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DAILY ALCOHOL CONSUMPTION AND FATAL CORONARY HEART DISEASE[1]

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For a series of 568 married white men who died from coronary heart disease (CHD) and an equal number of matched controls, information was collected on a large number of variables, including daily alcohol consumption. The crude matched pair ratio estimate for any versus no daily drinking was 0.6 (95% confidence limits 0.4 to 0.7). After controlling for additional confounding variables the risk ratio for any versus no daily alcohol consumption was 0.6 (0.5–0.8). This preventive effect was restricted to light drinkers, defined as those who drank ≤59.2 ml (2 oz) of alcohol daily. These data provide strong evidence against a causal role of daily alcohol consumption in fatal CHD and are consistent with a small preventive effect of any versus no daily drinking which is attributable only to light but not heavy drinkers.

alcohol drinking; coronary disease

Conflicting results have been reported about the possible role of daily alcohol consumption in coronary heart disease (CHD). The data from three prospective cohort studies, the Framingham Heart Study (1), the Kaiser Foundation Health Plan Study (2), and the Japanese Migrant Study (3), showed negative associations, suggesting a possible preventive role of daily alcohol consumption. There was no association reported in the Western Electric Company cohort study (4) data and a positive relationship in data from a Swedish cohort study (5). A recently published case-control study (6) of 402 hospitalized cases of non-fatal myocardial infarction (MI) and 2572 controls concluded an absence of any major overall association but tended to corroborate prior studies showing a negative association.

The purpose of the present report, a case-control study, is to evaluate the relationship, if any, between daily alcohol consumption and fatal CHD taking into account a wide variety of possibly associated variables. The design uses, as patients, a group of deaths due to CHD within a community and, as controls, neighbors of these patients.

MATERIALS AND METHODS

Study population

The study population of cases and controls was restricted to married white men

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Abbreviations: CHD, coronary heart disease; CHF, congestive heart failure; HDL, high density lipoproteins; MI, myocardial infarction.

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age 30 to 70 years residing in two Florida counties. Subjects were identified by weekly reviews of death certificates during a 16-month period. Only men whose death was attributed to CHD within 24 hours after onset of symptoms were included.

One living control, individually matched as to age within the same decade and neighborhood of residence, was selected for each case by a systematic household survey.

Of 1019 wives of eligible patients, 174 were non-respondents and an additional 196 had to be excluded due to non-cooperation. The final study population therefore consisted of 649 case-control pairs. Of these 649 pairs, 81 were excluded because of missing or unknown values, so that the data analyses are based on 568 case-control pairs.

Methods of procedure

Letters of introduction were sent to the wives of eligible patients. An interviewer telephoned the wives to obtain permission for a home interview, which was conducted between two weeks and two months after the death of the patient. For each wife of a case interviewed a wife of a control was interviewed. The interviewer asked questions concerning the husband's daily alcohol consumption during the three months prior to death for the case and three months prior to interview for the control, as well as questions about other coronary risk factors. More detailed descriptions have appeared in a previous publication (7).

Methods of data analysis

The alcohol variable was measured first by asking whether whiskey, beer, or wine was consumed daily and, if so, how many ounces were drunk. Then, to determine the actual number of ounces of alcohol consumed daily, we assigned weighted scores according to the Framingham classification where daily number of ounces of alcohol = 1.00 (daily number of highballs or cocktails) + 0.40 (daily number of 8 oz (236.8 ml) glasses of beer) + 0.67 (daily number of 4 oz (118.4) glasses of wine).

Initially, we calculated the crude matched pair risk ratio estimate and 95 per cent confidence limits to quantitate the crude association between daily alcohol consumption (classified as a binary response variable, i.e., yes, no) and coronary deaths (8). To control for possible additional confounding variables, we used multiple logistic regression analysis (9, 10). In this method, within pair differences among discordant pairs for all available variables are entered into the equation one by one in decreasing order of strength of association with the dependent variable, here daily alcohol consumption. When other variables are included, the residual association between alcohol and coronary deaths is measured by an adjusted matched pair risk ratio estimate defined as exp(alpha) where alpha is the regression constant from the logistic model. To determine whether the association was significant we calculated approximate 95 per cent two-sided confidence limits.

Results

Among the 568 pairs, there were 94 in which the case was a drinker and the control a non-drinker and 161 in which the control was a drinker and the case a non-drinker; the crude matched pair risk ratio estimate for any versus no daily drinking is 0.6 (95 per cent two-sided confidence limits from 0.4 to 0.7).

Table 1 shows the results of the paired multiple logistic regression analysis. The variables significantly associated with daily alcohol consumption categorized as a binary response variable (yes, no) are first, daily number of cigarettes smoked (on a logarithmic scale) followed in order by history of hospitalization for congestive heart failure (CHF), Jewish religion, and relative weight (based on the Framingham classification) (1). The adjusted risk ratio for any versus no daily alcohol con-

sumption is 0.6 (0.4 to 0.8), indicating a preventive role of any compared with no daily alcohol consumption in fatal CHD (table 2).

To explore the effects of quantity of alcohol consumed we repeated the analyses on two subgroups of the aforementioned 255 (94 + 161) discordant pairs; the first group consisted of 124 where one member was a light drinker (≤2 oz (≤59.2 ml) of alcohol daily) and the other a non-drinker. The second group consisted of the remaining 131 pairs where one member was a heavy drinker (>2 oz (>59.2 ml) alcohol daily) and the other a non-drinker. We chose 2 ounces of daily alcohol consumption to distinguish light from heavy drinkers since it represented the median daily alcohol consumption among drinkers.

