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Moderate Alcohol Consumption and Risk of Coronary Heart Disease Among Women With Type 2 Diabetes Mellitus

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Background—Moderate alcohol consumption is associated with reduced risk for coronary heart disease (CHD) in generally healthy populations. We assessed prospectively the association between moderate alcohol intake and CHD risk in women with type 2 diabetes mellitus, a group at high risk for cardiovascular disease.

Methods and Results—We studied women in the Nurses' Health Study who reported a diagnosis of diabetes mellitus at ≥ 30 years of age. During 39 092 person-years of follow-up from 1980 to 1994, there were 295 CHD events documented among this population, including 194 cases of nonfatal myocardial infarction and 101 cases of fatal CHD. Odds ratios derived from logistic regression were used to estimate relative risks (RRs) for CHD as a function of usual alcohol intake, with adjustment for potential confounders. Compared with diabetic women reporting no alcohol intake, the age-adjusted RR for nonfatal or fatal CHD among diabetic women reporting usual intake of 0.1 to 4.9 g (< 0.5 drinks) of alcohol daily was 0.74 (95% CI 0.56 to 0.98), and among those reporting usual intake ≥ 5 g/d, it was 0.48 (95% CI 0.32 to 0.72) (P for trend < 0.0001). Inverse associations between alcohol intake and CHD risk remained significant in multivariate analysis adjusting for several other coronary risk factors (0.1 to 4.9 g/d: RR 0.72 [95% CI 0.54 to 0.96]; ≥ 5 g/d: RR 0.45 [0.29 to 0.68]).

Conclusions—Although potential risks of alcohol consumption must be considered, these data suggest that moderate alcohol consumption is associated with reduced CHD risk in women with diabetes and should not be routinely discouraged. (*Circulation*. 2000;102:494-499.)

Key Words: diabetes mellitus ■ coronary disease ■ alcohol ■ risk factors

Type 2 diabetes mellitus (DM) is associated with a 3-fold increase in risk for coronary heart disease (CHD), independent of other coronary risk factors,¹ and appears to confer particular risk in women.² Presumed contributors to the increased coronary risk in DM include associated dyslipidemia,³ hypertension,⁴ insulin resistance,⁵ and hypercoagulability.⁶ It is important to determine whether approaches that reduce CHD risk in nondiabetic populations may also have a role in the setting of diabetes.

older-onset diabetic individuals.¹³ Observations that several coronary risk factors associated with DM, including dyslipidemia,⁷ hyperinsulinemia,¹⁴ and coagulation disturbances,¹⁵ may be favorably affected by alcohol likewise suggest the potential for particular cardiovascular benefit of moderate alcohol use in this high-risk group. We therefore assessed in a large prospective cohort study of nurses whether moderate alcohol intake is associated with a reduced risk for CHD among diabetic women.

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Moderate alcohol intake has been associated with a significant reduction in risk for myocardial infarction (MI) among generally nondiabetic populations,⁷⁻¹² but the effect of alcohol on cardiovascular risk in those with diabetes has been little studied. A recent report noted an inverse association between alcohol consumption and CHD mortality among

Methods

The Nurses' Health Study (NHS) is a prospective cohort study of 121 700 female nurses aged 30 to 55 years at study inception in 1976 and residing in 1 of 11 US states. Participants complete biennial questionnaires on lifestyle factors and medical conditions, the details of which have been reported previously.¹⁶ The present investigation included 5103 women who reported a physician diagnosis of DM at ≥ 30 years of age, from baseline through 1992; were free of CHD

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(including MI, angina, and/or coronary revascularization), stroke, or cancer (other than nonmelanoma skin cancer) at or before the 1980 questionnaire (when alcohol intake was first assessed); and who provided information on usual alcohol consumption.

Confirmation of DM

Women were included who reported the diagnosis of DM at ≥ 30 years of age. In a previous validation study,¹ 84% of a random subset of these women had the diagnosis of type 2 DM confirmed on medical record review based on National Diabetes Data Group criteria for DM¹⁷ (ie, fasting plasma glucose level ≥ 140 mg/dL and/or random plasma glucose level or plasma glucose level 2 hours after 75-g glucose load ≥ 200 mg/dL, on 2 or more occasions), in the absence of ketoacidosis.

