Alcohol Consumption as a Risk Factor for High Blood Pressure
Munich Blood Pressure Study

VICTORIA CAIRNS, PH.D., ULRICH KEIL, M.D., PH.D., DAVID KLEINBAUM, PH.D.,
ANGELA DOERING, M.D., AND JUTTA STIEBER, M.D.

SUMMARY The Munich Blood Pressure Study (MBS), a 1980-81 cross-sectional study (with follow-up) of a random sample of 3198 Munich citizens aged 30-69 years (response rate 69%), revealed hypertensive blood pressure (BP) values in 17.7% of men and 10.7% of women (WHO criteria). One of the main goals of the MBS was to search for social, behavioral, and environmental risk factors for hypertension. The relationship between BP and five possible risk factors — alcohol consumption (g/day), cigarette smoking, oral contraceptive use, years of education, obesity (BMI) — has been examined. The major emphasis of this report is the relationship of alcohol consumption to BP. Multiple linear and logistic regression analyses were run controlling for both age and sex. All second- and third-order interactions between the independent variables were tested during a backward-stepping procedure. Alcohol consumption appeared as a significant main effect in many of the analyses. The coefficient of the alcohol variable ranged from 0.02 to 0.06 for men and women in the separate linear regression analyses for systolic and diastolic BP. Thus, for example, according to the model, the daily consumption of 1 liter of beer (40 g alcohol) may cause an increase in diastolic BP in women of 2.4 mm Hg. (Hypertension 6: 124-131, 1984)

KEY WORDS • hypertension • epidemiologic studies • multiple regression • obesity • cigarette smoking • risk factors

A n association between heavy drinking and hypertension was first reported in 1915 for French servicemen. Since then, many observational studies in community, industry, and clinic populations have found alcohol consumption to be associated with increased blood pressure (BP) and hypertension. The acceptance of this association as causal has been hampered by the difficulty in distinguishing between the effects of alcohol itself and those of various accompanying lifestyle factors, such as smoking, obesity, and educational attainment. It is also possible that both hypertension and alcoholism develop secondary to stress. The major purpose of this paper is to consider the alcohol-blood pressure association in a multivariate context. Moreover, since the relationship between alcohol consumption and hypertension had not been investigated in the Federal Republic of Germany, these findings can be added to those of other countries.

From the Department of Epidemiology, Medis Institute, Research Center GSF, Munich, Germany (Drs. Cairns, Keil, Doering, and Stieber) and the Department of Biostatistics, University of North Carolina, Chapel Hill, North Carolina (Prof. Kleinbaum).
Address for reprints: Dr. Victoria Cairns, Department of Epidemiology, Medis Institute, Research Center GSF, Ingolstaedter Landstrasse 1, 8042 München-Neuherberg, F.R. Germany.
Received April 6, 1983; revision accepted August 4, 1983.
pletion of the interview so that each person had been in a sitting position for about 30 minutes before the first BP recording. There were intervals of 3 minutes between each of three BP measurements, which were performed under the standardized conditions of the American Heart Association. Each time, the 1st, 4th, and 5th phases of the Korotkoff sounds and the pulse rate were recorded. Three cuff sizes (13 × 23 cm, 13 × 28 cm, 14 × 35 cm) were used according to the circumference of the right upper arm. All BP data were based on the 1st and 5th phases of the Korotkoff sounds and on the calculation of the mean of the second and third BP measurements from the one occasion.

Each subject was asked how much beer, wine, and spirits he or she had drunk on the previous workday and over the previous weekend. The following conversions were made:

1 liter beer = 40 g alcohol;
1 liter wine = 100 g alcohol;
1 shot spirits (0.02 liters) = 6.2 g alcohol.

From this, the average number of grams of alcohol consumed per day was derived. Alcohol consumption was treated both as a continuous variable and as a categorical variable. Two different, dichotomous variables (ALC1 and ALC2) were created using cutoff points of 40 and 60 g of alcohol per day, respectively. Since only 2.8% of the women drank 60 g of alcohol or more per day compared to 24.8% of the men, the variable ALC2 was not used for the women.

The body mass index (BMI) was calculated as weight (kg) divided by height (m) squared. In defining the dichotomous smoking variable (SMK), a smoker was defined as someone who smokes now or who gave up smoking within the last 6 months, and who smokes or smoked more than 5 cigarettes per day. Analyses were also conducted including as smokers those people who smoke or smoked one to five cigarettes per day and/or pipes or cigars. Other variables included the number of years of education and self-reported oral contraceptive or noncontraceptive estrogen use.

