Given the social significance of alcohol worldwide it is not surprising that there is continuing strong interest in the relation between alcohol and hypertension. Recent research continues to address unresolved questions concerning the balance between the medical hazards and the cardiovascular benefits of alcohol, the possible significance of different types of beverage, the role of different drinking patterns for cardiovascular morbidity and mortality, and mechanisms underlying the pressor effects of ethanol. These issues all need to be considered in the context of social aspects of drinking and effects on noncardiovascular morbidity and mortality. Epidemiologic data relating the type or quantity of alcohol consumption to blood pressure or cardiovascular disease needs to be viewed with circumspection from a number of viewpoints. First, heavier drinkers or problems drinkers are far less likely to participate in surveys than others. Second, alcohol consumption past or present is notoriously underreported. Third, drinking patterns are difficult to quantify and often not reported at all. The type, quantity, and pattern of drinking are all highly correlated with socioeconomic and other lifestyle behaviors, many of which may not be measured or not measured accurately enough for adequate adjustments in statistical models. A relation between average weekly alcohol consumption, blood pressure level, and hypertension prevalence has been consistent worldwide and continues to be studied in different populations to evaluate its contribution in relation to other risk factors. However, because of the above issues some caveats should be put on interpretation of data where authors emphatically imply causal relationships from statistical associations.

The more recent cross-sectional studies have concentrated on the effects of pattern of drinking and the consumption of alcohol with or without food, beverage type, and the relative effects of alcohol on hypertension subtypes.

**Pattern of Drinking, Beverage Type, and Effects on Hypertension Subtypes**

The relationship between the pattern of alcohol drinking and the risk of hypertension was addressed in a cross-sectional study of 2609 New Yorkers free from other cardiovascular disease and considered further in an accompanying editorial in which some of the key issues surrounding the topic were discussed. Compared with lifetime abstainers, those who reported drinking on a daily basis or apart from food had a significantly higher risk of hypertension, but this effect disappeared after accounting for the amount of alcohol consumed in the previous 30 days. When current drinkers only were studied and adjustment made for the amount of alcohol consumed in the previous 30 days, the risk of being hypertensive increased (6%) only in those who said they drank without food. Preference for any one type of beverage did not influence the association but the important potential confounder of dietary habits was not assessed. Nevertheless this finding was consistent with an earlier study of Italian wine drinkers coming from a different cultural background, and a follow-up in that population showed drinking alcohol outside of meals was associated with higher risk of death from all causes and cardiovascular disease. Similar relationships were seen between drinking outside of meals or snacks and increased risk of myocardial infarction in a recent case control study in men.

The possible effects of individual alcoholic beverages on blood pressure continue to arouse interest. However, the previously cited study from western New York found no consistent beverage-specific associations with hypertension risk in North Americans drinking beer, wine, or spirits. A cross-sectional study in Chinese men that examined the associations between alcohol intake and isolated systolic, combined systolic and diastolic, and isolated diastolic hypertension found that those in the highest alcohol intake category (≥30 drinks/week) were twice as likely as nondrinkers to have any of these hypertensive subtypes with population attributable risks of 13.9%, 13.4% and 12.0%, respectively. In this Chinese study, liquor drinking was associated with a higher odds ratio of isolated systolic hypertension, but this finding was probably because the liquor drinkers generally drank more alcohol. Another study grouped Japanese male workers on the basis of their total consumption of beer, sake, shochu (traditional Japanese spirits), whiskey, or wine. Blood pressure was highest in the shochu group but an analysis adjusting for total alcohol consumption resulted in disappearance of this difference. The interest in the effects of specific