We sent supplementary questionnaires to all women who self-reported a diagnosis of diabetes; the questionnaire asked about details of diagnosis (eg, diagnostic tests and symptoms) and therapy. Women were considered to have definite type 2 DM if they reported 2 or more glucose levels diagnostic of diabetes on the basis of National Diabetes Data Group criteria and the absence of ketoacidosis at the time of diagnosis. As described in a previous report,¹ the diagnosis of type 2 DM was confirmed by medical record review in 61 (98.4%) of 62 women considered to have definite type 2 DM by supplementary questionnaire. A secondary set of analyses was conducted that included only women with definite type 2 DM by these criteria.

Alcohol Consumption

A food frequency questionnaire was included on the biennial NHS questionnaire in 1980, 1984, 1986, and 1990 that asked about the average frequency of consumption of specified foods and beverages during the preceding 12 months. Questions about intake of beer, wine, and spirits were included separately. Total alcohol intake was calculated in grams by adding usual intake of alcoholic beverages, assuming the following content: 1 12-oz can of beer, 12.8 g; 1 4-oz glass of wine, 11.0 g; standard drink of spirits, 14.0 g.¹⁸ A previous validation study among a subset of participants indicated a high correlation between reported alcohol intake on the questionnaire and dietary records ($r=0.9$).¹⁹ Also, high correlations were noted between alcohol consumption reported on the 1980 and 1984 questionnaires and between these parameters and direct measures of HDL cholesterol.¹⁹

Other Covariates

Because alcohol intake was first assessed in 1980, baseline information for these analyses was derived from the 1980 questionnaire, including the following: weight; smoking; menopausal status; postmenopausal hormone use; personal history of diabetes mellitus, hypertension, and hypercholesterolemia; parental history of MI; physical activity level; aspirin use; dietary fat; multivitamin use; and use of vitamin E supplements. Information on these covariates was also updated subsequently. Body mass index was calculated with height reported on the baseline questionnaire in 1976.

CHD End Points

Women were asked on biennial questionnaires whether they had had a diagnosis of MI in the preceding 2-year interval. Women who reported MI were asked for permission to review their medical records.

Self-reports of nonfatal MI were considered confirmed if information in the medical record met the following World Health Organization criteria: characteristic symptoms with either diagnostic ECG changes and/or cardiac enzyme elevation. "Silent" MIs discovered on a routine ECG were excluded. If no records were available for self-reported MI but there was confirmation of hospitalization and information from an interview or letter supporting the diagnosis, self-reported diagnosis was considered probable; probable cases were also included in these analyses (17% of total cases).

Deaths were generally reported by family members of participants or were identified by search of the National Death Index. The

follow-up rate was $>98\%$. For mortality attributed to cardiovascular disease, permission was requested from the next of kin to review records. A fatal MI was confirmed if medical records or autopsy report indicated this diagnosis. Coronary death was diagnosed if the death certificate reported coronary disease as the cause of death, there was no other more likely cause apparent, and the participant was reported to have coronary disease previously (by questionnaire or per next of kin). A diagnosis of fatal coronary disease was never made on the basis of the death certificate alone. We also included in the coronary death category those who had sudden death, ie, death within 1 hour of symptom onset in a woman without known disease that could explain the death.

Data Analysis

Person-time for each participant was calculated from the time of return of the 1980 questionnaire (when alcohol intake was first assessed) for women diagnosed with diabetes in 1980 or before or, if diabetes was diagnosed after 1980, from the time that diabetes was first reported until a nonfatal MI, death, or June 1, 1994. Women reporting cardiovascular disease on a questionnaire were excluded from subsequent follow-up. If a participant had a nonfatal MI and then subsequently died of CHD, we considered only the nonfatal MI in the analysis.

Women were divided into categories based on usual daily alcohol consumption. Incidence rates were calculated by dividing the number of events by the person-time of follow-up for each category of intake. Relative risks (RRs) were calculated as the rate for a given category of alcohol intake compared with the referent category ("no alcohol"). Tests for trend were performed by assigning the median value to increasing categories of alcohol use; a 2-tailed P value <0.05 was considered statistically significant. Pooled logistic regression was used to model coronary disease event rates over each 2-year follow-up interval in relation to alcohol intake reported on the prior questionnaire, with adjustment for other potential confounders. Covariates were updated biennially when possible (age, cigarette smoking, body mass index, menopausal status/postmenopausal hormone use, multivitamin use, and vitamin E supplement use). Aspirin use was assessed in 1980, 1982, 1984, and 1988 and was updated accordingly. Vigorous exercise was assessed in 1980. Hypoglycemic

