

Alcohol Consumption and Cardiovascular Disease Mortality in Hypertensive Men

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Background: Heavy alcohol drinking is associated with a dose-dependent increase in blood pressure, but data on the relation between alcohol consumption and mortality in hypertensive patients are sparse.

Objective: To assess the relation between light to moderate alcohol consumption and total mortality from cardiovascular disease (CVD) among men with hypertension.

Participants and Design: From the Physicians' Health Study enrollment cohort of 88882 men who provided self-reported information on alcohol intake, we identified a group of 14125 men with a history of current or past treatment for hypertension who were free of myocardial infarction, stroke, cancer, or liver disease at baseline.

Main Outcome Measure: Comparison of total and CVD mortality among men with hypertension who had reported to be either nondrinkers or rare drinkers, or light to moderate drinkers.

Results: During 75710 person-years of follow-up, there were 1018 deaths, including 579 from CVD. Compared with individuals who rarely or never drank alcoholic beverages,

those who reported monthly, weekly, and daily alcohol consumption, respectively, had multivariate adjusted relative risks (RRs) for CVD mortality of 0.83 (95% confidence interval [CI], 0.62-1.13), 0.61 (CI, 0.49-0.77), and 0.56 (CI, 0.44-0.71) ($P < .001$ for linear trend). In the same groups, RRs for total mortality were respectively 0.86 (CI, 0.67-1.10), 0.72 (CI, 0.60-0.86), and 0.73 (CI, 0.61-0.87) ($P < .001$ for linear trend). Among men with a systolic blood pressure of 140 mm Hg or higher or a diastolic blood pressure of 90 mm Hg or higher, the RRs for CVD mortality were, respectively, 1.00 (referent), 0.82 (CI, 0.56-1.21), 0.64 (CI, 0.48-0.85), and 0.56 (CI, 0.42-0.75) ($P < .001$ for linear trend). On the other hand, we found no significant association between moderate alcohol consumption and cancer mortality ($P = .8$ for linear trend).

Conclusion: These results, which require confirmation in other large-scale studies, suggest that light to moderate alcohol consumption is associated with a reduction in risk of total and CVD mortality in hypertensive men.

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A RECENTLY PUBLISHED AMERICAN Heart Association Science advisory states that "patients who are hypertensive should avoid alcoholic beverages."¹ In contrast, earlier American Heart Association dietary guidelines² and a statement from the Sixth Report of the Joint National Committee on the Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC VI)³ recommends limiting alcohol intake to no more than 2 drinks per day for men, and 1 drink per day for women, for the prevention and treatment of hypertension. Heavy alcohol consumption has been shown in observational studies to have a strong positive association with elevated blood pressure.⁴⁻⁶ Clinical trials^{7,8} have demonstrated

that reduction in alcohol intake among individuals who drink heavily (ie, 3 or more drinks per day) can lower blood pressure in normotensive and hypertensive men.

In the general population, epidemiologic studies have consistently demonstrated an inverse association between moderate alcohol consumption and cardiovascular disease (CVD) mortality.⁹⁻¹⁴ Whether or not alcohol consumption has a similar association with CVD mortality among individuals with hypertension, however, is a largely unexamined and controversial question. We therefore used prospective data from the Physicians' Health Study enrollment cohort to assess whether light to moderate alcohol intake is associated with reduced total and CVD mortality among hypertensive men.

Author affiliations are given at the end of the article. The authors have no relevant financial interest in this article.

METHODS

STUDY POPULATION

The Physicians' Health Study was a randomized, double-blind, placebo-controlled trial of low-dose aspirin (325 mg every other day) and beta carotene (50 mg every other day) in the primary prevention of CVD and cancer. Potentially eligible participants were male physicians living in the United States. In 1982 letters of invitation, informed consent forms, and baseline questionnaires were mailed to 261 248 male physicians listed on the American Medical Association's mailing tape. By February 1984, 104 353 completed questionnaires had been returned. We excluded persons with missing alcohol data, and those reporting a history of myocardial infarction (MI), stroke, cancer, or liver disease at baseline. These exclusions resulted in a total of 88 882 subjects, 14 125 of whom had self-reported current or past treatment for hypertension and thus comprised our study population.