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**Hypertension Highlights**

**Alcohol and Hypertension**

**An Update**

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beverages has been evoked in part by the so-called French Paradox of a relatively low incidence of coronary disease in France despite a high intake of saturated fat, a phenomenon that has been attributed to the consumption of red wine.8 It has also been suggested that wine drinkers may be protected from the blood pressure–raising effects of regular moderate to heavy alcohol consumption, perhaps because of antioxidant and vasodilator effects of polyphenolic flavonoids improving endothelial function.9 In our opinion, these suggestions have been laid to rest in a randomized crossover trial10 that confirmed suggestions from population studies that moderate alcohol consumption raises blood pressure regardless of source. In that study, normotensive men showed similar elevations of awake ambulatory systolic blood pressure and heart rate after 4 weeks of either beer or red wine (40-g ethanol equivalent per day) compared with a control-abstinence period. De-alcoholized red wine had no effect on blood pressure, and neither this beverage nor alcohol containing red wine had any effect on flow or glyceryl trinitrate–mediated dilation. It was concluded that results from population studies suggesting differential effects of red wine compared with other beverages on blood pressure were most likely because of confounding lifestyle differences in wine drinkers. Twenty-four–hour endothelin-1 excretion was increased with beer and wine drinking, leading to the suggestion that this might reflect increased vascular endothelin-1 production as at least a contributor to the pressor effects of alcohol.10

A systematic review11 of alcohol intervention studies confirmed the previous findings of an initial meta-analysis by Xin et al12 with similar estimates for the effect of alcohol restriction to reduce systolic and diastolic blood pressure by 2.7 mm Hg and 1.4 mm Hg, respectively. However, these authors also compared data from studies that used conventional clinic or office review of blood pressure with those that incorporated ambulatory or home blood pressure monitoring, highlighting biphasic effects of alcohol on blood pressure with an early presumably vasodilator effect of alcohol leading to a reduction in blood pressure (in the immediate hours after exposure) and a later effect (the next day) of raising blood pressure.

**Effects on Large Vessel Structure and Function**

How might alcohol exert its coronary protective effects over and above influences on high-density lipoprotein (HDL) cholesterol, platelet function, and fibrinogen and in the face of adverse effects on blood pressure and homocysteine metabolism? Cross-sectional data from Holland described an inverse or J-shaped relation between alcohol intake and measures of aortic stiffness in middle-aged and older men and pre- and postmenopausal women13,14 but not in younger men.15 They recognized that these results might be confounded by other lifestyle factors but suggested that if they were causally linked direct effects of alcohol to improve vascular compliance might contribute to any cardioprotective effect. As discussed in an accompanying editorial,16 the findings are not easy to reconcile with the association of alcohol with isolated systolic hypertension6 nor with a 9-year longitudinal study showing an association between alcohol consumption and increased aortic stiffness in middle-aged Japanese men who were initially free of aortic stiffness.17

**Alcohol, the Metabolic Syndrome, and Diabetes**

The nature of the association between liver enzymes, body fat distribution, alcohol consumption, and the risk of hypertension has been explored further in the Western New York longitudinal study of 1455 men and women.18 It had been assumed that the increased y glutamyltransferase (yGT) levels seen in hypertensive subjects were caused alcohol. In this 6-year longitudinal study, yGT levels within the normal range were associated with incident hypertension in both drinkers and nondrinkers, but only in participants who were above median measures of fatness. The authors interpreted these findings to indicate that serum yGT may predict hypertension among individuals with increased central fat distribution, with fatty liver representing an important underlying mechanism for the association. A closely related area of interest concerns the possible link between alcohol consumption, diabetes, and the metabolic syndrome. Alcohol has been linked not only to an increase in blood pressure but to several other elements of this syndrome, in particular the increase in triglyceride levels, central adiposity, and elevated uric acid. However, alcohol simultaneously acts to increase HDL-cholesterol levels, so whether it makes any significant contribution to the metabolic syndrome has remained controversial. In the 1998 Korean National Health and Nutrition Examination Survey19 the consumption of >30 g alcohol/d was associated with an increase in blood pressure in men, a high blood glucose in women, and higher triglycerides in both men and women, whereas for both sexes and across all alcohol consumption categories there was a significant increase in HDL-cholesterol. Despite these contrasting effects on different components, overall there was a dose–response relationship between increasing alcohol intake and the odds of having the metabolic syndrome. In contrast, a report on 4510 white participants from the National Heart and Blood Institute Family Heart Study in the United States, after careful adjustment for confounders including education, diet, and physical activity,20 actually found a substantially reduced prevalence of the syndrome across all beverage types compared with “never drinkers” (odds ratio down to 0.32 for wine drinkers only). Data from the Third National Health and Nutrition Examination Survey21 also suggested alcohol consumption was inversely associated with the prevalence of several components of the syndrome, low-serum HDL cholesterol, elevated serum triglycerides, high waist circumference, and hyperinsulinemia, a finding that was strongest among whites and among beer and wine drinkers. These contrasting results from several different population studies suggest that any overall effects of alcohol on the metabolic syndrome are probably dictated by a number of competing and confounding influences, such as volume and type of alcohol consumed, gender, race, and ethnicity. Therefore, an effect of alcohol to induce hypertension and the metabolic syndrome by impairing insulin resistance is doubtful. This is supported by a randomized controlled alcohol intervention trial involving a
Safe Levels of Drinking for Hypertensive People in the Context of Alcohol and the Global Burden of Hypertensive Disease

