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Alcohol Use and Mortality from Coronary Heart Disease: The Role of High-Density Lipoprotein Cholesterol

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■ *Objective:* To study the association between alcohol consumption and death from coronary heart disease and to determine the extent to which the association can be explained by the high-density lipoprotein (HDL) cholesterol level.

■ *Design:* A cohort study involving men enrolled in the Multiple Risk Factor Intervention Trial (MRFIT).

■ *Setting:* Community-based study.

■ *Participants:* Men (n = 11 688) at high risk for developing coronary heart disease but without clinical evidence of it. More than 90% of the men were white, and the average age was 46 years. Five percent of the men abstained from alcohol during the trial, 81% consumed fewer than 21 alcoholic drinks per week, and 14% consumed more than 21 alcoholic drinks per week.

■ *Measurements:* Average alcohol intake over 7 years was calculated for MRFIT participants who were alive at the end of the trial and who had at least three follow-up records of alcohol consumption. Post-trial mortality during a 3.8-year period was assessed.

■ *Results:* The adjusted relative risk for death from coronary heart disease for each increase of 7 drinks per week was 0.89 (95% CI, 0.80 to 1.00), with an apparent dose-response relationship. The average HDL level was associated with the average alcohol intake in a least-squares regression model ($\beta = -0.0074$; $P < 0.01$). When the average HDL level was included in the proportional hazards model for mortality from coronary heart disease, the absolute value of the coefficient for average drinks per week declined 45%, yielding an adjusted relative risk for each additional 7 drinks per week of 0.94 (CI, 0.84 to 1.05).

■ *Conclusion:* In middle-aged men who are light to moderate drinkers, the inverse association between alcohol consumption and death from coronary heart disease can be explained, in large part, by the HDL cholesterol level, which increases with alcohol consumption. However, alcohol consumption cannot be recommended because of the known adverse effects of excess alcohol use.

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Many epidemiologic studies have shown that light to moderate alcohol intake is associated with lower mortality from coronary heart disease when compared with abstention from alcohol (1-7). However, the apparent advantage of moderate drinking still remains controversial.

In most studies, men who do not drink are used as a standard against which to measure the effects of alcohol consumption. When nondrinkers serve as the comparison group, two problems should be addressed. First, the nondrinkers may include both lifelong teetotalers and ex-drinkers. Some studies have shown ex-drinkers to be at very high risk for coronary heart disease (8-11). A possible explanation for this phenomenon is that ex-drinkers may stop drinking alcohol because of declining health. Therefore, it is important to differentiate ex-drinkers from teetotalers in an analysis of alcohol consumption and coronary heart disease. Second, alcohol use, like many other social behaviors, is a dynamic process and may change over time. Nondrinkers may become drinkers, and heavy drinkers may become moderate drinkers or abstainers. Many epidemiologic studies of alcohol intake and death from coronary heart disease (3-6, 12-14) have based the measure of alcohol consumption on a single recollection by the individual subject, a method that does not reveal changes in alcohol drinking behavior over time. Because there may be considerable intraindividual variability in alcohol intake over time, data based on a single recollection may seriously impair the analysis and interpretation of results. To address these two problems, data on alcohol consumption should be collected over time.

An analysis of the association between alcohol and death from coronary heart disease also requires reliable measures of possible confounding variables. A strong association exists between alcohol consumption and cigarette smoking (4, 6), and the relation between alcohol intake and blood pressure should also be considered, because alcohol intake increases blood pressure (15), and increased blood pressure is associated with death from coronary heart disease (16). Among other differences between drinkers and nondrinkers, diet has also been proposed as a possible confounder (17, 18).

The Multiple Risk Factor Intervention Trial (MRFIT) has provided one of the largest available databases for studying the association between alcohol intake and

mortality from coronary heart disease. The database is especially suitable for several reasons. The trial enrolled men at high risk for coronary heart disease but excluded men with definite evidence of such disease, thus providing a pool of participants more likely to develop the disease under study without introducing the possibility of baseline illness among alcohol abstainers. Persons believed to be consuming excessive amounts of alcohol were also excluded (although no standard cutoff level was defined in terms of number of drinks per week), thus restricting the study to the association between coronary heart disease and light to moderate alcohol consumption. At screening and at six subsequent annual visits, participants were asked about both their drinking habits and other factors (such as diet and smoking) that could confound the association between alcohol and coronary heart disease, thus providing extensive information over time.

