

Alcoholic Beverages and Myocardial Infarction in Young Women

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Abstract: Moderate alcohol consumption has been associated with a reduction in the risk of myocardial infarction (MI) in men. To evaluate this relation in young women, we studied 513 patients with first infarctions and 918 hospital controls, all of whom were less than 50 years of age. The estimated relative risk of MI for current drinkers, after allowance for potential confounding factors, was 0.7 (95 per cent confidence interval. 0.5 – 1.0), and the apparent reduction in risk was strongest for women who drank wine. There was no evidence of an effect among ex-drinkers. (*Am J Public Health* 1981; 71:82-85.)

Introduction

Moderate consumption of alcoholic beverages has been associated with a decreased frequency of coronary artery disease in men.¹⁻⁶ This relation has not been studied in young women, a population with a lower risk of cardiovascular disease and with different drinking patterns.¹

In this report, we evaluate the consumption of alcoholic beverages in relation to myocardial infarction (MI) in women under 50 years of age.

Subjects and Methods

The data were obtained from a case-control study mounted to evaluate the effects of oral contraceptives and other factors on MI risk in women less than 50 years of age.⁷

The methods have been described elsewhere.⁷ In brief, we contacted the coronary care units of 155 hospitals located in and around Boston, New York City, and Philadelphia at weekly intervals. Nurse-interviewers were sent to administer standard questionnaires to women under age 50 for whom there was a suspicion of MI; they also interviewed, as potential controls, women under age 50 in the same hospitals with

diagnoses other than MI. The data described in this report were collected between July 1976 and June 1979; 96 per cent of eligible subjects participated.

Information was obtained on personal characteristics and habits, medical history, and medication history. With regard to alcoholic beverages, each patient was asked if she was a current drinker (during the year before admission), ex-drinker (last drank more than one year before admission), or never drank. For current drinkers, the frequency of drinking (\geq four days, and $<$ four days per week) and the type of beverage usually consumed were recorded.

Cases

All women (565 patients) who met the following criteria were potential cases: age 30 to 49 years; no history of previous MI or of treated angina pectoris; primary diagnosis of MI. The charts of the first 450 patients were reviewed: those for whom the World Health Organization criteria⁸ for the diagnosis of MI were not met (6 per cent), or whose infarction had occurred after admission for some condition other than MI or for whom there was a history of prior cardiovascular surgery (5 per cent) were excluded, leaving 398 cases. The charts of the remaining 115 patients were not reviewed because we judged that a primary discharge diagnosis of MI was sufficiently accurate for our purposes. Thus, the final case series comprised 513 cases; their median age was 44 years and 89 per cent were White.

Controls

All women who met the following criteria were potential controls: age 30 to 49 years; no history of previous MI or of treated angina pectoris; primary diagnosis judged to be unrelated to alcohol consumption. There were 918 such patients: the diagnoses were disc disorders (40 per cent), acute infections (mostly respiratory) (33 per cent), benign skin conditions (21 per cent), and appendicitis (6 per cent). Drinking habits were similar among the diagnostic categories (Table 1). The median age in the control series was 40 years and 89 per cent were White.

Control for Confounding

We estimated the risk of MI for drinkers compared with that for subjects who never drank alcoholic beverages (relative risk).⁹ To control the distorting effects of all potential confounding factors simultaneously, the relative risks were estimated from a multiple logistic regression function for the probability of MI¹⁰ which was fitted by the method of maximum likelihood.¹¹ The function included indicator terms for the categories of alcohol consumption and for age, geograph-

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TABLE 1—Consumption of Alcoholic Beverages among 899 Controls According to Diagnosis*

Diagnosis	Total No. of Subjects	Never Drank No. (%)	Current Drinker† No. (%)	Ex-drinker‡ No. (%)
Disc Disorder	353	51 (14)	289 (82)	13 (4)
Acute Infection	299	52 (17)	230 (77)	17 (6)
Benign Skin Condition	188	34 (18)	142 (76)	12 (6)
Appendicitis	59	9 (15)	50 (85)	0 (0)

*19 controls with an unknown value for alcohol consumption or cigarette smoking are excluded.

†Drank within the year before admission.

‡Last drank at least one year before admission.

ic location of the hospital, year of admission, number of visits to a physician or clinic in the year before admission, religion, years of education, menopausal status, cigarette smoking, treated hypertension, treated diabetes, history of abnormal blood lipids, history of obesity, and history of oral contraceptive use within the month before admission.

Results

The proportion of current drinkers was greater among the controls (79 per cent) than among the cases (71 per cent), and a greater proportion of cases (23 per cent) than controls (16 per cent) never drank (Table 2). Compared with women who never drank, the estimated relative risk of MI was 0.6 for current drinkers ($p < 0.005$) and 1.0 for ex-drinkers. Upon allowance by multiple logistic regression analysis for all identified potential confounding factors, the estimate was 0.7 for current drinkers and 1.0 for ex-drinkers; upon allowance by multiple logistic regression analysis for all identified potential confounding factors, the estimate was 0.7 for current drinkers (95 per cent confidence interval, 0.5–1.0) and 0.8 for ex-drinkers (95 per cent confidence interval, 0.4–1.6).