TABLE 1. Age-Adjusted Baseline (1980) Characteristics of Women With DM at Baseline as a Function of Usual Alcohol Consumption

	Usual Daily Alcohol Intake, g		
	None	0.1–4.9	≥ 5
Age, y	49.4	48.1	48.6
BMI, kg/m ²	29.4	27.6	25.5
Current smoker, %	22	27	40
Family history of MI, %	24	25	23
Hypertension, %	48	45	43
High cholesterol, %	17	18	18
Regular aspirin use, %	47	47	46
Current PMH use, %	11	16	15
Vitamin E supplement use, %	14	16	16
Multivitamin use, %	35	38	41
*Regular physical activity, %	37	40	47
Total fat intake, % total kcal	40	40	38
Saturated fat intake, % total kcal	16	16	15
Dietary fiber intake, g/d	15	15	13

BMI indicates body mass index; Family history of MI, parental myocardial infarction before age 60 years; PMH, postmenopausal hormones. *Regular physical activity refers to vigorous exercise at least once per week. All variables were self-reported by questionnaire, and all but age were age adjusted.

TABLE 2. RR for Fatal and Nonfatal CHD Associated With Alcohol Consumption

	Usual Daily Alcohol Intake, g			<i>P</i> _{trend}
	None	0.1–4.9	≥5	
Total CHD				
Cases, n	204	65	26	
Person-years	22 715	10 326	6051	
Age-adjusted RR (95% CI)	1.0	0.74 (0.56–0.98)	0.48 (0.32–0.72)	0.0001
Multivariate RR* (95% CI)	1.0	0.72 (0.54–0.96)	0.45 (0.29–0.68)	0.0003
Nonfatal CHD				
Cases, n	132	45	17	
Age adjusted RR (95% CI)	1.0	0.78 (0.56–1.09)	0.48 (0.29–0.80)	0.003
Multivariate RR* (95% CI)	1.0	0.79 (0.56–1.12)	0.47 (0.28–0.79)	0.005
Fatal CHD				
Cases, n	72	20	9	
Age adjusted RR (95% CI)	1.0	0.66 (0.40–1.08)	0.48 (0.24–0.95)	0.01
Multivariate RR* (95% CI)	1.0	0.60 (0.36–1.01)	0.43 (0.21–0.88)	0.03

*Adjusting for age (5-year categories), time period (7 periods), body mass index (5 categories), cigarette smoking (never, past, current smoking of 1–14, 15–24, and ≥25 cigarettes/d), parental history of MI before age 60 y, hypertension, hypercholesterolemia, menopausal status/postmenopausal hormone use (premenopausal, postmenopausal without hormone replacement, postmenopausal with past hormone replacement, postmenopausal with current hormone replacement), aspirin use (<1/wk, 1–6/wk, ≥7/wk), multivitamin use, vitamin E supplement use, and physical activity level (vigorous exercise at least once per week).

medication use was assessed by supplementary questionnaire sent to all participants after a new report of diabetes and also in 1988; women reporting sulfonylurea or insulin use or both on at least 1 of these assessments were considered in subgroup analyses to be users of hypoglycemic medication.

Primary analyses included all women self-reporting diabetes at age 30 years and older; a second set of analyses were limited to women who met criteria for definite type 2 DM by supplementary questionnaire.

Results

Alcohol intake tended to be light to moderate in this cohort of diabetic women. Women who reported no alcohol intake accounted for 58.1% of person-time, and women who consumed 0.1 to 4.9 g of alcohol per day (one third to one half drink per day) accounted for 26.4% of person-time. Only 5.7% of the person-time reflected women who consumed ≥15 g of alcohol daily. Because of the limited person-time represented by this higher category, the primary analyses were conducted with those who drank ≥5 g of alcohol daily (15.5% of person-time) as the highest category.

Baseline (1980) characteristics of the cohort, specifically those who reported diabetes in 1980 or before, are shown in Table 1. Compared with women who reported drinking no alcohol, women who reported any regular alcohol intake tended to have lower body mass index and less hypertension and were more likely to use postmenopausal hormones and vitamin supplements and to engage in regular physical activity. However, women who reported regular alcohol intake were also more likely to be smokers.