Statistical Methodology

To examine the relationship between BP and the possible risk factors mentioned above, multiple linear and logistic regression analyses were run on the MBS data when controlling for age. The two dependent variables were systolic BP and diastolic BP, with analyses always done separately for each dependent variable as well as separately for men and women. Independent variables that were considered included age, BMI, alcohol consumption, smoking, years of education, and oral contraceptive use. Age was viewed as a control variable, while the other variables were viewed as possible risk factors for high BP.

Both linear and logistic regression models were fitted, each of which initially included all independent variables and all second- and third-order interactions. A backward stepping strategy was undertaken to determine a best model, which involved eliminating insignificant variables in a hierarchical manner. In all analyses, adjustments were made to the significance level, $\alpha = 0.05$. The use of a hierarchical strategy in variable selection, together with adjustment of the significance level according to the number of tests being made, represents the principal methodological contribution of our analyses when compared to past studies on risk factors for hypertension (see Appendix).

Analyses were performed that both included and excluded subjects taking antihypertensive medication (9.7% of men and 13.7% of women). Additional analyses (including and excluding subjects on antihypertensive medication) were carried out on a restricted subset of subjects under 60 years of age (60 men and 79 women were on medication, and 798 men and 851 women were not on medication). Hypertensives on medication were excluded since their blood pressures were artificially lowered and because they had been observed to drink less alcohol than those not on medication (Figure 1). The restriction to subjects under 60 years of age was made to minimize the possible effect of selective survival, since those at high risk (older, overweight, with high BP) may have died before they could be included in this cross-sectional study. Evidence for selective survival in men was found from comparison of regression lines relating BP to BMI for different age categories; in men over 60, the relationship between BMI and BP was weaker. This would happen if the older, overweight men with high blood pressure died before they could be included in the study.

![Figure 1. Average daily alcohol consumption by men and women, and proportion reporting antihypertensive medication use.](http://hyper.ahajournals.org/Downloaded from http://hyper.ahajournals.org/)

Downloaded from http://hyper.ahajournals.org/ by guest on May 29, 2017
Results

Alcohol Consumption and Blood Pressure

Figure 1 shows the alcohol consumption of men and women. The percentage of subjects on antihypertensive medication by alcohol consumption category is also shown. The proportion taking antihypertensive medication was very small among these subjects consuming 40 g of alcohol or more per day. About 12% of the men and 32% of the women did not drink any alcohol at all. Of the men, 24.8% drank 60 g of alcohol or more per day compared to only 2.8% of the women.

The mean systolic and diastolic BP values of men and women drinking different amounts of alcohol are shown in Figure 2. It can be seen that systolic and diastolic BP values are higher in men who drank 60 g of alcohol or more per day. For women, the curve is unclear with regard to systolic BP, but, as for men, a rise in diastolic BP is seen in the group of women drinking the most alcohol.

In Figures 3 and 4, the men and women have been stratified into 10-year age groups. Again, a slight tendency for higher systolic and diastolic BP values in men drinking 60 g of alcohol or more per day can be seen in nearly all age groups (Figure 3). A clear trend for higher diastolic BP with increasing alcohol consumption is seen for women in the age groups 30–39 and 60–69 (Figure 4). The picture is less clear for systolic BP values.

Figure 5 shows the prevalence of hypertension by alcohol consumption category and by age and sex. In men and women aged 30–39 and 40–49 years, there was a tendency for an increase in the proportion of hypertensives with greater alcohol consumption. In the age groups 50–59 and 60–69 years, such a tendency was found neither in men nor in women.

Multiple Regression Analyses

The above analyses on the BP-alcohol relationship do not take into account other risk factors that are potential confounders, such as BMI, smoking, years of education, and oral contraceptive use, which may be related to both BP and alcohol consumption. Therefore, multiple linear regression analyses were per-
formed, treating BP (SBP and DBP separately) as the dependent variable, and age, BMI, smoking, alcohol consumption, oral contraceptive use, and years of education as the independent variables. The variable years of education dropped out of the models as insignificant, so years of education is excluded in the following results. Addition of the variable oral contraceptive use or noncontraceptive estrogen use did not affect the coefficient of the variable alcohol in the models for SBP and DBP in women. These results will be reported elsewhere.

Table I contains the coefficients of the variables found from the “best-fitting” linear regression models when all subjects on antihypertensive medication are excluded. The results for all subjects are similar. The partial F values appear in brackets to the right of the coefficients. Only those interaction terms that were significant are listed. The adjusted significance levels, α/k, were strictly followed.

