Alcohol Consumption and Blood Pressure

The Lipid Research Clinics Prevalence Study

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SUMMARY The relationship between alcohol consumption and systolic and diastolic blood pressure (BP) was examined in 2482 men and 2301 women 20 years of age or older in nine North American populations. Men at the highest level of alcohol consumption (≥ 30 ml alcohol per day) had the highest BP, while women either at the highest level of alcohol consumption or consuming no alcohol had the highest BP. Men aged ≥ 35 years of age consuming ≥ 30 ml alcohol per day were 1.5 to 2 times more likely to be hypertensive than non-drinkers. Multivariate analysis showed systolic and diastolic BP in both men and women to be positively and significantly (p < 0.05) related to alcohol consumption, and this relationship was independent of the potential confounding effects of age, obesity, cigarette smoking, regular exercise, education, and gonadal hormone use in women. The regression coefficients indicated that an average of 30 ml of alcohol per day would produce a 2 to 6 mm Hg increase in systolic BP. Analyses suggested the univariate U-shaped alcohol-BP association in women was confounded by differences in obesity and cigarette smoking in nondrinking women, and by very low alcohol consumption in hypertensive women using medication. Additional analyses indicated that alcohol consumed in the 24 hours prior to the study was much more strongly associated with elevated BP than alcohol consumed in the week prior to the study excluding the previous 24 hours. We conclude that alcohol appears to have a modest but consistent and independent effect on systolic and diastolic BP.


KEY WORDS • hypertension • epidemiologic studies • population studies • obesity • cigarette smoking

THERE is mounting evidence from both clinical and epidemiologic studies that alcohol consumption, especially at higher levels, is associated with elevations in blood pressure (BP), both systolic and diastolic, although the elevation in systolic BP is generally of greater magnitude. Previous studies have varied greatly in their assessment of both alcohol consumption and BP, and in their assessment of potential confounding factors that might be involved in this association.

A consistent finding has been that drinkers at the highest levels of alcohol consumption show an increase in BP.\textsuperscript{1-10} Other studies have shown an increase in liver function abnormalities in hypertensive patients, presumably due to increased alcohol intake.\textsuperscript{11-13} However, some studies have indicated certain subsets of drinkers of small or moderate amounts of alcohol had BPs no higher or even lower than nondrinkers.\textsuperscript{1-4} In addition, most acute challenge studies in both animals and human subjects have shown little or no short-term effect of alcohol on BP,\textsuperscript{14-20} although positive results have been reported.\textsuperscript{21, 22} Studies of chronic alcohol administration in animals have reported no significant long-term effect on BP.\textsuperscript{23-25} If alcohol can indeed raise BP, how can the nonlinear relationship in some epidemiologic studies and the uncertain results from challenge studies be explained? Finally, many studies have left potentially important confounding variables uncontrolled.

In an attempt to better define the alcohol-BP association, we studied this relationship as part of the Lipid Research Clinics Prevalence Study. The BP was analyzed both as a continuous variable and as categorical hypertension. In the continuous analysis we controlled for the potential confounding effects of age, obesity, cigarette smoking, regular exercise, education, and gonadal hormone use in women by using multiple linear regression.
Methods

Details of the Lipid Research Clinics Prevalence Study are described elsewhere. Subjects were recruited from nine separate North American study populations. The study involved two sequential examinations. At Visit 1, fasting plasma cholesterol and triglyceride were measured, and a brief interview, including questions on age and education, was completed. A 15% random sample of participants was recalled for a much more extensive examination at Visit 2, and constitute the population for this report. A detailed medical and family history was taken, including questions on alcohol use, cigarette smoking, exercise, medications, and a 24-hour dietary recall of nutrient intake. Height (in cm) and weight (in kg) were measured. The BP was recorded four consecutive times using alternately a standard mercury sphygmomanometer and a random zero mercury sphygmomanometer. The latter device, by adding a varying increment, prevents the observer from knowing the true BP. The BP was measured in the right arm with the subject seated. The systolic and fifth phase diastolic pressures were recorded. For this analysis, the average of the two random zero systolic and diastolic recordings were used.