BASELINE DATA

Information about alcohol consumption and several other coronary risk factors was collected at baseline. The physicians were asked in a single question how often they usually consumed alcoholic beverages (including beer, wine, and liquor) and offered 7 possible responses: rarely/never; 1 to 3 per month; 1 per week; 2 to 4 per week; 5 to 6 per week; 1 per day; and 2 or more per day. We interpreted the responses to be the number of drinks consumed in the specified period. Information was also collected about other self-reported coronary risk factors, including age, cigarette smoking (never, past, or current [<20 cigarettes/d or ≥ 20 cigarettes/d]), cholesterol level, use of cholesterol-lowering medications (yes/no), systolic (SBP) and diastolic (DBP) blood pressure, frequency of vigorous exercise (≥ 1 /wk), and history of angina pectoris (yes/no) and diabetes mellitus (yes/no). Body mass index was calculated from self-reported weight in kilograms divided by the square of the height in meters.

END POINTS

Death was the study's primary end point for the entire enrollment cohort. Deaths were identified through systematic searches of the National Death Index, and death certificates were obtained for all individuals to determine the cause of death. All deaths occurring before February 1, 1988, were recorded. Trained nosologists who had no knowledge of the cohort members' alcohol consumption status classified deaths by using the first revision of the *Ninth International Classification of Diseases (ICD-9)* in conjunction with the *Automated Classification of Medical Entities Decision Tables*. Causes were assigned to deaths that occurred during a mean follow-up of 5.4 years. End points included total, total CVD, MI, stroke, other CVD, and cancer deaths.

STATISTICAL ANALYSIS

Data on frequency of alcohol consumption were a priori collapsed into 4 categories of alcohol intake: rarely/never, monthly (1-3 drinks per month), weekly (1-6 drinks per week), and daily (≥ 1 drink per day). We first calculated and compared means with 1-way analysis of variance and proportions of baseline risk factors among the alcohol intake categories. For each physician, person-years of follow-up were counted from the date when the enrollment questionnaire was returned to January 31, 1988, or to the date of death. Cox proportional hazards models were used to compute age- and multivariate-adjusted relative risks and 95% confidence intervals (CIs) for each category of alcohol use, using the relative risks (RRs) for men in the rarely/

never group as the referent. Multivariate models were adjusted for potential confounders by including terms for age, smoking, exercise, body mass index, history of angina, and diabetes. Additional analyses excluded the first 2 years of follow-up to determine if there existed possible confounding caused by recent changes in alcohol consumption due to terminal stages of illness. Linear trends were tested across the categories of alcohol consumption by using alcohol intake as an ordinal variable in the model. All statistical analyses were done using the SPSS statistical package, version 10.0 (SPSS Inc, Chicago, Ill).

RESULTS

During a mean follow-up of 5.4 years (a total of 75 710 person-years), 1018 (7.2%) of 14 125 men died. The causes of death are shown in the following tabulation:

Cause of Death	No. (%)
CVD	579 (56.9)
Acute MI	209 (20.5)
Stroke	63 (6.2)
Ischemic coronary heart disease	154 (15.2)
Sudden death	1 (0.1)
Other CVD	152 (14.9)
Cancer	224 (22.0)
Other causes	215 (21.1)

At baseline, 17% of the study population reported using alcohol rarely or never, 11% reported monthly alcohol use (1-3 drinks per month), 40% reported weekly alcohol use (1-6 alcoholic beverages per week); and the remaining 32% reported daily alcohol use (≥ 1 drink per day). Baseline characteristics of the study population are presented in **Table 1**. Men who reported daily alcohol intake tended to be older than the other men, to be former or current smokers, and to report more physical activity, less diabetes, and a greater aspirin use. Mean levels of self-reported SBP and DBP were highest in the group of daily drinkers ($P < .001$).