The results from many studies, including MRFIT, have suggested a direct association between alcohol intake and plasma high-density lipoprotein (HDL) cholesterol levels (19-21). Further, the HDL cholesterol level was inversely related to mortality from coronary heart disease in MRFIT (22). Our study examines the association between alcohol intake and death from coronary heart disease and the extent to which the association can be explained by the HDL level.

Methods

Participants

The design and methods of the MRFIT have been reported previously (23, 24). Briefly, the MRFIT was a primary prevention trial to determine the effects of multifactor intervention on death from coronary heart disease in a population of high-risk men randomly assigned either to a special intervention program or to their usual sources of medical care in the community. Altogether, 12 866 men 35 to 57 years of age enrolled in the trial from December 1973 to February 1976. The men were classified at a first screening examination as high risk based on serum cholesterol level, diastolic blood pressure, and daily cigarette consumption. Eligibility was based on a risk score derived from the Framingham Heart Study; men were eligible for participation if their risk score was in the upper 15% (later changed to the upper 10% after one third of the screening had been completed). Men with clinical evidence of coronary heart disease were excluded from the trial. Except where noted, men were excluded from this analysis if they were missing three or more annual records of alcohol consumption or if they died during the trial. These exclusions left 11 688 men in the analysis group.

Data Collection and Measurements

Participants were asked about their alcohol consumption at the second screening visit and at every annual visit through 72 months. Nutritionists asked how many times per week (on average) the participant consumed beer, wine, whiskey, or other liquor, and how many alcoholic drinks were consumed in a usual drinking day. (No information was available to determine the type of alcoholic beverage usually consumed.) However, participants were not asked at the screening visit whether they were ex-drinkers. In our study, alcohol intake was measured as the reported number of drinks of alcoholic beverages consumed per week. Participants were retrospectively classified by average alcohol intake during the trial (baseline and six annual visits).

From the time of randomization through 28 February 1982 when intervention and active follow-up ended, a mortality review committee classified specific causes of death by reviewing hospital records, autopsy reports, and information from physicians and next-of-kin. From the end of the trial in March 1982 through December 1985, vital status was determined using the National Death Index and information from the Social Security Administration (23). Two trained nosologists coded each death certificate, and disagreements were adjudicated by a third nosologist. Deaths from coronary heart disease were identified by codes 410 through 414 and 429.2 from the International Classification of Diseases, Ninth Revision (ICD-9). Mortality ascertainment through December 1985 is estimated to be essentially 100% complete.

Other methods of data collection used during the trial included a demographic and behavioral questionnaire, medical history and examination, and hematologic and biochemical testing, including plasma lipid fractionation. Data on HDL subtypes were not available. Central laboratory methods for serum and plasma determinations, methods for blood pressure measurements, and the dietary assessment method have been described previously (22).

Statistical Analysis

Tests for interaction showed that the associations under examination were similar for the special-intervention and usual-care groups. Therefore, all results derive from an analysis of the two study groups together. Cox proportional hazards models were used to investigate the associations among alcohol intake, HDL level, and death from coronary heart disease. Alcohol intake was treated both as a continuous variable and as a categorical variable. (Categories were as follows: 0 drinks per week; > 0 but ≤ 7 drinks per week; > 7 but ≤ 14 drinks per week; > 14 but ≤ 21 drinks per week; and > 21 drinks per week.) Because few participants reported consuming more than 21 drinks per week, further division of the category of highest alcohol consumption yielded unstable results.

Death from coronary heart disease was examined for a 3.8-year period beginning at the end of the trial (March 1982) and continuing through December 1985. Mortality during this period was examined for its association with the average values for alcohol intake and HDL level, where the averages were calculated from values obtained at baseline and at six annual visits. The models were adjusted for both the screening and average values (from the first through the sixth annual visit) of serum cholesterol and number of cigarettes smoked per day, as well as for age. No adjustment was made for blood pressure.