Systolic Blood Pressure in Men

The continuous alcohol variable appeared as a significant main effect in the model for systolic BP in men (but in no significant interaction terms) in the analyses using all men aged 30–69 years and using only men aged 30–59 years. In both analyses, the coefficients of the alcohol variable were positive: 0.04 and 0.05, respectively. The results for all men showed that the consumption of 60 g of alcohol per day was associated with a predicted increase in systolic BP of 2.4 mm Hg; within the age group 30–59 years, an increase in SBP of 3.0 mm Hg was predicted.

The dichotomous alcohol variables, ALC1 and ALC2, dropped out of the models for SBP, all ages combined, but ALC1 remained in the model for the age group 30–59 years. In the latter case, the coefficient of ALC1 was 2.4, indicating a predicted increase of 2.4 mm Hg in SBP in those men under 60 who drank 40 g of alcohol or more per day.

The variable BMI remained in the model in all analyses as a significant main effect. No interaction terms were significant. The variable SMK did not remain in the model either as a main effect or in an interaction term. Age, BMI, and alcohol consumption explained 12% to 13% of the variance of SBP in men.

Diastolic Blood Pressure in Men

The continuous alcohol variable appeared as a significant main effect in the model for diastolic BP in men in the analyses for all men and in those for men aged 30–59 years. Again the coefficients were positive (0.02 and 0.03, respectively), indicating that those
consuming more alcohol had higher DBP values. Again, when alcohol consumption was treated as a dichotomous variable (ALC1 and ALC2), it dropped out as insignificant from all of the models for DBP for all ages combined, but for the age group 30–59 years ALC2 appeared as a significant main effect, with a coefficient of 1.9.

The variable SMK appeared in a significant interaction term with BMI, which could have been caused by overweight smokers with high blood pressure dying before they could be included in the study. The BMIxage interaction term was also significant in the analyses for all men (30–69 years), again indicating possible selective survival. According to the model, consumption of 40 g of alcohol or more per day was associated with an increase in DBP in men on the average of 0.8 to 1.2 mm Hg.

**Systolic Blood Pressure in Women**

The variable BMI remained as a significant main effect in all models for systolic BP in women. The continuous alcohol variable and the dichotomous variable ALC1 did not remain in the models either as main effects or in interaction terms. The variable SMK also did not remain in the models either as a main effect or in an interaction term. All the terms involving alcohol and SMK dropped out as insignificant in the analyses involving all women and involving only those women under 60 years of age. Age and BMI explained 20% to 24% of the variance of SBP in women.

**Diastolic Blood Pressure in Women**

The continuous alcohol variable appeared as a significant main effect in the model for diastolic BP in women. The analyses using all women and in the analyses using only those under 60 years of age. In

---

**Table 1. Coefficients of the Variables and Partial F Values (in brackets) from the "Best-Fitting" Linear Regression Models, with Systolic and Diastolic Blood Pressures as the Dependent Variables, for Men and Women Separately Excluding All Those on Antihypertensive Medication**

<table>
<thead>
<tr>
<th>Blood pressure</th>
<th>Age</th>
<th>BMI</th>
<th>Alcohol</th>
<th>SMK</th>
<th>Constant</th>
<th>BMI × SMK</th>
<th>BMI × Age</th>
<th>R²</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Systolic</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30–69 yrs</td>
<td>0.31</td>
<td>1.18 (56.0)</td>
<td>0.04 (9.8)</td>
<td></td>
<td>(0.6)</td>
<td>84.94</td>
<td></td>
<td>0.12</td>
</tr>
<tr>
<td>30–59 yrs</td>
<td>0.32</td>
<td>1.37 (70.3)</td>
<td>0.05 (12.0)</td>
<td></td>
<td>(1.2)</td>
<td>79.16</td>
<td></td>
<td>0.13</td>
</tr>
<tr>
<td>Women</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30–69 yrs</td>
<td>0.56</td>
<td>1.17 (81.7)</td>
<td></td>
<td>(2.9)</td>
<td>(0.0)</td>
<td>67.80</td>
<td></td>
<td>0.24</td>
</tr>
<tr>
<td>30–59 yrs</td>
<td>0.55</td>
<td>1.22 (77.0)</td>
<td></td>
<td>(2.9)</td>
<td>(0.0)</td>
<td>67.28</td>
<td></td>
<td>0.20</td>
</tr>
<tr>
<td><strong>Diastolic</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30–69 yrs</td>
<td>1.16</td>
<td>3.31</td>
<td>0.02 (6.0)</td>
<td>20.36</td>
<td>-2.97</td>
<td>-0.82 (13.0)</td>
<td>-0.05 (21.9)</td>
<td>0.10</td>
</tr>
<tr>
<td>30–59 yrs</td>
<td>0.11</td>
<td>1.35</td>
<td>0.03 (7.6)</td>
<td>20.05</td>
<td>42.27</td>
<td>-0.80 (11.7)</td>
<td></td>
<td>0.13</td>
</tr>
<tr>
<td>Women</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30–69 yrs</td>
<td>0.09</td>
<td>0.78 (81.7)</td>
<td>0.06 (11.6)</td>
<td></td>
<td>(0.0)</td>
<td>51.39</td>
<td></td>
<td>0.11</td>
</tr>
<tr>
<td>30–59 yrs</td>
<td>0.21</td>
<td>0.82 (11.1)</td>
<td>0.07 (11.1)</td>
<td></td>
<td>(0.4)</td>
<td>45.36</td>
<td></td>
<td>0.14</td>
</tr>
</tbody>
</table>