The estimated relative risk did not vary appreciably according to the frequency of drinking: for 306 cases and 616 controls who usually drank on less than four days per week, the estimate was 0.6; and for 56 cases and 95 controls who drank more often it was 0.7.

There was no evidence that the relative risk varied according to age or predisposition to MI: the point estimates were less than unity in each five-year age group; among non-smokers, moderate smokers, and heavy smokers; and among women apparently predisposed to MI because of hypertension, diabetes mellitus, history of abnormal blood lipids, history of obesity, or oral contraceptive use as well as among women who had none of these factors.

The preferred drink among current drinkers who usually drank one type of beverage was liquor, followed by wine, and then beer (Table 3). The effect of alcohol appeared to be different for different types of beverage: among subjects who usually drank only one type, the proportions of liquor and beer drinkers were similar among cases and controls (X^2_1 (liquor vs beer) = 0.3, $p > 0.5$) while there was a significant deficit of wine drinkers among the cases (X^2_1 (wine vs liquor plus beer) = 22, $p < 0.01$). The point estimate of relative risk was less than unity for each type of beverage, but only the

TABLE 2—Relation of Myocardial Infarction to Consumption of Alcoholic Beverages among 511 Cases and 899 Controls*

	Total No.	Never Drank No. (%)	Current† Drinker No. (%)	Ex-drinker‡ No. (%)
Cases	511	117 (23)	362 (71)	32 (6)
Controls	899	146 (16)	711 (79)	42 (5)
Relative Risk Estimate		1.0**	0.6	1.0
95% Confidence Interval			0.5–0.8	0.6–1.6
Adjusted ^a Relative Risk Estimate		1.0**	0.7	0.8
95% Confidence Interval			0.5–1.0	0.4–1.6

*2 cases and 19 controls with an unknown value for alcohol consumption or cigarette smoking are excluded.

†Drank within the year before admission.

‡Last drank at least one year before admission.

**Reference category.

^aAdjusted for age, location of hospital, religion, years of education, menopausal status, number of visits to a physician or clinic in preceding year, cigarette smoking, hypertension, diabetes, history of abnormal blood lipids, history of obesity, year of admission, and oral contraceptive use within preceding year.

TABLE 3—Relation of Myocardial Infarction to Current Consumption of Alcoholic Beverages among 511 Cases and 899 Controls, according to Type of Beverage usually Consumed*

	Current Drinker					
	Never Drank	Liquor	Wine	Beer	More Than One Type	Unknown
Cases	117	138	48	40	123	13
Controls	146	193	167	63	244	44
Relative Risk Estimate	1.0†	0.9	0.4	0.8	0.6	0.4

*32 cases and 42 controls who last drank more than one year before admission are excluded.

†Reference category

estimate for wine was statistically significant ($p < 0.001$). Upon allowance for confounding factors, these findings were not materially altered: for drinkers of wine, in particular, the relative risk estimate was 0.5 (95 per cent confidence interval, 0.3-0.8)

Discussion

The current findings suggest that consumption of alcoholic beverages is associated with a reduction in the risk of MI: in this study of young women, current drinkers were estimated to have a significantly reduced risk of first infarction relative to those who never drank. We had no information on the quantity of alcohol consumed. However, since 86 per cent of the current drinkers reported that they drank on less than four days per week, probably most of them were "moderate" drinkers. There was no evidence of an effect among ex-drinkers.

Biased recording of drinking habits is unlikely to explain our findings, since alcohol consumption was but one of a large number of items on which information was routinely collected.

Although certain illnesses are caused by, or may affect, alcohol consumption, we believe that the selection of our control series was unbiased in regard to drinking habits: the diagnoses are not known to be related to alcohol use, and drinking habits were similar among the major diagnostic categories.

The association was not accounted for by identified confounding factors. In particular, to reduce the possibility of bias resulting from prodromal symptoms influencing drinking habits, subjects with angina pectoris were excluded; other factors that were inversely related to drinking, such as hypertension, diabetes, Jewish religion, and lower level of education, were controlled in the analysis, as were factors associated with a higher frequency of alcohol use, especially cigarette smoking. However, insofar as information on certain factors, such as the presence and severity of certain medical conditions (e.g., hypertension), can never be ascertained with precision, it remains possible that confounding was incompletely controlled.

Personality characteristics could account for part of the association¹² if individuals exhibiting "relaxed" behavior

("type B") are more likely to be moderate drinkers than those exhibiting "hard-driving" behavior ("type A"). Our study did not evaluate this possibility, nor have any of the other studies to date. However, we are unaware of any evidence of an association between drinking habits and type A or B personality.

Our results accord with findings from studies of men which, taken together, suggest that the rate of coronary artery disease is reduced by about 30 per cent among moderate drinkers.¹⁻⁶ The finding of a similar relation in men and in women, despite differences in drinking patterns and underlying predisposition to MI,¹ suggests that the association is not due to as yet unidentified correlates of drinking and MI. Moreover, a significant reduction in MI risk has been related to beer in one study,⁴ to beer, wine, and liquor in another,⁶ and to wine in our own, suggesting that a protective effect is related to alcohol itself, rather than to some other substance in alcohol-containing beverages.