During 39 092 person-years of follow-up from 1980 to 1994, we documented 295 CHD events among this population, including 194 cases of nonfatal MI and 101 cases of fatal CHD.

Age-adjusted rates of CHD were significantly lower in women who reported moderate alcohol intake than in those who reported drinking no alcohol. The age-adjusted risk for nonfatal or fatal CHD associated with drinking 0.1 to 4.9 g of alcohol daily was 0.74 (95% CI 0.56 to 0.98); for drinking ≥5 g of alcohol daily, it was 0.48 (95% CI 0.32 to 0.72) (*P* for trend <0.0001). These reduced risks remained statistically significant in a multivariate analysis that also adjusted for body mass index, smoking, family history of MI, hypertension, hypercholesterolemia, menopausal status, postmenopausal hormone use, aspirin use, multivitamin use, vitamin E intake, and physical activity level. After adjustment for these factors, the RR for nonfatal or fatal CHD associated with intake of 0.1 to 4.9 g/d alcohol was 0.72 (95% CI 0.54 to 0.96), and for ≥5 g/d, it was 0.45 (0.29 to 0.68). Risk reductions associated with modest alcohol intake were similar for nonfatal and fatal events (Table 2).

There was no evidence that risk reductions for CHD associated with alcohol differed by other CHD risk factor status. Stratification by smoking status, body mass index, presence of hypertension, and family history of MI yielded similar results, although confidence intervals for the estimates of risk reduction associated with modest alcohol intake tended to include 1 because of smaller sample sizes. (Table 3).

Because women consuming little or no alcohol might conceivably have limited their alcohol intake secondary to illness, we conducted secondary analyses excluding women who reported on the 1980 questionnaire that they had greatly reduced their alcohol consumption over the last decade. Compared with nondrinkers, the multivariate RR for CHD among women reporting usual alcohol intake of 0.1 to 4.9 g/d was 0.72 (95% CI 0.51 to 1.01); among those reporting alcohol intake of ≥5 g/d, it was 0.51 (95% CI 0.32 to 0.80) (*P* for trend 0.006).

TABLE 3. Multivariate RR for Nonfatal and Fatal CHD Associated With Alcohol Consumption in Different Subgroups of Women

	Usual Daily Alcohol Intake, g			P trend
	None	0.1–4.9	≥5	
Current cigarette smoking				
Yes	1.0	0.92 (0.54–1.58)	0.50 (0.26–0.96)	0.04
No	1.0	0.76 (0.55–1.06)	0.49 (0.29–0.81)	0.007
Body mass index (kg/m ²)				
<25	1.0	1.04 (0.48–2.25)	0.61 (0.26–1.42)	0.21
≥25	1.0	0.86 (0.61–1.21)	0.43 (0.25–0.75)	0.003
Hypertension				
Yes	1.0	0.84 (0.60–1.16)	0.54 (0.34–0.85)	0.009
No	1.0	0.73 (0.42–1.28)	0.25 (0.10–0.64)	0.005
Family history of MI*				
Yes	1.0	1.05 (0.63–1.78)	0.41 (0.18–0.93)	0.03
No	1.0	0.71 (0.51–1.00)	0.45 (0.28–0.72)	0.001

Results are expressed as RRs (95% CI). RRs are adjusted for age (5-year categories), time period (7 periods), body mass index (5 categories), cigarette smoking (never, past, current smoking of 1–14, 15–24, and ≥25 cigarettes/d), parental history of MI relative before age 60 y, hypertension, hypercholesterolemia, menopausal status/postmenopausal hormone use (premenopausal, postmenopausal without hormone replacement, postmenopausal with past hormone replacement, postmenopausal with current hormone replacement), aspirin use (<1/wk, 1–6/wk, ≥1/wk), multivitamin use, vitamin E supplement use, and physical activity level (vigorous exercise at least once per week); the stratifying variable was not included in the model.

*Parent <60 y of age.

We also conducted analyses excluding the first 2 years of follow-up after alcohol consumption was reported (ie, excluding 62 cases), and again results were essentially unchanged. Compared with women reporting no alcohol consumption, the multivariate RR for CHD was 0.67 (95% CI 0.48 to 0.93) among those drinking 0.1 to 4.9 g/d and 0.43 (95% CI 0.27 to 0.69) among those drinking ≥5 g/d.