For total mortality, there was a significant inverse trend across alcohol intake classes in both the age-adjusted and multivariate-adjusted models ($P < .001$ for linear trend for both models). Compared with nondrinkers, weekly and daily drinkers had a significantly lower risk of death of 28% (95% CI, 14%-40%) and 27% (95% CI, 13%-39%) after multivariate adjustment (**Table 2**). Similarly, there was a reduced risk of CVD mortality with increased levels of alcohol intake in all models (P for trend $< .001$). Compared with those reporting rarely or never consuming alcohol, weekly drinkers had a multivariate RR of 0.61 (95% CI, 0.49-0.77), and daily drinkers had a multivariate RR of 0.56 (95% CI, 0.44-0.71) for CVD mortality (**Table 3**). We found no clear association between alcohol consumption and cancer mortality, but few men consumed heavy amounts of alcohol. Of the CVD deaths, 209 (36.1%) were attributed to acute MI. The relationship between alcohol consumption and death from MI was L-shaped, with a multivariate-adjusted RR of 0.55 (95% CI, 0.38-0.80) among those who reported weekly alcohol consumption, and a multivariate-adjusted RR of 0.47 (95% CI, 0.32-0.69) for daily drinkers (P for trend $< .001$) (Table 3). There were 63 deaths from stroke mortality (10.9% of CVD deaths); a nonsignificant U-shaped relation, with the lowest risk among men in the weekly consumption group, had a multivariate-adjusted RR of 0.68.

Table 1. Baseline Characteristics by Level of Alcohol Consumption Among Men With Hypertension

Patient Characteristic	Frequency of Alcohol Consumption			
	Rarely/Never (n = 2438)	Monthly (n = 1505)	Weekly (n = 5710)	Daily (n = 4472)
Age, mean (SD), y	60.7 (10.3)	58.5 (10.1)	58.4 (9.9)	61.9 (9.4)
BMI, mean (SD)	26.0 (3.9)	25.9 (3.6)	25.9 (3.4)	25.5 (3.1)
SBP, mean (SD), mm Hg	137.9 (13.7)	136.7 (13.0)	136.7 (12.5)	139.3 (13.1)
DBP, mean (SD), mm Hg	84.3 (7.4)	84.7 (7.0)	84.9 (6.7)	85.3 (6.8)
Diabetes, %	12.9	9.5	6.1	5.2
Hypercholesterolemia, %	13.8	16.8	13.6	14.9
Exercise at least once per week, %	58.5	58.9	68.2	66.9
Angina, %	8.1	6.7	6.6	7.8
Cigarette consumption, %				
Never smoked	53.0	48.7	41.9	29.3
Former smoker	36.3	41.2	48.4	55.6
Currently <20 cigarettes/d	3.4	4.9	3.6	4.6
Currently ≥20 cigarettes/d	7.4	5.3	6.1	10.6
Aspirin intake more than once per week, %	35.7	33.5	38.6	43.5

Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by the square of height in meters); DBP, diastolic blood pressure; SBP, systolic blood pressure.

Table 2. Risk of Total, CVD, and Cancer Mortality Among Men With Hypertension

	Frequency of Alcohol Consumption				P Value for Trend
	Rarely/Never (n = 2438)	Monthly (n = 1505)	Weekly (n = 5710)	Daily (n = 4472)	
Deaths, No.	252	103	321	342	
Person-years	12 738	8122	30 887	23 965	
Total mortality, RR (95% CI)					
Age adjusted	1.00	0.75 (0.59-0.94)	0.63 (0.53-0.74)	0.68 (0.58-0.80)	<.001
Multivariable adjusted*	1.00	0.86 (0.67-1.10)	0.72 (0.60-0.86)	0.73 (0.61-0.87)	<.001
CVD mortality, RR (95% CI)					
Age adjusted	1.00	0.73 (0.54-0.98)	0.56 (0.45-0.70)	0.56 (0.46-0.70)	<.001
Multivariable adjusted*	1.00	0.83 (0.62-1.13)	0.61 (0.49-0.77)	0.56 (0.44-0.71)	<.001
Cancer mortality, RR (95% CI)					
Age adjusted	1.00	0.81 (0.47-1.39)	0.87 (0.60-1.27)	1.01 (0.70-1.46)	.69
Multivariable adjusted*	1.00	1.05 (0.58-1.91)	1.07 (0.69-1.64)	1.21 (0.79-1.85)	.80

Abbreviations: CI, confidence interval; CVD, cardiovascular disease; RR, relative risk.

*Adjusted for age, smoking, diabetes mellitus, angina, body mass index, and physical activity.

There was no clear association between alcohol intake and other CVD deaths.