Alcohol intake data were obtained both from the general interview questionnaire, which assessed intake for the past week, and the 24-hour dietary recall, which assessed intake during the previous 24 hours only. The amount of beer, wine, spirits, and liqueurs consumed was converted to milliliters of ethanol using the following formula: mls of ethanol = [(no. bottles or cans of beer) (12 oz) (0.045 oz ethanol per oz beer) + (no. glasses of wine) (3 oz) (0.122 oz ethanol per oz wine) + (no. mixed drinks) (1.5 oz) (0.43 oz ethanol per oz spirits) + (no. liqueurs) (1 oz) (0.30 oz ethanol per oz liqueur)] × [29.6 ml per oz]. Participants were asked if they currently smoked cigarettes and, if so, how many cigarettes they smoked per day. Quetelet index (weight/height^2 × 1000) was employed as a measure of obesity. Age at last birthday was recorded. Antihypertensive medication use in both sexes and alcohol consumption category in men and women, unadjusted and adjusted for Quetelet, smoking, regular exercise, and gonadal hormone use in women.

This analysis was done on 2482 men and 2301 women 20 years of age or older in these nine populations who were white and had reliable data on measured variables. The number of nonwhite random sample adults was insufficient for analysis.

Results

Figure 1 shows the relationship between alcohol consumption category and systolic and diastolic BP in men and women in three age ranges, 20–34, 35–49, and ≥50 years, both unadjusted (solid line) and adjusted for differences in Quetelet, cigarette smoking, exercise, and gonadal hormone use in women among different alcohol consumption groups. The unadjusted data indicate that in men the BP was slightly higher with increased alcohol intake, but considerably higher in the heaviest drinkers. A U-shaped relationship was present for women ≥35 years of age, where the highest BPs were present in nondrinkers and the heaviest drinkers. In both men and women, these trends were more pronounced for systolic than diastolic pressure. The adjusted data show little change in men, but in women ≥35 years of age the adjusted BPs were lower in the nondrinking category and higher in the heaviest drinking category.

Figure 2 shows the proportion of hypertensives in each alcohol consumption category. Hypertension was defined as a systolic BP ≥140 and/or a diastolic
Table 1. Proportion of Subjects on Antihypertensive Medication by Age and Alcohol Consumption Category in Men and Women

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Men, alcohol consumption (ml/day)</th>
<th>Women, alcohol consumption (ml/day)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>20-34 yrs (n)</td>
<td>25-50 yrs (n)</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>151</td>
</tr>
<tr>
<td>Medication (%)</td>
<td>0.7</td>
<td>2.4</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>121</td>
</tr>
<tr>
<td>Medication (%)</td>
<td>0</td>
<td>3.7</td>
</tr>
<tr>
<td></td>
<td>20</td>
<td>87</td>
</tr>
<tr>
<td>Medication (%)</td>
<td>1.3</td>
<td>3.8</td>
</tr>
</tbody>
</table>

Table 1 shows in each specific age-sex-alcohol consumption group the proportion of subjects taking antihypertensive medications. In men, the heaviest drinking category has the highest proportion of antihypertensive medication users. In contrast, nondrinking women had the highest proportional antihypertensive medication use.

Figure 3 further explores the observed sex difference in Table 1, and shows the average alcohol consumption in milliliters per day in age-sex specific groups for normotensives, hypertensives not taking medication, and hypertensives taking medication. In men, normotensives consumed the least alcohol, hypertensives not on medications somewhat more, and hypertensives on medication the most. By contrast, women hypertensives on medication consumed the least alcohol, and normotensives and hypertensives not taking medication had similar consumption levels.

The multiple linear regression model used in the multivariate analysis was systolic (or diastolic) blood pressure $\geq$ 90 mm Hg and/or use of antihypertensive medication.
BP = B or B2 • age + B3 • alcohol + B4 • Quetelet + B5 • smoking + B6 • exercise + B7 • education + B8 • gonadal hormone use (women only) + B9 • clinic. The clinic term is a class variable that identifies each of
the study populations surveyed by nine North American Lipid Research Clinics. It was included in the regression model, in order first to test for homogeneity of effects among the study populations and, having excluded interactions, to adjust for differences between the populations. Separate analyses were done for systolic and diastolic BP.