Among the 124 pairs discordant for light drinking there were 36 where the case was a light drinker and the control a non-drinker and 88 where the control was a light drinker and the case a non-drinker.

TABLE 1

Variables significantly associated with daily alcohol consumption (any versus none) for a series of 568 pairs of married white men at risk of fatal CHD

Variable	Regression coefficient	Standard error	*t*-value
Daily number of cigarettes	0.261	0.061	4.26
Hospitalized for CHF	−2.313	1.091	−2.12
Jewish religion	−1.785	0.492	−3.63
Relative weight	0.012	0.005	2.51

TABLE 2

Crude and adjusted matched pair risk ratio estimates and 95% confidence limits for various levels of alcohol consumption in 255 discordant pairs of white men at risk of fatal CHD

Comparison	Discordant pairs	Crude RR	95% confidence limits	Adjusted RR	95% confidence limits
Any vs. none	255	0.6	0.4–0.7	0.6	0.5–0.8
≤2 ounces (≤59.2 ml) vs. none	124	0.4	0.3–0.6	0.4	0.3–0.6
>2 ounces (>59.2 ml) vs. none	131	0.8	0.6–1.2	0.7	0.4–1.1

Thus, the crude matched pair risk ratio estimate for light versus no daily drinking was 0.4 (0.3 to 0.6). After controlling for other variables among the paired logistic analysis, the adjusted risk ratio for light versus no daily alcohol consumption was 0.4 (0.3 to 0.6) (table 2).

In contrast, the crude matched pair risk ratio estimate for heavy versus no daily drinking was 0.8 (0.6–1.2) and the adjusted risk ratio estimate was 0.7 (0.4 to 1.1) (table 2).

DISCUSSION

The data are consistent with the hypothesis that daily alcohol consumption is associated with a small decreased risk of coronary death. This effect, however, is confined to light drinkers (≤2 oz (≤59.2 ml) of alcohol daily) and is not present for heavy drinkers. There is no evidence of any higher rate of fatal CHD among daily drinkers of alcohol.

The findings from the present investigation are similar to those reported from three cohort studies (1–3) and one case-control study of non-fatal MI (6) These findings may be in conflict with those of a study which reported a positive relationship between alcohol and CHD using registration with the local Temperance Board as an index of alcohol consumption (5). It is possible that the use of Temperance Board registration as a measure of alcohol consumption leads to a spurious association since other causal variables in CHD may be incorporated into this definition. On the other hand, the conflict may be more apparent than real since the protective effect found in the present study is restricted to light drinkers, a group not likely to enroll with a Temperance Board.

It is tempting to speculate that a preventive role of alcohol consumption may be mediated via increases in high density lipoproteins (HDL). The HDL fraction, which is elevated with alcohol consumption (11), has been shown to be protective against CHD (12). The validity of such

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speculation awaits the development of data regarding alterations in specific subfractions of HDL among drinkers and non-drinkers as well as cases and non-cases of CHD.

It must be mentioned that the findings of the present study may be affected by several potential sources of bias, namely, selection, observation and confounding.

Selection bias: This potential source of bias clearly exists in the selection of wives for interview in that wives of patients may have been more or less available for interview than wives of controls. So far as availability is associated with husbands' daily alcohol consumption, a systematic error in either direction in estimate of an alcohol effect might occur. Since it was not possible to specify the total number of eligible neighborhood controls we could not determine whether this potential source of bias was present.

Observation bias: This potential source of bias also exists in the design of this study in that wives may not accurately report the daily alcohol consumption of their husbands. This inaccuracy may have been systematically different between wives of patients and those of controls, resulting in a nonrandom error, since patients had died and controls were alive. If, for example, wives thought that daily alcohol consumption was healthy, wives of patients might have underestimated their husbands' alcohol consumption, whereas wives of controls might have overestimated. This direction of systematic error would lead to an overestimate of a preventive role of alcohol consumption. A systematic inaccuracy in the opposite sense could also be supposed. A random error in wives' reporting would always tend to reduce the magnitude of the association. Since we collected information from the husbands of 48 control wives regarding their own alcohol consumption we were able to determine whether this potential source of bias was present among controls. Among the controls, the husbands and wives reported very similar daily alcohol consumption. It was, of course, not possible to evaluate this potential source of bias among cases.

Confounding bias: It may be supposed that our results may, in part, be related to the fact that history of a prior coronary event (i.e., prior MI, angina, or CHF) may lead patients to change their daily alcohol consumption. To determine whether this source of bias was present, we repeated our analysis, restricting the sample to those pairs in which there was no history of a prior event. In this restricted analysis, the results were virtually identical to those of our principal analysis, indicating that changes in daily alcohol consumption in cases following a coronary event do not account for these findings.

In summary, we believe the most plausible interpretation of our finding to be an inverse relation between any daily alcohol consumption and risk of coronary death which is confined to light drinkers (≤2 oz (59.2 ml) of alcohol daily). Furthermore, it would be of interest to determine whether the alcohol effect is different for whiskey, wine or beer. Finally, the results of other analytic studies, especially those which consider other variables related to light drinking, such as perhaps psychosocial factors or lipoprotein subfractions, would be helpful to decide whether alcohol has a preventive role in CHD.

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