On the basis of coronary protective effects of red wine, Bulpitt posed the question “How many alcoholic drinks might benefit an older person with hypertension?” Largely using meta-analyses from general population studies, he concluded that hypertensive patients aged >60 years who drank >16 drinks a week should be advised to reduce their consumption. This is broadly in accord with international guidelines on the management of hypertension. Some of the pitfalls of limiting the focus to coronary artery disease were discussed in an accompanying editorial. Care also needs to be taken in extrapolating data quantitatively across cultures with widely differing coronary rates and social circumstances. Connor et al showed how some ethnic communities living within Westernized populations, such as Maoris in New Zealand, show a far higher burden from death, disease, and disability from alcohol. In Japan stroke remains the most common cause of cardiovascular death, coronary deaths are relatively low, and there is a linear relation between alcohol consumption and hemorrhagic stroke. Youthfulness carries an increased share of hazards associated with alcohol caused by drinking patterns, risky behaviors, and greater periods of exposure to alcohol-related liver disease and cancers, and the age dependence of alcohol risks and benefits has been well illustrated by Jackson and Beaglehole.

Further data on the relative benefits and risks of light to moderate alcohol consumption in hypertensive patients was provided from a study of total and cardiovascular mortality in a population of 14 125 men derived from the Physicians Health Study cohort and identified with a history of past or a population of 14,125 men derived from the Physicians Health Study cohort, which found that in drinkers there was no reduction of alcohol consumption by 80% for 4 weeks in moderate drinkers, which was unable to detect any effect of changing alcohol on glucose or insulin homeostasis.

dietary differences that differentiate beer, wine, and spirits drinkers as well as marked lifestyle differences, including patterns of alcohol consumption, that are likely to have impacted on many disease processes contributing to total mortality. The most recent study measuring cardiovascular outcomes in drinking hypertensive people was in the Losartan Intervention For Endpoint reduction in hypertension (LIFE) study cohort, which found that in drinkers there was no decrease in composite cardiovascular risk when being treated with losartan compared with atenolol because a decrease in the incidence of myocardial infarction in the drinkers was offset by an increase in the risk of stroke.

In terms of the overall significance of the effects of alcohol to elevate blood pressure, an analysis from the landmark World Health Organization Global Burden of Disease 2000 Comparative Risk Analysis study assessed the risks and benefits of alcohol by region and then globally and attributed 16% of all hypertensive disease to alcohol. To add to the controversy concerning “safe” levels of drinking, Jackson et al concluded that issues of bidirectional confounding in population studies had been underestimated in relation to alcohol and coronary heart disease and that any benefits of moderate alcohol consumption on coronary disease were likely to be outweighed by harmful effects (ie, there was “probably no free lunch”).

References


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