For the multivariate analysis, men of all ages were combined, while women were divided into those 20-49 and ≥ 50 years of age and analyzed separately. This latter division was to account for the different gonadal hormone medications used in these age groups, predominately oral contraceptives before age 50 and predominately postmenopausal estrogens thereafter. Table 2 shows the regression coefficients for the major variables of interest for this analysis. In both men and women age, alcohol, and Quetelet were all significantly and positively associated, and cigarette smoking significantly and negatively associated, with systolic BP. An exception was cigarette smoking in women ≥ 50 years of age where the p value was ≥0.09, a reflection of the smaller number of subjects in this analysis, since the magnitude of the coefficient was −0.16, larger than the coefficient for men or younger women. Multiplying the regression coefficients in tables 2, 3, and 4 by the following units gives the predicted change in BP in mm Hg for variable change of one unit: age = years, Quetelet = integers, alcohol = ml per day, and cigarettes = cigarettes per day. The coefficients for alcohol ranged from 0.07 to 0.16, suggesting that, on the average, a 30 ml (± 1 oz) intake of alcohol per day is associated with systolic BP 2 to 6 mm Hg higher than in a nondrinker. The multivariate analysis for diastolic BP showed similar results although, as expected, the magnitude of the coefficients tended to be smaller. The associations of systolic and diastolic BP with education, exercise, and gonadal hormone use in women will be detailed in a separate report.

Table 3 shows separate regressions for systolic BP deleting users of antihypertensive medication. This additional analysis was done because of the suggestions in table 1 and figure 3 that hypertensive women, especially those on therapy, may behave differently from normotensive women with respect to alcohol consumption. Results for men and younger women were essentially unchanged from the results shown in table 2. In women ≥ 50 years of age, excluding users of antihypertensive medications increased the magnitude of the regression coefficient for alcohol 31% for systolic and 33% for diastolic BP, and similarly increased the statistical confidence in the estimate.

An attempt was made to determine if the effect of alcohol was greater if ingested recently. Separate regression models were run which were identical to those shown in table 2 except that alcohol was replaced by two terms: alcohol in the previous 24 hours ("alc 24"), and alcohol in the previous week minus the amount in the previous 24 hours ("alc minus"). The results are shown in table 4. The numbers of subjects included in the regression models are slightly smaller than in table 2 because of the exclusion of a few subjects with unreliable dietary recalls. From the magnitude of these coefficients, the effect of alcohol consumed in the previous 24 hours appears at least three times as great as all the alcohol consumed in the rest of the previous week. In men, the coefficients for both "alc 24" and "alc minus" contributed independently and additively to systolic and diastolic BP. In women, only the "alc 24" coefficient in the younger women for systolic BP was statistically significant. Again, the number of women ≥ 50 years of age was relatively small for multivariate analysis. To directly compare the magnitude of the alcohol co-

### Table 2. Multiple Regression Coefficients and Statistical Significance for the Associations of Age, Alcohol, Quetelet, and Cigarette Smoking with Systolic and Diastolic Blood Pressure in Men and Women

<table>
<thead>
<tr>
<th>Variable</th>
<th>Men ≥ 20 (n = 2376)</th>
<th>Women 20-49 (n = 1461)</th>
<th>Women ≥ 50 (n = 693)</th>
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<tr>
<td></td>
<td>Reg coef</td>
<td>p value</td>
<td>Reg coef</td>
</tr>
<tr>
<td>Systolic BP</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Age</td>
<td>0.41</td>
<td>0.0001</td>
<td>0.29</td>
</tr>
<tr>
<td>Alcohol</td>
<td>0.10</td>
<td>0.0001</td>
<td>0.07</td>
</tr>
<tr>
<td>Quetelet</td>
<td>9.98</td>
<td>0.0001</td>
<td>10.51</td>
</tr>
<tr>
<td>Cigarettes</td>
<td>-0.08</td>
<td>0.0002</td>
<td>-0.11</td>
</tr>
<tr>
<td>Diastolic BP</td>
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<td></td>
<td></td>
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<tr>
<td>Age</td>
<td>0.18</td>
<td>0.0001</td>
<td>0.29</td>
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<tr>
<td>Alcohol</td>
<td>0.06</td>
<td>0.0001</td>
<td>0.06</td>
</tr>
<tr>
<td>Quetelet</td>
<td>8.36</td>
<td>0.0001</td>
<td>7.77</td>
</tr>
<tr>
<td>Cigarettes</td>
<td>-0.06</td>
<td>0.0003</td>
<td>-0.09</td>
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efficients in tables 2 and 3 with those in table 4, the former coefficients should be divided by 7 since they reflect the increase in BP from average daily alcohol consumption rather than for the total weekly consumption measured. Thus, the coefficients for “alc 24” are uniformly larger, and the coefficients for “alc minus” uniformly smaller, than those for “alcohol.”