Univariate and multivariate linear regression, analysis of variance, and Pearson correlation coefficients were used to assess the association between the average alcohol intake and the average HDL level. To estimate the extent to which the association between alcohol intake and coronary heart disease mortality could be attributed to the HDL level, the coefficients for drinks per week from proportional hazards models with and without adjustment for the HDL level were compared.

Results

Participants

Of the 12 866 men enrolled in the trial, 12 860 had a record of alcohol consumption at baseline. In addition, 11 688 men survived through the end of the trial and had at least three records for alcohol consumption during the trial. Except where noted, this latter group was used for the analysis. The joint distribution of alcohol intake at baseline and the average intake for years 1 through 6 of the trial is given in Table 1. Of the 874 participants who reported abstinence at baseline, 596 (68.2%) reported not drinking at every subsequent annual visit. Overall, the average level of alcohol consumption during the trial was the same as at baseline for 55.4% of the men, was less than at baseline for 26.8% of the men, and was greater than at baseline for

Table 1. Distribution of Reported Weekly Alcohol Consumption at Baseline and Average for Years 1 through 6 of the Trial*

Reported Number of Alcoholic Drinks per Week at Baseline	MRFIT Participants, *n*					
	Average Reported Number of Alcoholic Drinks per Week for Years 1 through 6 of the Trial					
	0	≤ 7	≤ 14	≤ 21	> 21	Total
None	596	229	29	13	7	874
1 to 7	225	3345	657	112	49	4388
8 to 14	19	996	1066	411	190	2682
15 to 21	15	326	664	502	373	1880
22 or more	32	109	348	402	969	1860
Total	887	5005	2764	1440	1588	11 684

* Baseline data were unavailable for four men included in the analysis; these men are not included in this table. MRFIT = Multiple Risk Factor Intervention Trial.

17.7% of the men. Of the 10 810 men who reported drinking at baseline, 2170 (20.0%) reported sustained abstinence at some point in the trial (data not shown).

Characteristics of the study participants grouped by alcohol consumption during the trial are summarized in Table 2. The average age was 46.4 years; approximately 7% were black, 89% were married, 84% were high school graduates, and 28% were college graduates. Sixty-two percent of the study population smoked cigarettes (the percentage of smokers among the lighter drinkers and teetotalers was considerably smaller), and 62% of the men were hypertensive.

Alcohol Intake, HDL Cholesterol Level, and Diet

The association between alcohol intake and HDL level was examined in three ways. First, the association was examined using data from the visits at baseline; data from the annual visits at years 2, 4, and 6; and the average values from baseline and years 1 through 6 of the trial. Least-squares regression coefficients for the HDL level as a function of number of alcoholic drinks per week ranged from 0.0056 at baseline to 0.0075 when averaged over the trial, and all were statistically significant at $P < 0.001$.

Least-squares regression coefficients for change in HDL level as a function of change in the number of alcoholic drinks consumed per week were calculated for change from baseline to year 2, year 4, and year 6. The regression coefficients were 0.0025, 0.0027, and 0.0030, respectively, and all were statistically significant at $P < 0.001$.

Finally, both an adjusted and unadjusted average for the HDL level was computed in each category of average alcohol intake. Adjustment was made for the average number of cigarettes smoked per day and the average weight from baseline through the sixth annual visit. As shown in Figure 1, both unadjusted and adjusted levels of HDL increased as the average number of drinks per week increased.

We also examined the associations between alcohol intake and dietary intake of total fats, polyunsaturated fats, and cholesterol, nutrients that are known to be associated with death from coronary heart disease. After adjusting for caloric intake, no significant association was found between the consumption of these nutrients and alcohol (data not shown).

