend to diminish the association rather than to create spurious trends. Moreover, previous reports have indicated that the underreporting occurs mainly for problem drinkers, while those who abstain or those who consume alcoholic beverages on only a few occasions probably report their alcohol use accurately.[7,22] About a third of the men and half of the women of both races reported abstaining from alcohol. These data are comparable to data reported by other sources.[42]

Although the effects of moderate alcohol consumption on HDL cholesterol represent a likely mechanism for the inverse association between moderate alcohol consumption and coronary heart disease, alcohol may affect coronary heart disease by other mechanisms, including its effects on fibrinolytic activity, coagulation, blood pressure, coronary vasoreactivity, and sociobehavioral factors.[5]

The beneficial effects of moderate alcohol intake and its potential for lowering the risk of coronary heart disease, have been discussed and debated in several recent articles.[43–50] Even if the effect of moderate alcohol consumption on HDL cholesterol levels or on the occurrence of coronary heart disease is likely to be causal, it is still undesirable to recommend increased alcohol use as a means of reducing the risk of coronary heart disease.[46,48–50] First, it is not clear yet whether manipulation of HDL cholesterol levels would lead to an alteration of coronary heart disease risk. Moreover, heavy alcohol consumption may have a direct toxic effect on the myocardium [8,24,51,52] and may cause paroxysmal arrhythmias or precipitate congestive heart failure.[24,52] Blood pressure also increases with increased consumption of alcohol at about four drinks per day.[3,53,54] In a recent report, the increase in both systolic and diastolic blood pressure was influenced more by the frequency of drinking than by the quantity of alcohol consumed. However, those who drank less often than weekly tended to have lower blood pressure than abstainers or those who drank weekly or more often.[55] In a large prospective study conducted by Colditz, even moderate alcohol intake was found to be related to an increased risk of hemorrhagic stroke.[43]

Non–coronary heart disease mortality rates increase with high alcohol consumption.[10,43–50] Women who consume three to nine drinks per week may be at increased risk of breast cancer.[43] Heavier alcohol consumption of more than two to three drinks per day has been shown to be related to oral and esophageal cancer, cirrhosis, and serious liver diseases.[44] The social cost of increased alcohol consumption, as well as an increase in road accidents, is considerable.[44,56]

In view of the independent effects of smoking and body mass index on HDL cholesterol levels, it is difficult to recommend preventive measures that include alcohol use. Rather, it is suggested that efforts to reduce coronary heart disease risks concentrate on the cessation of smoking, weight control, and physical activity, known to increase HDL cholesterol and enhance health by other means as well. □