Additional regressions were done examining the effect of alcohol on BP in small groups of persons reporting consumption of only one type of alcoholic beverage: beer only, or wine only, or spirits only. These analyses were limited by the small numbers of subjects, but the coefficients for beer and spirits were positive and statistically significant for men for both systolic and diastolic pressure, while the wine coefficient was positive for systolic and negative for diastolic pressure, neither result being statistically significant. None of the individual beverage results for women was statistically significant, but the beer and spirits coefficients were positive for both systolic and diastolic pressures while the wine coefficients for both pressures were negative.

Discussion

Our unadjusted data show increased BP with increasing alcohol consumption in men and a J- or U-shaped relationship in women (fig. 1). Similar relationships have been reported previously from large population studies. In the study by Klatsky et al., also cross-sectional in nature, the associations for men were more linear than our data and in women were more J-shaped than U-shaped. These differences possibly reflect alcohol consumption categories. Our highest consumption category (≥ 30 ml per day) is roughly equivalent to intermediate alcohol consumers in the Klatsky study. Thus, the trends are similar, but the range of alcohol consumption in our study is more limited.

The adjusted curves in figure 1 for men are essentially unchanged from the unadjusted curves, but for women ≥ 35 years of age, the BP in nondrinkers is reduced and the BP in the heaviest drinking category is increased. The adjusted curves in women reflect more obesity and less smoking in nondrinking women compared to women in the highest alcohol consump-

<table>
<thead>
<tr>
<th>Variable</th>
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<th>Women 20–49 (n = 1436)</th>
<th>Women ≥ 50 (n = 570)</th>
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<td>Reg coeff</td>
<td>p value</td>
<td>Reg coeff</td>
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<td>Systolic BP</td>
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<td>Age</td>
<td>0.35</td>
<td>0.0001</td>
<td>0.30</td>
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<td>Alcohol</td>
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<td>0.0001</td>
<td>9.62</td>
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<td>Cigarettes</td>
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<td>0.0001</td>
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<tr>
<td>Diastolic BP</td>
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<td>Age</td>
<td>0.16</td>
<td>0.0001</td>
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<td>-0.09</td>
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<table>
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<tr>
<th>Variable</th>
<th>Men ≥ 20 (n = 2334)</th>
<th>Women 20–49 (n = 1436)</th>
<th>Women ≥ 50 (n = 673)</th>
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<tr>
<td></td>
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<td>p value</td>
<td>Reg coeff</td>
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<tr>
<td>Systolic BP</td>
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<td></td>
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<tr>
<td>ALC 24</td>
<td>0.027</td>
<td>0.0001</td>
<td>0.033</td>
</tr>
<tr>
<td>ALC minus</td>
<td>0.010</td>
<td>0.0001</td>
<td>0.004</td>
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<tr>
<td>Diastolic BP</td>
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</tr>
<tr>
<td>ALC 24</td>
<td>0.019</td>
<td>0.0001</td>
<td>0.015</td>
</tr>
<tr>
<td>ALC minus</td>
<td>0.005</td>
<td>0.0029</td>
<td>0.004</td>
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</table>
tion category. Since women nondrinkers tended to be more obese than drinkers, and our study confirmed (table 2) the well-known obesity-BP correlation, nondrinking women would have selectively higher BP due to more obesity. In addition, they were less likely to smoke cigarettes, and cigarettes had an independent negative relationship to BP in multivariate analysis (table 2). The effect of cigarettes is discussed in more detail later in this paper. Nondrinking men also smoked less, but the differential was not as great as in women, and nondrinking men were not more obese than drinkers. Nondrinkers have been shown to be more likely to be nonsmokers in most studies. Why nondrinking women should be more obese is not clear but may reflect a complex interaction of factors.

We attempted to determine if high alcohol intake was a marker for hypertension, defined as a systolic BP $\geq 140$ and/or a diastolic BP $\geq 90$ and/or use of antihypertensive medication. These results also indicated a positive association in men and a U-shaped relationship in women. Other studies have suggested that "problem drinkers" are about twice as likely to be hypertensive as controls, and the Klatsky study showed a doubling of hypertension in whites and an approximately 50% increase in blacks in subjects drinking six or more drinks a day as compared to nondrinkers. Since hypertension is established by a cutpoint at the extreme of a normal distribution, a shift of that distribution to the right even by a small amount will cause a considerable increase in the area under the curve above the cutpoint. Thus, the effect of alcohol, even if modest, will lead to a large increase in those falling into the hypertensive category.