Alcohol Intake and Death from Coronary Heart Disease

Table 3 shows the association between average alcohol intake from baseline through year 6 of the trial and death from coronary heart disease during a 3.8-year period after the end of the trial. The unadjusted rate of death from coronary heart disease per 1000 person-years declined from 5.29 for nondrinkers to 2.58 for those consuming more than 21 drinks per week. Inter-

Table 2. Characteristics of Participants Grouped by Alcohol Consumption during the Trial

Variable	Men Abstaining throughout Trial (*n* = 596)	Men Consuming 14 or Fewer Drinks per Week* (*n* = 7910)	Men Consuming More than 14 Drinks per Week* (*n* = 3182)	Men with at Least Three Annual Alcohol Records Who Were Alive on 2/28/82 (*n* = 11 688)
Age, *y*	47.9	46.4	45.9	46.4
Diastolic blood pressure, *mm Hg*	90.1	90.6	91.0	90.7
Hypertension, %†	64.4	62.3	61.9	62.3
Serum cholesterol, *mmol/L (mg/dL)*	6.6 (254.0)	6.6 (254.5)	6.5 (251.5)	6.6 (253.7)
Plasma HDL cholesterol, *mmol/L [mg/dL]*‡	1.0 (38.0)	1.1 (40.8)	1.2 (46.2)	1.1 (42.1)
Body mass index, kg/m^2	27.7	27.9	27.2	27.7
Cigarette smokers, %	50.5	59.0	72.8	62.3
Cigarettes per day	37.5	32.7	35.7	33.9
Black, %	8.4	8.2	4.5	7.2
Married, %	89.5	90.3	85.8	89.0
High income, %§	4.3	8.3	9.5	8.4
Low income, %§	17.8	11.2	11.0	11.4
High school graduates, %	75.0	85.2	84.5	84.5
College graduate, %	18.1	28.9	27.3	27.9

* Average consumption from baseline through year 6 of the trial.
† Hypertension was defined by a diastolic blood pressure 90 mm Hg or more at baseline or by self-reported use of antihypertensive medication.
‡ HDL = high-density lipoprotein.
§ High income refers to those reporting a total family income of more than $35 000 before taxes and deductions. Low income refers to those reporting a total family income of $11 999 or less before taxes and deductions.

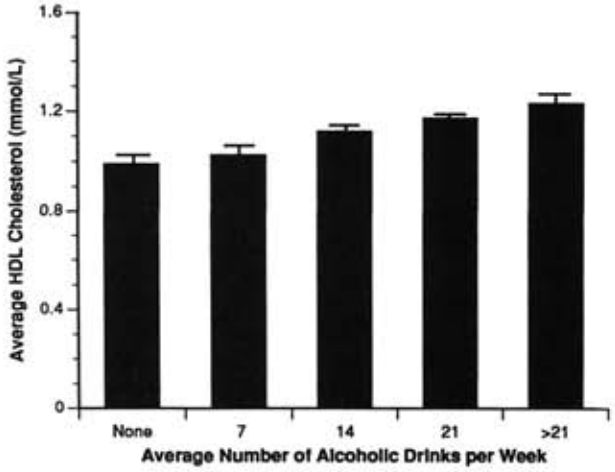

Figure 1. Average high-density lipoprotein cholesterol level and alcohol consumption. HDL = high-density lipoprotein.

mediate rates were found for lower levels of consumption; however, the relative risks and mortality rates were similar in the two categories encompassing consumption of 1 to 14 drinks per week and were similar in the two categories encompassing consumption of more than 14 drinks per week. Only three deaths (1.01/1000 person-years) from coronary heart disease occurred among baseline abstainers who later reported drinking (data not shown), and all three occurred among men who consumed, on average, fewer than 7 drinks per week. The adjusted risk for death from coronary heart disease also declined as alcohol consumption increased; for each increase of an average of 7 drinks per week, the relative risk for death from coronary heart disease was 0.89 ($P = 0.043$). When the model was adjusted for the HDL cholesterol level, risk for death from coronary heart disease continued to decline with alcohol consumption, but the gradient was not as steep, and the coefficient for alcohol in the proportional hazards model was no longer statistically significant (relative risk for increase of 7 drinks per week, 0.94; $P > 0.2$) (Figure 2).