To test whether the effects of alcohol on mortality were sustained in the highest recorded level of alcohol intake in this population, we calculated the RRs for total and CVD mortality for the 664 men consuming 2 or more drinks a day (there were 61 deaths in this group, including 35 from CVD). For both total and CVD mortality, the significant inverse linear trends remained. Compared with nondrinkers, the men consuming 2 or more drinks a day had a multivariate-adjusted RR of 0.72 (95% CI, 0.49-1.10) for CVD mortality. The P trends in multivariate-adjusted models were significant (P for linear trend = .001 for total mortality and <.001 for CVD mortality).

We did not adjust for history of high cholesterol levels in our primary models because this risk factor may be in the casual pathway of alcohol-associated mortality. Analyses repeated with adjustment for high cholesterol levels (reported blood cholesterol level of 260 mg/dL

[6.20 mmol/L] or higher, or history of treatment for high cholesterol levels), did not materially affect RRs. After excluding the first 2 years of follow-up, the same significant inverse association between alcohol consumption and total or cardiovascular mortality was sustained.

To test whether the effects of light to moderate alcohol intake were modified by age, we calculated the RR for total mortality for men younger than 60 years and 60 years and older. In both age groups there were similar reductions in risks of total mortality, with an inverse trend across alcohol intake groups (**Table 4**).

To test whether the benefit of light to moderate alcohol intake was dependent on blood pressure control, we calculated the RR for CVD mortality for men whose self-reported blood pressure was within the goal range for controlled hypertension (SBP < 140 mm Hg and DBP < 90 mm Hg), and for a group of men whose self-reported blood pressure control was not satisfactory (SBP ≥ 140 mm Hg or DBP ≥ 90 mm Hg). The associa-

Table 3. Risk of Myocardial Infarction, Stroke, and Other Cardiovascular Disease Mortality Among Men With Hypertension

	Frequency of Alcohol Consumption				P Value for Trend
	Rarely/Never (n = 2438)	Monthly (n = 1505)	Weekly (n = 5710)	Daily (n = 4472)	
MI mortality, RR (95% CI)					
Age adjusted	1.00	0.68 (0.42-1.08)	0.57 (0.41-0.81)	0.46 (0.32-0.66)	<.001
Multivariable adjusted	1.00	0.67 (0.42-1.09)	0.55 (0.38-0.80)	0.47 (0.32-0.69)	<.001
Stroke mortality, RR (95% CI)					
Age adjusted	1.00	0.78 (0.31-1.97)	0.71 (0.36-1.37)	0.81 (0.43-1.50)	.78
Multivariable adjusted	1.00	0.80 (0.31-2.09)	0.68 (0.33-1.39)	0.74 (0.38-1.44)	.73
Other CVD mortality, RR (95% CI)					
Age adjusted	1.00	0.76 (0.41-1.44)	0.99 (0.64-1.52)	0.99 (.065-1.52)	.85
Multivariable adjusted	1.00	0.77 (0.40-1.48)	0.91 (0.57-1.45)	0.94 (0.59-1.49)	.89

Abbreviations: CI, confidence interval; CVD, cardiovascular disease; MI, myocardial infarction; RR, relative risk.

*Adjusted for age, smoking, diabetes mellitus, angina, body mass index, and physical activity.

Table 4. Risk of Total Mortality Among Men With Hypertension by Age Group

	Frequency of Alcohol Consumption				P Value for Trend
	Rarely/Never	Monthly	Weekly	Daily	
Patients aged <60 y, No.	1164	832	3218	1814	
Deaths, No.	49	27	75	55	
Total mortality, RR (95% CI)					
Age adjusted	1.00	0.82 (0.51-1.31)	0.57 (0.40-0.82)	0.65 (0.44-0.96)	.02
Multivariable adjusted*	1.00	1.02 (0.61-1.69)	0.73 (0.49-1.08)	0.77 (0.50-1.17)	.30
Patients aged ≥60 y, No.	1274	673	2493	2658	
Deaths, No.	203	76	246	287	
Total mortality, RR (95% CI)					
Age adjusted	1.00	0.72 (0.56-0.94)	0.65 (0.54-0.78)	0.68 (0.57-0.82)	<.001
Multivariable adjusted*	1.00	0.82 (0.62-1.09)	0.72 (0.59-0.88)	0.71 (0.59-0.87)	.004

Abbreviations: CI, confidence interval; RR, relative risk.

*Adjusted for age, smoking, diabetes mellitus, angina, body mass index, and physical activity.

tion between CVD mortality and alcohol intake was similar in the 2 groups (**Table 5**).