In addition to a greater Quetelet and less smoking, nondrinking women were different in another way that may help explain the U-shaped curve. The data in table 1 show that, in contrast to men, nondrinking women were the group most likely to be taking antihypertensive medication. Figure 3 further explores this phenomenon. In men, the group with presumably the most severe hypertension, i.e., those taking medication, consumed the most alcohol, consistent with an effect of alcohol on BP. Hypertensives not taking medication were intermediate in alcohol consumption between medication takers and normotensives, again consistent with the hypothesis. In women, the situation was markedly different. Normotensives and hypertensives not taking medications had similar alcohol consumption levels, while hypertensives taking medications drank very little, less than 4 ml per day or less than 1 oz per week (29.6 ml) in all age groups.

The data for women could suggest either that some alcohol intake reduces the probability of hypertension in women or, alternatively, that a behavioral selection bias exists; i.e., hypertensive women, especially those on medications, drink less because of their hypertension. The latter seems more likely unless we postulate that somehow alcohol differentially affects BP in men and women. This behavioral selection bias appears to be independent of and additive to the effects of obesity and (non)-cigarette smoking in producing high BP in nondrinking women, since in the multivariate analysis the regression coefficients for alcohol increased for older women when users of antihypertensive medications were eliminated from the analysis. Perhaps women who are on antihypertensive therapy are more likely to comply with health-related admonitions from physicians, peers, or other sources of information. It is also possible they may be less likely to report their true alcohol consumption in a medical study if they have also reported antihypertensive therapy. Whether the drinking is absent or underreported, the result is the same; high BP will be recorded in reported nondrinkers. Thus, the U-shaped curve in women probably reflects differences in obesity and cigarette smoking between alcohol consumption categories; selective absence or underreporting of drinking in hypertensive women, especially those on medication; and a positive association between alcohol and BP similar to that observed in men, rather than some unusual physiologic phenomenon.

We did not adjust the curves in figure 1 for antihypertensive medication use, since it was not intuitively clear exactly how this adjustment should be made. It is interesting to note, however, that if one were to eliminate all antihypertensive medication users from figure 1, the effect should theoretically be to reduce the association, assuming that alcohol use promotes BP. However, table 1 shows the opposite would be true for women $\geq 35$ years of age since BP would be reduced in nondrinkers much more than in any of the drinking categories, and least of all in the heaviest drinkers. The multivariate analysis in table 3 suggests this, as well. Overall, we feel alcohol and BP are positively associated in both men and women, although there may be a threshold of about 20 or 30 (reported) ml of alcohol per day, or 1½ to 2 drinks, before a noticeable effect is seen.

The multivariate data in table 2 demonstrate a strong relationship between age and BP and obesity and BP, relationships amply documented in many previous studies. Cigarettes show a negative relationship to BP, such that smoking a pack of cigarettes a day is associated with a systolic BP reduction of 2 to 3 mm Hg. Cigarettes have been inconsistently related to BP in previous studies, but the most common finding is a weak, negative relationship in studies that have controlled for the effect of obesity simultaneously. However, a recent report indicated small and inconsistent changes in BP in persons quitting smoking. Smoking increases BP acutely, and it has been suggested that smokers not smoking before or during a health study examination might experience a downward rebound in BP. Another possibility is that the known negative association between smoking and obesity has somehow been inadequately controlled in our analysis, and smoking is serving as a surrogate variable for additional unquantified "thinness." A behavioral bias might also be true for cigarettes. If hypertensive men and women are less likely to smoke due to medical or other influences, or are less likely to report smoking if they do smoke, at least part of the negative association of
smoking with BP might be spurious. However, the data for smoking in table 3 do not support this hypothesis.