Insofar as women with more severe diabetes might be less apt to consume alcohol and might independently be at greater risk for CHD, we also attempted to control for severity of diabetes. Results were similar in analyses that controlled for duration of diabetes and for history of insulin or sulfonylurea use, as assessed at 2 different time points. With adjustment for duration of diabetes, the multivariate RR for CHD associated with daily intake of 0.1 to 4.9 g of alcohol was 0.72 (95% CI 0.53 to 1.00) and that associated with daily intake of ≥5 g was 0.48 (95% CI 0.30 to 0.75). Similarly, after adjustment for hypoglycemic medication, daily intake of 0.1 to 4.9 g of alcohol was associated with a multivariate RR for CHD of 0.74 (95% CI 0.54 to 1.01), and intake of ≥5 g/d was associated with an RR of 0.52 (95% CI 0.33 to 0.81).

In analyses limited to the subgroup of women who reported insulin or sulfonylurea therapy (who made up 54% of the total person-time), there still appeared to be a reduction in CHD risk associated with moderate alcohol intake. Multivariate RRs for CHD were 0.58 (95% CI 0.39 to 0.87) for 0.1 to 4.9 g/d and 0.59 (95% CI 0.34 to 1.02) for ≥5 g/d. Among women not reporting use of these medications, corresponding multivariate RRs were 1.26 (95% CI 0.73 to 2.20) and 0.48 (95% CI 0.21 to 1.07).

We also conducted analyses of alcohol and CHD risk limited to women whose diabetes was considered to be definite type 2 DM based on the supplementary questionnaire (n=249 cases). The distribution of alcohol intake in this subgroup was highly comparable to that in the cohort as a whole. Among these confirmed cases, modest alcohol consumption was likewise associated with reduced CHD risk. Multivariate RR for CHD associated with intake of 0.1 to 4.9 g/d alcohol was 0.74 (95% CI 0.54 to 1.02), and for ≥5 g/d, it was 0.66 (95% CI 0.43 to 1.00).

As noted, few women in our cohort reported alcohol intake of ≥15 g/d, and they contributed only 13 cases of CHD. When we examined this group separately, the RR for CHD among women consuming 5 to 14.9 g/d alcohol was 0.38 (95% CI 0.21 to 0.67), and among those consuming ≥15 g/d, it was 0.56 (95% CI 0.31 to 0.99).

Discussion

Women with type 2 DM are at high risk for subsequent CHD events. The present findings suggest that light to moderate alcohol intake is associated with an approximate halving of this risk. The RR reduction for CHD associated with modest alcohol consumption among diabetic women in this cohort is comparable to that seen in other women,⁸ and given the high risk for CHD associated with DM, the absolute risk reduction would be expected to be even greater.

Several plausible biological explanations exist for a beneficial effect of alcohol on CHD risk in individuals with type 2 DM. Effects on lipids are likely to be a major contributor. Although excessive alcohol consumption can raise triglyceride levels, alcohol intake also is associated with higher levels

of HDL, including HDL₂ and HDL₃,⁷ and in small intervention studies has directly raised HDL levels.²⁰ Reduced HDL levels are both characteristic of DM³ and a strong independent predictor of CHD events.²¹ Indeed, the inverse association between alcohol consumption and CHD has been noted to be attenuated markedly when HDL levels are taken into consideration.⁷

Effects of alcohol on reducing coagulability¹⁵ likewise are particularly relevant in the diabetic population. Type 2 DM is associated with a clear tendency to thrombosis,⁶ including increased levels of plasminogen activator inhibitor-1,²² impaired fibrinolysis,²³ and increased platelet adhesion and aggregability by *in vitro* testing.²⁴

Other coronary risk factors are more common in type 2 DM and also may be improved with modest alcohol consumption. Half or more of type 2 diabetics have hypertension.⁴ Although excessive alcohol intake is associated with increased blood pressure, moderate (<2 drinks/d) intake may be associated with lower blood pressures, at least in women.^{25,26} Fasting insulin levels are elevated in type 2 DM and may be reduced in the setting of regular alcohol consumption.¹⁴ Hyperinsulinemia has been reported to predict CHD in men, although this remains controversial in women.⁵

Data are scarce regarding the association between alcohol consumption and CHD in the setting of diabetes. A recently published study of 983 female and male participants in the Wisconsin Epidemiologic Study of Diabetic Retinopathy (WESDR) revealed a significant inverse association between alcohol use and CHD mortality.¹³ As in the present study, usual alcohol intake was modest among that cohort. Compared with nondrinkers, reductions in CHD mortality were 40% for participants consuming <2 g of alcohol daily, 55% for those consuming 2 to 13 g of alcohol daily, and 73% for those consuming \geq 14 g/d. Females, who constituted 55% of the cohort, were not evaluated separately, however, and nonfatal CHD was not assessed in that report.