COMMENT

Our results show that light to moderate alcohol consumption in men with hypertension, defined as any history of past or current antihypertensive therapy, was inversely associated with the risk of total and cardiovascular mortality. These associations were independent of major coronary risk factors for coronary heart disease, and are consistent with earlier reports of an inverse association between light to moderate drinking and the risk of total and cardiovascular mortality in primary and secondary prevention.⁹⁻¹⁶ Therefore, the observed benefits of light to moderate alcohol intake on total and CVD mortality may extend to men with hypertension.

To our knowledge, only a few studies have examined alcohol consumption and cardiovascular mortality in hypertensive subjects.^{6,17} Palmer et al¹⁷ observed that male drinkers with hypertension had a reduced risk of stroke mortality and possibly of coronary heart disease mortality. The total mortality of drinkers and nondrinkers was not significantly different because there is an increased risk of death from noncirculatory causes with increased alcohol consumption.

A J-shaped association exists between alcohol consumption and blood pressure, and a blood pressure elevation begins with approximately 3 drinks per day for both sexes and all racial groups.¹⁸⁻²¹ Prospective observational studies²¹⁻²³ and clinical trials^{7,8} have indicated that lowering high levels of alcohol consumption (≥3 drinks per day) is associated with a reduction in blood pressure. There appears to be a J- or U-shaped relation between alcohol consumption and total mortality.^{12-16,24} These curves likely reflect benefits of light to moderate alcohol consumption on cardiovascular mortality, and detrimental effects of heavy consumption, particularly on cancer mortality.^{25,26} An examination of total and CVD mortality among the men consuming 2 or more drinks per day showed little or no association for total and CVD mortality. However, few men had 2 or more drinks per day. This may reflect the inflection point of a U- or J-shaped relation between alcohol consumption and mortality, which is consistent with the hypothesis that the detrimental effects of heavy drinking may begin to appear with 2 or 3 drinks per day.

Because the balance of adverse and beneficial effects of drinking on mortality depends on age and background cardiovascular risk, those with higher cardiovascular risk tend to have the greatest benefit of light to moderate alcohol consumption.^{10,27} When we com-

Table 5. Risk of Cardiovascular Mortality Among Men With Hypertension by Blood Pressure Level

	Frequency of Alcohol Consumption				P Value for Trend
	Rarely/Never	Monthly	Weekly	Daily	
Patients with SBP <140 and DBP <90 mm Hg, No.	975	658	2419	1605	
Deaths from CVD, No.	51	20	62	50	
Age adjusted, RR (95% CI)	1.00	0.69 (0.41-1.16)	0.60 (0.41-0.87)	0.53 (0.36-0.79)	.009
Multivariable adjusted, RR (95% CI)	1.00	0.76 (0.44-1.29)	0.62 (0.42-0.93)	0.53 (0.34-0.81)	.02
Patients with SBP ≥140 or DBP ≥90 mm Hg, No.	1348	777	3046	2698	
Deaths from CVD, No.	100	38	116	118	
Age adjusted, RR (95% CI)	1.00	0.72 (0.50-1.05)	0.59 (0.45-0.77)	0.56 (0.43-0.73)	<.001
Multivariable adjusted, RR (95% CI)	1.00	0.82 (0.56-1.21)	0.64 (0.48-0.85)	0.56 (0.42-0.75)	<.001

Abbreviations: CI, confidence interval; CVD, cardiovascular disease; DBP, diastolic blood pressure; RR, relative risk; SBP, systolic blood pressure.
*Adjusted for age, smoking, diabetes mellitus, angina, body mass index, and physical activity.

pared the results of our analysis with the similar analysis done in the whole enrollment cohort,¹⁴ thus including hypertensive and nonhypertensive men, the RRs were similar for total mortality and somewhat higher for CVD mortality, suggesting that the hypertension status has little or no effect on the effects of alcohol. The selection of subjects with hypertension, a group with higher cardiovascular risks, probably accounted for the 45% reduction in CVD mortality risk in our study. The CVD mortality risk reduction was primary driven by the significant 45% to 53% reduction in MI mortality risk. The relationship between alcohol and total mortality remained L-shaped, probably owing to light to moderate alcohol consumption levels among study participants; therefore, decreased CVD mortality risk was not offset by an increase in mortality related to heavy alcohol consumption.