The analyses in table 4 indicated a much greater effect on BP for alcohol consumed during the 24 hours before the study than alcohol consumed earlier. This suggests that most, but perhaps not all, of the effect of alcohol is from relatively recent consumption. It is possible that subjects might have been somewhat more accurate in their recall of alcohol consumed during the past 24 hours than during the previous 6 days, but it is unlikely this alone would account for the dramatic differences in regression coefficients. Though men did report somewhat more alcohol consumed in the previous 24 hours than would be expected from their weekly consumption, it was reassuring to us that women did not. These data may help clarify the mechanism of the effect of alcohol on BP. It appears that most of the increase in BP occurred from alcohol consumed during the previous 24 hours. However, our subjects were asked to fast for 12 hours before the study, so presumably none had drunk alcohol during that period. Thus, much of the BP increase could be related to alcohol withdrawal. Urinary excretion of epinephrine is greater during withdrawal than during alcohol administration, and plasma norepinephrine levels are highest 13 to 24 hours after alcohol cessation. In addition, plasma arginine vasopressin and plasma renin activity are increased during the withdrawal phase. Whether the effect on BP begins during the alcohol intoxication or withdrawal period is more physiologic than clinical interest, since either way the effect is due to alcohol. If one drinks, one must eventually withdraw. In addition, there is little comfort in the knowledge that the effect of alcohol is primarily short-term, since a repeated acute effect is essentially a chronic one.

It has been suggested that stress is a possible factor that might independently lead to both heavy drinking and hypertension, and thus alcohol might erroneously appear to be causal. Similarly, hypertension and alcoholism appear to have strong genetic components, and if the genetics of these conditions were somehow linked this could also result in an erroneous association. However, our evidence that alcohol consumed in the previous 24 hours is more important than that consumed earlier in the week tends to suggest a direct physiologic effect.

It is unclear whether the type of beverage consumed is important. Analyses of subjects drinking beer only or spirits only showed higher BP with increased alcohol. Subjects drinking wine only were the exception, but the range of intake in this group was small, the number of subjects was small, and the coefficients were not statistically significant. Further studies of these issues with larger numbers of subjects would be helpful in determining if different alcoholic beverages differentially affect BP. Other studies have attempted to examine wine, beer, and liquor separately in studying coronary heart disease, but did not attempt to isolate subjects drinking only one kind of beverage. Isolation of subjects drinking only one type of alcohol would seem to be crucial since heavier drinkers of a given type of alcohol might be heavier drinkers of another type of alcohol as well.

Our results indicate a relatively modest influence of alcohol on BP, in that they can be converted to suggest that about two drinks per day (30 ml of alcohol) will produce a 2 to 6 mm Hg increase in systolic BP, by whatever mechanism. These kinds of cross-sectional data need to be interpreted cautiously. First, alcohol consumption is usually underreported, some heavy drinkers completely denying any intake. The potential effect of such a bias is complex. If heavy drinkers reported little or no alcohol intake, our data would be an underestimate of the true alcohol effect. However, if most subjects tended to underreport alcohol intake in a proportional manner, our data might be an overestimate of the true alcohol effect since the observed increase in BP in each successive drinking group would actually be attributable to more alcohol than was reported. Probably both of the above biases occurred with our subjects, but the former may well have been dominant, and our data are probably not an overestimate of the true alcohol effect. Second, our data are averages that apply to populations, not individuals. It is possible that the BP of some persons may be affected more by intoxication and/or withdrawal than others, and this might be important clinically. Third, most of our subjects reported relatively low alcohol consumption, and thus our data cannot directly address the problem of extreme alcohol intakes as in alcoholism.

Recent evidence has suggested that moderate alcohol consumption may be protective against the development of coronary heart disease, perhaps by elevating high density lipoprotein cholesterol. A prepaid health plan study also suggested a protective effect, in that persons drinking six or more drinks daily had a decrease in hospitalizations for all coronary events. However, this same group had an increase in total hospitalizations, and hospitalizations for other cardiovascular disease, stroke, and hypertension. Similarly, a study from Honolulu showed a reduction in coronary mortality with drinking, but an increase in stroke, cancer, and cirrhosis of the liver deaths. A study from Finland of brain infarctions in patients less than 40 years of age showed that victims were 2 to 4 times more likely than the general population to have had a bout of alcohol drinking within 24 hours.

It seems clear that any putative protective effect of alcohol in coronary disease must be balanced against the noxious effect of alcohol in contributing to stroke and total mortality. Alcohol contributes to total mortality by affecting biology, physiology, and behavior. Our data suggest that one effect of alcohol is to promote hypertension. The risk of elevated BP for mortality has been demonstrated in the Framingham study and other epidemiologic studies, and has recently been dramatically illustrated in two intervention trials. Thus, the concern over alcohol's effect on BP, even though this effect may be modest, seems appropriate.
Acknowledgments

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Lipid Research Clinics Epidemiology Committee


Lipid Research Clinics Directors Committee


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