Among males but not females with type 2 DM participating in the WHO Multinational Study of Vascular Disease in Diabetes,²⁷ moderate alcohol consumption was also associated with significant reduction in CHD mortality. However, this report involved only 123 diabetic women and likewise did not consider nonfatal CHD events.

The potential benefits of alcohol consumption must of course be balanced against potential risks. Risks particularly relevant in the setting of diabetes include exacerbation of diabetic neuropathy²⁸ and retinopathy²⁹ and suppression of hepatic gluconeogenesis,³⁰ with attendant risk of hypoglycemia. However, effects on the nervous system,²⁸ retina,²⁹ and hepatic glucose output and ketogenesis³¹ tend to be dose related and less a risk with modest alcohol consumption. Also, risk for hypoglycemia can be minimized by consuming alcohol with a meal rather than in the unfed state. Despite reports that alcohol may cause deterioration in glucose tolerance³² or even precipitate ketoacidosis³¹ in the setting of DM, other studies have found no significant effects of modest alcohol intake with meals on insulin requirement³³ or on postprandial insulin and glucose levels³⁴ in diabetic individuals. Data were not available in the present cohort to address

hypoglycemia or other potential correlates of alcohol consumption.

Alcohol consumption tended to be modest in this cohort, and we thus cannot address the associations between heavier alcohol consumption and CHD in the setting of diabetes. However, observations of a U-shaped relationship between alcohol intake and CHD risk in generally healthy populations^{11,12} suggest a caution against more than moderate intake of alcohol. As emphasized in a recent editorial,³⁵ the fact that some individuals cannot effectively limit themselves to light to moderate intake further reinforces the need to be cautious in any report on the potential benefits of alcohol consumption.

These observational data cannot prove that alcohol causes a reduction in CHD risk. Conceivably, women who consume modest amounts of alcohol may be healthier in general than nondrinkers. Our observations that women reporting modest alcohol intake did not appear to differ markedly from nondrinkers in many potential confounders, and, moreover, that adjustment for potential confounders in multivariate analyses did not significantly affect results, support an independent effect of alcohol intake on CHD risk; still, the possibility of unmeasured or incompletely controlled confounding cannot be excluded. Additionally, the consistency of results after the exclusion of those who reported a reduction in alcohol intake and after adjustment for hypoglycemic medication use or duration of diabetes suggests that greater severity of illness or associated medication use does not wholly explain the higher CHD risk in nondrinkers. Also, the biological plausibility of a causal relationship between modest alcohol intake and reduction in CHD risk through effects on lipids and coagulability, as well as the consistency of the present findings with findings among nondiabetic populations, supports the likelihood of a causal association.

Other limitations of the present study warrant consideration. Because diabetes is self-reported, there is potential for misclassification. Nonetheless, self-report of this diagnosis has previously been shown to be valid in this cohort.¹ Diagnostic criteria for DM have changed recently³⁶ such that some women who would not have been considered to have diabetes by standards used when this cohort reported diabetes would now be considered to have this diagnosis. Nonetheless, this would not affect the case status of the women included in the present report. Additionally, hypoglycemic medication use was not assessed at regular follow-up intervals, and thus this exposure is also subject to misclassification. Although many women who did not report use of these medications through 1988 may have used them subsequently, the natural history of diabetes makes it less likely that women who report their use will have discontinued use over follow-up; hence, the finding of an association between alcohol consumption and reduced CHD risk even in women using hypoglycemic medications remains reassuring. Finally, this study does not address the relationship between alcohol consumption and coronary events among diabetic women with prevalent CHD.

Among this cohort of women with type 2 DM, light to moderate alcohol intake was associated with significant reduction in risk for fatal or nonfatal CHD. Although observed associations cannot prove causation, and although all

diabetic patients should be counseled regarding the potential risks of alcohol intake, particularly in excess or in the absence of food intake, the present findings suggest that modest alcohol consumption may have favorable cardiovascular effects and should not be routinely discouraged in this population.

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