To assess the strength of this association over a longer follow-up period with more deaths, models were developed to test the association between baseline alcohol consumption and death from coronary heart disease from randomization through 31 December 1985, an average of 10.5 years. Data used in this analysis were from 12 860 men, 430 of whom died from coronary heart disease. The unadjusted rate of death was 3.95/1000 person-years for nondrinkers, declining steadily to 2.58/1000 person-years for those consuming more than 21 drinks per week. For each increase of 7 drinks per week, the relative risk for death from coronary heart disease was 0.94 ($P = 0.034$) when the HDL level was not included in the model and 0.96 ($P > 0.2$) when the HDL level was included in the model. Thus, the association between baseline alcohol consumption and subsequent mortality from coronary heart disease was generally weaker than the association between alcohol intake averaged over 7 years and death in the 3.8-year period after the end of the trial, despite the larger number of participants and the greater number of deaths in the former analysis.

Discussion

Most epidemiologic studies of alcohol consumption and death from coronary heart disease estimated the amount of alcohol consumption using only a single recollection (3-6, 12, 13, 25). Only the Chicago Western Electric Company study (11) used data from three surveys to determine alcohol consumption at baseline. In our study, we used data from six consecutive annual visits plus a screening visit. Thus, we could differentiate persistent nondrinkers from those who quit or started drinking alcohol during the 7-year period. Even though we did not know a participant's history of alcohol drinking before the 7-year trial period, we could identify those who were nondrinkers for at least 7 years.

Of the 874 participants who were classified as nondrinkers at screening, 278 reported drinking during the trial. Therefore, 31.8% of nondrinkers at screening were incorrectly classified, if "nondrinking" is defined as

Table 3. Average Alcohol Consumption and Post-Trial Mortality from Coronary Heart Disease during a 3.8-Year Period*

Reported Number of Alcoholic Drinks per Week Averaged from Baseline through Year 6 of the Trial	Men	Average Number of Drinks per Week	Deaths from CHD	CHD Death Rate†	Adjusted Relative Risk (95% CI)‡	Adjusted Relative Risk with HDL Level in the Model (95% CI)
	← *n* →					
0	596	0	15	5.29	1.00	1.00
≤ 7	4996	3.4	85	3.55	0.74 (0.42 to 1.29)	0.76 (0.43 to 1.32)
≤ 14	2914	10.3	51	3.68	0.75 (0.42 to 1.35)	0.84 (0.47 to 1.52)
≤21	1542	17.2	19	2.60	0.50 (0.25 to 1.00)	0.59 (0.30 to 1.19)
> 21	1640	31.1	20	2.58	0.51 (0.26 to 1.00)	0.63 (0.31 to 1.26)
Relative risk for each additional 7 drinks per week in adjusted proportional hazards models					0.89 $P = 0.04$	0.94 $P > 0.2$

* Data are from the Multiple Risk Factor Intervention Trial. CHD = coronary heart disease; HDL = high-density lipoprotein.
† Calculated per 1000 person-years.
‡ Proportional hazards models were adjusted for age at randomization, serum cholesterol, and number of cigarettes per day at initial screening, and average values for serum cholesterol and number of cigarettes per day, where the average is calculated years 1 through 6 of the trial.