As to mechanisms, it has been proposed that moderate alcohol use may exert its effect by elevating high density lipoprotein levels;^{13, 14} possible effects on platelet aggregation are also being evaluated.¹⁵ For the present, however, a satisfactory explanation for a protective effect is lacking.

REFERENCES

1. Shurtleff D: Some characteristics related to the incidence of cardiovascular disease and death: Framingham study, 16-year follow-up. IN: Kannel WB, Gordon T (eds): The Framingham Study, section 26. Washington, DC; US Govt Printing Office, 1970.
2. Klatsky AL, Friedman GD, Siegelaub AB: Alcohol consumption before myocardial infarction. *Ann Intern Med* 1974; 81:294-301.
3. Stason WB, Neff RK, Miettinen OS, *et al*: Alcohol consumption and nonfatal myocardial infarction. *Am J Epidemiol* 1976; 104:603-608.
4. Yano K, Rhoads GG, Kagan A: Coffee, alcohol and risk of coronary heart disease among Japanese men living in Hawaii. *N Engl J Med* 1977; 297:405-409.
5. Hennekens CH, Rosner B, Cole DS: Daily alcohol consumption and fatal coronary heart disease. *Am J Epidemiol* 1978; 107:196-200.
6. Hennekens CH, Willett W, Rosner B, *et al*: Effects of beer, wine and liquor in coronary deaths. *JAMA* 1979; 242:1973-1974.
7. Shapiro S, Slone D, Rosenberg L, *et al*: Oral-contraceptive use in relation to myocardial infarction. *Lancet* 1979; 1:743-747.
8. Ischemic Heart Disease Registers: Report of the Fifth Working Group. Copenhagen: World Health Organization, 1971.

9. Miettinen OS: Estimability and estimation in case referent studies. *Am J Epidemiol* 1976; 103:226-235.
10. Truett J, Cornfield J, Kannel WA: Multivariate analysis of the risk of coronary heart disease in Framingham. *J Chron Dis* 1976; 20:511-525.
11. Walker SA, Duncan DB: Estimation of the probability of an event as a function of several independent variables. *Biometrika* 1967; 54:167-169.
12. Jenkins CD: Recent evidence supporting psychologic and social risk factors for coronary disease. *N Engl J Med* 1976; 294:987-994, 1033-1038.
13. Goldbourt U, Medalie JH: High density lipoprotein cholesterol and coronary disease incidence. *Cardiovasc Epidemiol Newsletter* 1977; 22:23.
14. Castelli WP, Gordon T, Hjortland MC, *et al*: Alcohol and blood lipids: The cooperative phenotyping study. *Lancet* 1977; 2:153-157.
15. Haut MJ, Cowan DH: The effect of ethanol on hemostatic properties of human blood platelets. *Am J Med* 1974; 56:22-23.

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Patterns of Alcohol Use among Veterans and Nonveterans: A Confirmation of Previous Findings

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Abstract: Analysis of alcohol consumption among veterans (N = 684) included in a 1977 national household survey of 3,322 adults confirmed previous findings based on 1975 national survey data. As previously found, higher levels of alcohol consumption were significantly related to being a veteran of U.S. military service. However, when demographic differences were adjusted between veterans and nonveterans, this was no longer the case. It was suggested that this confirmation of earlier findings strengthens the validity of these findings. (*Am J Public Health* 1981; 71:85-88.)

Introduction

Studies within Veterans Administration (VA) hospitals have often found over 20 per cent of the patients in these facilities diagnosable as alcoholic.¹⁻³ However, the great majority of veterans are not treated in VA hospitals, but remain in the community. Using 1975 national survey data collected for the National Institute on Drug Abuse, analyses of alcohol abuse among veterans have shown that a national

community-based sample of veterans do have a higher prevalence of defined alcohol abuse⁴ and heavy drinking⁵ than nonveterans. However, when demographic differences between veterans and nonveterans were statistically controlled, little or no differences remained between these groups.

Nevertheless, the research described above is limited because it was based on restricted measures of alcohol use and a moderate sample of veterans (N = 681). This brief report will present more recent data from a similar national survey conducted in 1977.⁶ The purpose of this analysis will be to confirm previous findings with a more recent national sample of veterans.

Materials and Method

The data for this analysis were collected in face-to-face interviews with a national probability sample of over 3,000 adults in 1977.* The interviews were conducted in 1977 and represented a series of epidemiologic studies conducted under the auspices of the National Institute of Drug Abuse.⁶

Data were analyzed with a specific approach in mind, based on the earlier findings: veterans as a group were ex-

*Because of the importance of younger adults to the subject areas of the research project, the design called for oversampling of the 18 to 34 age group. This sample was then weighted according to age, sex, education, and community type, to bring the sample distributions as close as possible to recent United States Census estimates. All statistics reported here have been adjusted by these demographic weights. For further information on this survey see Abelson and Fishburne.⁶

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