BMI = body mass index; SMK = smoking.
both analyses, the coefficient for the variable alcohol was positive: 0.06 and 0.07, respectively. Thus, for all women, the consumption of 40 g of alcohol per day was associated with a predicted increase in DBP of 2.4 to 2.8 mm Hg. The variable SMK did not remain in the models for women either as a main effect or in an interaction term. As expected, BMI was a significant main effect. Age, BMI, and alcohol consumption explained between 11% and 14% of the variance of DBP in women.

**Systolic and Diastolic Blood Pressure in Total Group**

Including all those people taking antihypertensive medication in the analyses did not markedly change the results. Some slight differences appeared in the analyses of DBP in women, however. The coefficient of the continuous alcohol variable decreased from 0.06 to 0.05 when all ages were considered and from 0.07 to 0.06 when women under 60 years were analyzed.

The different results with regard to alcohol consumption and BP in men and women may be due to the small number of subjects in certain extreme cells. The women and particularly the older women tended to drink and smoke less than men. Thus, if too few women in the sample were drinking heavily, it may not be possible to find a significant relationship between BP and alcohol consumption in women. This argument is supported by the finding that the confidence intervals for the coefficients of the variable alcohol as a main effect were seen to be twice as large for the women as for the men (Table 2).

![Figure 5](http://hyper.ahajournals.org/) Percentage of men and women who are hypertensive by age and alcohol consumption category.
TABLE 2. 95% Confidence Intervals for the Coefficient of the Variable Alcohol (g/day) from the "Best-Fitting" Linear Regression Models, for Men and Women Separately (All Ages) Excluding All Those on Antihypertensive Medication

<table>
<thead>
<tr>
<th>Blood pressure</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic</td>
<td>0.040 ± 0.024</td>
<td>0.046 ± 0.053</td>
</tr>
<tr>
<td>Diastolic</td>
<td>0.021 ± 0.008</td>
<td>0.061 ± 0.036</td>
</tr>
</tbody>
</table>

Substitution of the alcohol variable with a variable for total fluid intake (including alcohol) did not improve the prediction of BP. The variable for total fluid intake dropped out of more of the models as statistically insignificant than did the variable alcohol consumption. This may be due to a dilution effect. When alcohol was separated into beer, wine, and spirits consumption, the variable for beer consumption was frequently significant while that for wine consumption was not. Too few subjects drank large amounts of spirits to draw any conclusions about the effect of spirits consumption on BP.

For the logistic regression analyses, separate analyses were conducted for two different dichotomous BP variables: hypertension (SBP ≥ 160 mm Hg and/or DBP ≥ 95 mm Hg) and elevated BP (SBP ≥ 140 mm Hg and/or DBP ≥ 90 mm Hg). After excluding all participants on antihypertensive medication, 147 men (15.6%) had hypertensive BP values and 338 men (35.9%) had elevated BP values; of the women, 82 (8.1%) had hypertensive BP values and 208 (20.5%) had elevated BP values.

With regard to hypertension in men, the alcohol variable was found to be a significant main effect in both analyses (ages 30–69 and 30–59 years). The odds ratio for developing hypertension was estimated at 1.49 (95% confidence interval: 1.09, 2.04) for men drinking 80 g of alcohol per day compared to men drinking no alcohol. When elevated BP was used as the dependent variable in men, the interaction term Alcohol × SMK was significant, with a positive coefficient in the analyses of both age groups. With regard to hypertension and elevated BP in women, the variables alcohol and smoking dropped to insignificant levels during the backward-stepping in all the logistic regression analyses.