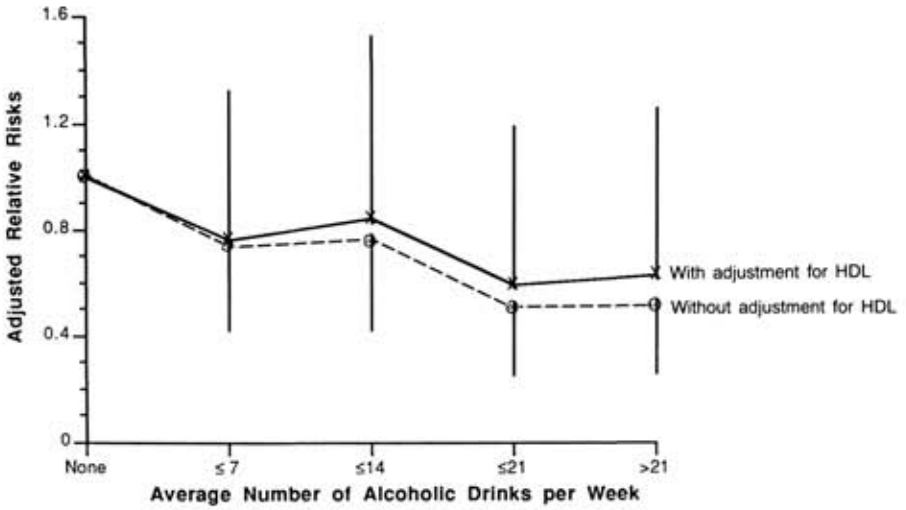

Figure 2. Relation between the risk for death from coronary heart disease and alcohol intake. The upper bound of the 95% CI (indicated by vertical black lines) is the upper bound for the model with adjustment for the high-density lipoprotein (HDL) cholesterol level. The lower bound of the 95% CI is the lower bound of the model without adjustment for the HDL level.

complete abstinence. Another advantage of using serial data on alcohol intake is that the average number of drinks per week during the trial period probably reflects a more typical lifetime drinking pattern, which is more likely to be associated with mortality, than does drinking behavior in a single year. This may explain why post-trial death from coronary heart disease was more strongly associated with the average number of drinks per week (relative risk, 0.89, $P = 0.043$) than with the baseline number of drinks per week (relative risk, 0.91, $P = 0.052$ [data not shown]) when using the same set of baseline covariates.

The MRFIT participants were selected on the basis of their serum cholesterol level, diastolic blood pressure, and daily cigarette smoking. Special intervention in one half of the MRFIT men consisted of dietary advice to lower blood cholesterol levels, counseling to achieve smoking cessation, and drug treatment to control hypertension. Therefore, it might be argued that all models should be adjusted for levels of the three variables used to select participants and for the average value for these variables over the trial period.

All models in this study were adjusted for two of the three screening variables: serum cholesterol and number of cigarettes per day. It is well known, however, that alcohol consumption is associated with increased blood pressure, which in turn is associated with increased incidence of death from coronary heart disease. These associations were also found in the present study. Therefore, the inclusion of diastolic blood pressure in models intended to illuminate the association between alcohol and death from coronary heart disease would tend to exaggerate alcohol's apparently protective effect, which in fact was the case: The relative risk for 7 additional drinks per week when blood pressure was included in the model and the HDL level was not was 0.92 for the 10.5-year analysis (compared with 0.94 without adjustment for blood pressure) and 0.88 for the 3.8-year post-trial analysis (compared with 0.89 without adjustment for blood pressure) (*see* Table 3).

Possible confounding from dietary habits in studies of alcohol and coronary heart disease has been proposed (17, 18). The observed association between alcohol and death from coronary heart disease, however, could not be attributed to an association between alcohol consumption and dietary habits, because no significant association was found between alcohol intake and consumption of dietary fats. Because other investigators found no statistically significant difference between smokers and nonsmokers in the association between alcohol and death from coronary heart disease (26), we did not assess the possible confounding effect of cigarette smoking.

Light to moderate drinking was inversely related to death from coronary heart disease in our study. This finding is consistent with many prospective studies (3-7). Several studies that included heavy drinkers have shown a J- or U-shaped relation between alcohol consumption and death from coronary heart disease (4, 6, 14). No such association was evident in this study, perhaps because individuals believed to be drinking excessively were excluded from the MRFIT. Curiously, mortality rates were lowest among baseline abstainers who later reported drinking, although this group included only 278 men, only 3 of whom died from coronary heart disease, and thus the rates are quite unstable.