To an unknown extent, elevated blood pressure may be attributable to heavy alcohol consumption in individuals with hypertension. However, we believe that very few male physicians in our study fall into this category for 2 reasons. First, only 3.2% of men in the overall Physicians' Health Study enrollment cohort (including both nonhypertensive and hypertensive individuals)¹⁴ consumed 2 or more alcoholic drinks per day, which would be deemed heavy in most epidemiological investigations and may lead to the development of hypertension. This study population of male physicians reported consuming lower amounts of alcohol than subjects in other studies. Second, some recent US studies indicate that light to moderate alcohol consumption has no adverse effects on the development of hypertension.^{28,29} Given the multifactorial etiology of hypertension, with genetic, biochemical, lifestyle, and dietary components, alcohol is but 1 factor that warrants consideration. For the above reasons, we believe deleterious effects of alcohol on hypertension in this population of hypertensive men are minimal and would have little effect on our analyses. Certainly other risk factors for hypertension—eg, physical activity, stress, diabetes, and high cholesterol level—fall into the same category as alcohol consumption and limit the extent to which we can extrapolate our results. Whether the inverse association between alcohol consumption and total and cause-specific mortality is causal remains unclear and requires additional research given its seemingly contradictory clinical recommendation.

The protective effect of moderate alcohol consumption has been suspected to be due, in part, to an increase

in the levels of high-density lipoprotein cholesterol and its subfractions.^{30,31} Alcohol has also been shown to decrease platelet aggregation^{32,33} and to increase fibrinolytic activity.³⁴ These protective mechanisms can explain the cardioprotective effects of light to moderate alcohol consumption observed in our study. We postulate that, at low levels of alcohol consumption, the cardioprotective effects of alcohol take effect while blood pressure control may still be achieved.

Our study has several potential limitations. First, alcohol intake was self-reported, and any random misclassification of alcohol intake would likely bias our relative risk toward the null, thus underestimating the observed effect. However, health professionals generally provide reliable self-reports of alcohol use.^{35,36} Second, our use of a single measure of alcohol consumption did not account for changes in alcohol intake over time. This could also lead to some misclassification, although drinking patterns were fairly stable over time in this cohort.³⁷ Third, while we have not validated the measurement of alcohol consumption in the Physicians' Health Study, we still believe that self-reported alcohol intake has reasonable validity based on studies among health professionals and stable drinking patterns over time in these men.³⁷ Fourth, we acquired information on average weekly intake with no details on drinking patterns. We could not differentiate a person having 1 drink each day from one having 7 drinks 1 day per week. Fifth, we were unable to compare the separate effects of wine, beer, and liquor consumption, but most data suggest that the benefits of alcohol are dependent on its ethanol content rather than other components.³⁸ Sixth, self-reported blood pressure and hypertension values may be subject to misclassification. However, a single self-report of blood pressure by physicians is highly correlated with measured SBP ($r=0.72$) and DBP ($r=0.60$).³⁹ In a pilot study the sensitivity of hypertension diagnoses in the Physicians' Health Study was 89%, which is consistent with results from similar cohorts of well-educated men.⁴⁰ Seventh, the reduced risk of CVD among drinkers may be explained by a tainting of the reference group, which may include former drinkers who stopped drinking because of comorbid conditions placing them at a greater risk of death.⁴¹ Although we were unable to exclude recent former drinkers, we excluded any physician who reported a history of MI, stroke, cancer, or liver disease. Finally, because

the study participants consisted of male physicians with relatively low levels of alcohol consumption, these results may not be generalized to populations with higher levels of alcohol consumption.

In summary, this study suggests that light to moderate alcohol consumption among men with hypertension is associated with a reduction in risk of total and cardiovascular mortality. The magnitude of risk reductions seems to parallel results from primary and secondary prevention studies of CVD in the general population. However, these results should be interpreted with caution and require confirmation from other studies. A discussion of alcohol intake should be a part of routine preventive counseling. In light of major clinical and public health problems associated with heavy drinking, recommendations regarding alcohol use must be made on an individual basis after carefully assessing cardiovascular risk profile and the risks and benefits of any changes in drinking behavior. However, patients with hypertension who are able to maintain light to moderate alcohol intake have no compelling reason to change their lifestyle and eliminate a possibly beneficial habit.

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