Discussion

Alcohol appeared as a significant main effect in most of the multiple linear regression analyses for men and women. However, the magnitude of the effect of alcohol did not appear to be as large as that of BMI. BMI was found to be the strongest main effect, which supports the results of many epidemiologic studies.9

In the Lipid Research Clinic Study (LRC Study), similar linear regression analyses were performed.10-12 Table 3 compares the alcohol coefficients from the LRC study with those of the MBS and those from a study by Mitchell et al.13 that contained 40% brewery workers. Neither the LRC study nor that of Mitchell et al. allowed for interactions between the variables, but both had more main effects in their models that were not dropped out. The results of the LRC study concerning the role of alcohol are fairly similar to those of the MBS, although the coefficients tended to be slightly higher in the LRC study. However, the study by Mitchell et al. showed much higher coefficients for alcohol than both the LRC and MBS studies. The findings of the study by Mitchell et al. are particularly important because it is known that brewery workers have a high beer consumption.

A discussion of the accuracy of the assessment of alcohol intake seems warranted with regard to the findings in brewery workers. A bias may result from the underreporting of alcohol consumption. Misclassification in the direction of underreporting may in fact obscure the true strength of the alcohol-BP relationship. It is also in line with this argument that the rela-
tionship of fluid intake (including alcohol intake) and hypertension turned out to be weaker than the pure alcohol-BP relationship, thus pointing again to a dilution effect. With regard to other potential confounders, it appeared that the relationship between alcohol consumption and systolic and diastolic BP in the MBS was independent of age, sex, BMI, cigarette smoking, oral contraceptive use, and years of education.

The limitations of a cross-sectional study with regard to causal inference must be kept in mind; the direction of the relationship cannot be definitively identified. Moreover, other factors that have not been considered could lead to a spurious relationship between alcohol and BP. However, although the underlying pathophysiologic mechanism that may cause the alcohol-related rise in BP is still obscure and the precise contribution of alcohol consumption to hypertension is not yet known, the results of the MBS and other studies offer some practical suggestions for high blood pressure control in the community. Limiting alcohol intake may provide a useful tool for the nonpharmacologic control of elevated BP, particularly in those persons with only mild hypertension. Cessation of alcohol ingestion, apart from leading to a reduction in BP, has also been observed to be associated with improved adherence to drug therapy for hypertension. Estimates that from 10% to as much as 30% of so-called essential hypertension may be due to excessive alcohol consumption suggest that a detailed history of alcohol consumption should be elicited from all patients with hypertension, especially those whose conditions do not respond to treatment.

References


Appendix

The hierarchical variable selection strategy involved proceeding backward in such a way that no lower order term in the model was eligible for removal from the model if it was contained as a component of a higher order product term still in the model. From the initial model, the insignificant third-order terms were first considered for removal using a standard backward-stepping algorithm. Then, second-order terms that were not contained as components of significant third-order terms still in the model were considered for removal, analogously. Finally, those main effects that were possible risk factors (BMI, alcohol, SMK) and that were not contained in any higher order terms still in the model were considered for removal in a similar fashion. Age was treated as a control variable, so it was retained as a main effect in all models. At no point was a variable allowed to reenter the model.

The procedure for monitoring the significance level during variable selection involved using an ad hoc Bonferroni-type rule within blocks of variables separated by order (i.e., main effects, second order, third order). The usual Bonferroni adjustment applied to forward-stepping algorithms uses a nominal significance level of $\alpha' = \alpha/k$, where $\alpha = 0.05$ is the desired level of significance and $k$ is the number of variables within a given block eligible for entry into the model at a given step. Such an adjustment, however, does not directly apply to backward-stepping, since a backward approach considers removal of variables from a model rather than entry to a model and uses as a test statistic the smallest partial F among those variables eligible for removal rather than the largest partial F among those variables eligible for entry. The following procedure for the backward control of the significance level was applied in the analyses reported here. This procedure supplies the same nominal significance levels when going backward as would be obtained from forward-stepping for each subset of variables within a given block. Specifically, if there are $n$ variables in a given block, then $\alpha' = \alpha/n$ being used at the last step.
Alcohol consumption as a risk factor for high blood pressure. Munich Blood Pressure Study.
V Cairns, U Keil, D Kleinbaum, A Doering and J Stieber

*Hypertension*. 1984;6:124-131
doi: 10.1161/01.HYP.6.1.124

*Hypertension* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1984 American Heart Association, Inc. All rights reserved.
Print ISSN: 0194-911X. Online ISSN: 1524-4563

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://hyper.ahajournals.org/content/6/1/124

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in *Hypertension* can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to *Hypertension* is online at:
http://hyper.ahajournals.org//subscriptions/