Findings regarding the level or range of alcohol consumption that carries a minimum risk for death from coronary heart disease have been inconsistent. Klatsky and coworkers (14) showed the lowest risk (relative risk, 0.60) among those consuming 3 to 5 drinks per day, and Dyer and coworkers (11) showed a minimum risk for those consuming 4 to 5 drinks per day (relative risk, 0.67). In the Framingham Heart Study (25), the incidence of death from coronary heart disease in men was reduced by about 40% in nonsmokers and by about 50% in heavy smokers when alcohol consumption was 10 to 15 oz per week (approximately 2.4 to 3.6 drinks per day). But Klatsky and coworkers (6) found that risks for drinkers relative to nondrinkers were significant only at the level of fewer than 2 drinks per day (relative risk, 0.61) and Colditz (5) reported the lowest risk (relative risk, 0.3) at the level of 0.1 to 8.9 g/d (approximately 0.007 to 0.65 drinks per day).

In our study, the relative risk for death from coronary heart disease among those consuming 1 to 14 drinks per week (fewer than 2 drinks per day) was lower than that

of nondrinkers, but the difference was not statistically significant. Compared with nondrinkers, the point at which the relative risk showed statistical significance was when the number of drinks per week exceeded 14 (more than 2 drinks per day). This finding is consistent with those of Klatsky and coworkers (14), Dyer and coworkers (11), and the Framingham Heart Study (4).

Many studies have shown consistent correlations between alcohol consumption and HDL levels (19-21, 27-29). Two of these studies analyzed data from the MRFIT (19, 20). Using data on 1084 men who participated in the MRFIT San Francisco clinic, Hulley and coworkers (20) demonstrated that levels of HDL and alcohol consumption were independently associated. Kuller and coworkers (19) showed both cross-sectional and longitudinal associations between alcohol consumption and HDL levels in the entire MRFIT cohort followed for 48 months. In our study, we used MRFIT data for the whole trial and confirmed the strong association between alcohol intake and HDL level.

If HDL is a mechanism underlying the relation between alcohol intake and death from coronary heart disease, then such a relation should be reduced or eliminated in a study controlling for the effect of the HDL level. In Cox proportional hazards models for death from coronary heart disease, the absolute value for the estimated coefficient for average number of drinks per week declined 45% (with a corresponding increase in adjusted relative risk from 0.89 to 0.94) and became statistically nonsignificant when HDL was added to the model. This finding is strong evidence that HDL is a causal mechanism that explains, at least in part, the apparently beneficial effect of alcohol. Other possible mechanisms still need to be investigated because HDL probably does not account for the entire association between alcohol and death from coronary heart disease.

Criqui and colleagues (12, 13) analyzed data from the Lipid Research Clinics Follow-Up Study and suggested that the HDL level was the mediating variable for about half of alcohol's protective effect against death from coronary heart disease. These investigators drew this conclusion after examining the change in relative risks of alcohol before and after adjustment for the HDL level. In their analysis, however, the relative risk associated with alcohol was not statistically significant even before adjustment for the HDL level. This lack of significance might be due to the small number of deaths from coronary heart disease (78 deaths among 4105 subjects). Langer and coworkers (7) drew a similar conclusion after analyzing data from the Honolulu Heart Study; however, these investigators examined the effect of alcohol on the incidence of coronary heart disease rather than on mortality from coronary heart disease. In our study, approximately 45% of the association between average alcohol consumption and death from coronary heart disease may be attributable to HDL. Thus, the findings and conclusions of these studies are consistent. Furthermore, the Lipid Research Clinics population included both men and women, and the Honolulu population consisted of Japanese-American men, facts that strengthen the generalizability of the results from our study of MRFIT men, more than 90% of whom were white.

In conclusion, the findings from this large cohort with long-term follow-up add to the likelihood that light to moderate alcohol use confers some protection against coronary heart disease. Nevertheless, although this relation may have application when advising selected patients, a public health policy to encourage nondrinkers to consume alcohol or occasional drinkers to increase alcohol consumption cannot be recommended, because of the known manifold adverse consequences of excess alcohol use.

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Millikan's wife was passing through the hall of the famous physicist's home when she overheard their maid on the telephone.

"Yes, this is where Dr. Millikan lives," she heard the maid say, "but he's not the kind of doctor that does anybody any good."

The Little, Brown Book of Anecdotes
Clifton Fadiman, Editor
Little, Brown & Co., 1985, p. 401

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