References

1. Heiss G, Johnson N, Reiland S, Davis CE, Tyroler HA. The epidemiology of plasma high-density lipoprotein cholesterol levels. The Lipid Research Clinics Program Prevalence Study. Summary. *Circulation.* 1980; 62:116–136.
2. Marmot MG. Alcohol and coronary heart disease. *Int J Epidemiol.* 1984;13:160–167.
3. Flegal KM, Cauley JA. Alcohol consumption and cardiovascular risk factors. *Recent Developments Alcohol.* 1985;3:165–180.
4. Moore RD, Pearson TA. Moderate alcohol consumption and coronary artery disease. A review. *Medicine.* 1986;65:242–267.
5. Criqui MH, Cowan LD, Tyroler HA, et al. Lipoproteins as mediators for the effects of alcohol consumption and cigarette smoking on cardiovascular mortality: results from the Lipid Research Clinics Follow-up Study. *Am J Epidemiol.* 1987;126:629–637.
6. Castelli WP, Gordon T, Hjortland M, et al. Alcohol and blood lipids. The Cooperative Lipoprotein Phenotyping Study. *Lancet.* 1977;2:153–155.
7. Hulley SB, Ashman P, Kuller L, Lasser N, Sherwin R. HDL-C levels in MRFIT. *Lipids.* 1979;14:119.
8. Siscovick DS, Weiss NS, Fox N. Moderate alcohol consumption and primary cardiac arrest. *Am J Epidemiol.* 1986;123:499–503.
9. Ernst N, Fisher M, Smith W, et al. The association of plasma high-density lipoprotein cholesterol with dietary intake and alcohol consumption. The Lipid Research Clinics Program Prevalence Study. *Circulation.* 1980;62(suppl IV):41–52.
10. Colsher PL, Wallace RB. Is modest alcohol consumption better than none at all? An epidemiologic assessment. *Ann Rev Public Health.* 1989;10:203–219.
11. Kaelber C, Mills G. Alcohol consumption and cardiovascular diseases: introductory remarks. *Circulation.* 1981;64(suppl III): 1–6.
12. Klats AL, Friedman GD, Siegelaum AB. Alcohol use and cardiovascular disease: the Kaiser-Permanente experience. *Circulation.* 1981;64(suppl III):32–41.
13. Gordon T, Ernst N, Fisher M, Rifkind BM. Alcohol and high-density lipoprotein cholesterol. *Circulation.* 1981;64(suppl III):63–67.
14. Brenn T. The Troms Heart Study: alcoholic beverages and coronary risk factors. *J Epidemiol Community Health.* 1986;40: 249–256.
15. Diehl AK, Fuller JH, Mattock MB, Salter AM, el-Gohari R, Keen H. The relationships of high density lipoprotein subfractions to alcohol consumption, other lifestyle factors, and coronary heart disease. *Atherosclerosis.* 1988;68:145–153.
16. Patterson CC, McCrum E, McMaster D, Kerr M, Sykes D, Evans AE. Factors influencing total cholesterol and high density lipoprotein cholesterol concentrations in population at high coronary risk. *Acta Med Scand.* 1988;728(suppl):150–158.
17. Meilahn EN, Kuller LH, Stein EA, Caggiula AW, Matthews KA. Characteristics associated with apoprotein and lipoprotein lipid levels in middle-aged women. *Atherosclerosis.* 1988;8:515–520.
18. Miller NE, Bolton CH, Hayes TM, et al. Association of alcohol consumption with plasma high density lipoprotein cholesterol and its major subfractions: the Caerphilly and Speedwell Collaborative Heart Disease Studies. *J Epidemiol Community Health.* 1988;42:220–225.
19. Fraser GE, Babaali H. Determinants of high density lipoprotein cholesterol in middle-aged Seventh-Day Adventists men and their neighbors. *Am J Epidemiol.* 1989;130: 958–965.
20. Kesteloot H, Geboers J, Joossens JV. On the within-population relationships between nutrition and serum lipids: the B.I.R.N.H. study. *Eur Heart J.* 1989;10: 196–202.
21. Linn S, Fulwood R, Rifkind B, et al. High density lipoprotein cholesterol levels among U.S. adults by selected demographic and socioeconomic variables. The Second National Health and Nutrition Examination Survey 1976–1980. *Am J Epidemiol.* 1989;129:281–294.
22. Giovannucci E, Colditz G, Stampfer MJ, et al. The assessment of alcohol consumption by a simple self-administered questionnaire. *Am J Epidemiol.* 1991;133:810–817.
23. Hulley SB, Gordon S. Alcohol and high-density lipoprotein cholesterol. Causal inference from diverse study designs. *Circulation.* 1981;64(suppl III):57–63.
24. Regan TJ. Alcohol and the cardiovascular system. *JAMA.* 1990;264:377–381.
25. *Total Serum Cholesterol Levels of Adults 20–74 Years of Age, United States, 1976–1980.* Washington, DC: US Dept of Health and Human Services; 1986. DHHS publication PHS 86-1686.
26. National Center for Health Statistics, Engel A, Massey J, Maurer K. Plan and operation of the Second National Health and Nutrition Examination Survey 1976–1980. *Vital Health Stat [1].* 1981;15. DHEW publication PHS 81-1317.
27. Forthofer RN. Investigations of nonresponse bias in NHANES II. *Am J Epidemiol.* 1983;117:507–515.
28. *Manual of Laboratory Operations, Volume 1, Lipid Research Clinics Program, Lipid and Lipoprotein Analysis.* Washington, DC: US Dept of Health, Education, and Welfare; 1974. DHEW publication NIH 75-628.
29. Holt M. *SURREGR—Standard Errors of Regression Coefficient from Sample Survey Data.* Research Triangle Park, NC: Research Triangle Institute; 1982.
30. Koch GG, Freedman DH, Freedman JL. Strategies in the multivariate analysis of data from complex surveys. *Int Stat Rev.* 1975;43:59–78.
31. Landis JR, Stanish WM, Freedman JL, Koch GG. A computer program for the generalized chi-square analysis of categorical data using weighted least squares (GENCAT). *Computer Programs Biomed.* 1976;6:196–231.
32. Survey Center Computer Support Group. *OSIRIS IV User's Manual.* Ann Arbor, Mich: Institute for Social Research; 1979.

33. Tyroler HA, Glueck CJ, Christensen B, Kwiterovich PO. Plasma high-density lipoprotein cholesterol comparisons in Black and White populations. The Lipid Research Clinics Program Prevalence Study. *Circulation.* 1980;62:99–107.
34. Eichner ER. Alcohol versus exercise for coronary protection. *Am J Med.* 1985;79: 231–240.
35. Salonen JT, Haynen H, Leino U, Kostianianen E, Sahi T. Relation of alcohol, physical activity, dietary fat and smoking to serum HDL-C and total cholesterol in young Finnish men. *Scand J Soc Med.* 1985;13: 99–102.
36. Robinson D, Ferns GA, Bevan EA, Stock J, Williams PT, Galton DJ. High density lipoprotein subfractions and coronary risk factors in normal men. *Atherosclerosis.* 1987;7:341–346.
37. Dai WS, LaPorte RE, Hom DL, et al. Alcohol consumption and high density lipoprotein cholesterol concentration among alcoholics. *Am J Epidemiol.* 1985;122:620–627.
38. Masarei JR, Puddey IB, Rouse IL, Lynch WJ, Vandongen R, Beilin LJ. Effects of alcohol consumption on serum lipoprotein-lipid and apolipoprotein concentrations: results from an intervention study in healthy subjects. *Atherosclerosis.* 1960;60:799–807.
39. Meilhan EN, Kuller LH, Stein EA, Caggiula AW, Matthew KA. Characteristics associated with apoprotein and lipoprotein lipid levels in middle-aged women. *Arteriosclerosis.* 1988;8:515–520.
40. Lupien PJ, Moorjani S, Jobin J, et al. Smoking, alcohol consumption, lipid and lipoprotein levels. *Can J Cardiol.* 1988;4: 102–107.
41. Diehl AK, Fuller JH, Mattock MB, Salter AM, el-Hohari R, Keen H. The relationship of high density lipoprotein subfractions to alcohol consumption, other lifestyle factors and coronary heart disease. *Atherosclerosis.* 1988;69:145–153.
42. Williams GD, Dufour M, Bertolucci D. Drinking levels, knowledge, and associated characteristics, 1985 NHIS findings. Public Health *Rep.* 1986;101:593–598.
43. Colditz GA. A prospective assessment of moderate alcohol intake and major chronic diseases. *Ann Epidemiol.* 1990;1:167–177.
44. Boffetta P, Lawrence G. Alcohol drinking and mortality among men enrolled in an American Cancer Society prospective study. *Epidemiology.* 1990;1:342–348.
45. Ellison RC. Cheers! *Epidemiology.* 1991; 1:337–339.
46. Blackburn H, Wagenaar A, Jacobs DR Jr. Alcohol: good for your health? *Epidemiology.* 1991;2:230–231.
47. Ellison RC. All things in moderation. *Epidemiology.* 1991;2:232–233.
48. Ferrence RG, Kozlowski LT. Alcohol drinking and mortality. *Epidemiology.* 1991;2:211–212.
49. Jackson R, Scragg R, Beaglehole R. Alcohol consumption and risk of coronary heart disease. *Br Med J.* 1991;303:211–216.
50. Marmot M, Brunner E. Alcohol and cardiovascular disease: the status of the U shaped curve. *Br Med J.* 1991;303:565–568.
51. Regan TJ, Haider B. Ethanol abuse and heart disease. *Circulation.* 1981;64(suppl III):14–19.
52. Kagan A, Yano K, Rhodes GG, McGee DL. Alcohol and cardiovascular disease: the Hawaiian experience. *Circulation.* 1981;64(suppl III):27–31.
53. Wallace RB, Lynch CF, Pomehn PR, Criqui MH, Heiss G. Alcohol and hypertension: epidemiologic and experimental considerations. The Lipid Research Clinics Program. *Circulation.* 1981;64(suppl III): 41–47.
54. Haskell WL, Camargo C Jr, Williams PT, et al. The effect of cessation and resumption of moderate alcohol intake on serum high-density-lipoprotein subfractions. A controlled study. *N Engl J Med.* 1984;310: 805–810.
55. Russel M, Cooper L, Frone M, Welte JW. Alcohol drinking patterns and blood pressure. *Am J Public Health.* 1991;81:452–457.
56. Shoenborn CA. *Exposure to Alcoholism in Family. United States 1988.* Washington, DC: US Dept of Health and Human Services; 1991:205.

Erratum

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Because an error was made in weighting data in Tables 1 and 3, the "Hours Missed by Those with Missed Hours" means and standard errors in both tables are incorrect. Other data and the paper's conclusions are not affected. For a revised reprint, contact Helen C. Gift, PhD, National Institute of Dental Research, Westwood Building, Room 536, National Institutes of Health, Bethesda, MD 20892.