Relation of blood pressure to reported intake of salt, saturated fats, and alcohol in healthy middle-aged population

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SUMMARY The association of blood pressure with reported intake of salt, saturated fats, and alcohol was studied in a sample of 8479 subjects based on a cross sectional survey in a population aged 30 to 64 years. A consistent association was found between the mean arterial pressure and the intake of alcohol (p<0.001) and saturated fats (p<0.01). There was also a weak association between blood pressure and dietary salt intake, but this association was mostly explained by the correlation of salt intake with alcohol and saturated fats. The observed relationships support the hypothesis that blood pressure is influenced by diet.

A few although not all recent experimental studies in man have observed a reduction in blood pressure associated with a reduction of dietary sodium intake. There are also indications that the amount and content of dietary fats and intake of alcohol may be related to blood pressure in man. The findings, however, from various studies with regard to association of all these three nutrients with blood pressure are somewhat controversial. The purpose of the present study was to provide additional information on the association of blood pressure with reported dietary intake of salt, saturated fats, and alcohol in a middle-aged healthy human population.

Study population and methods

The study is based on a random 6·5% sample of the population of two counties (North Karelia and Kuopio) from Eastern Finland, examined in 1977. The sampling frame consisted of people born between 1913 and 1947 (aged then between 30 and 64).

The original sample included 12 475 individuals, of whom 89·6% (11 174) participated in the survey. Approximately 6% of the participants responded to the questionnaire only. The data on physical measurements were obtained for 10 502 individuals. There were 1685 who had a history of diabetes, bronchial asthma, cardiac or cerebrovascular disease, or drug treatment for hypertension or hyperlipidaemias who were excluded from the study population. For 338, data on some of the study variables were missing, and these people were also excluded. The present analysis was carried out in the remaining 8479 (4199 men and 4280 women) with complete data and free of severe diseases.

The survey included a self-administered questionnaire, personal interview, and physical measurements. Casual blood pressure was measured from the right arm in a sitting position with a standard mercury sphygmomanometer by trained nurses who had been trained and standardised for the purpose of the survey. The fifth phase of the Korotkoff sounds was recorded as diastolic blood pressure. A venous blood specimen was taken after a median fast of nine hours. Serum total cholesterol was determined from frozen sera in random order in one laboratory using the Technicon Auto Analyzer Methodology (N-77, 1969).

Dietary intake of salt was estimated with three questions in the questionnaire: “How much do you use salt and eat salty foods compared with other people?” scored 1 to 5, “Do you add salt to your food before tasting?” scored 1 to 3, and “Do you consider the salt content of meals outside home compared with home made food (1) less salty, (2) same, or (3) more salty.” An additive index was constructed of these three items recoded 0 to 2. The intake of saturated fats was measured with a set of questions. The constructions of the index has been described earlier. Alcohol consumption was measured by two questions, one concerning the frequency of intake of strong alcoholic beverages and the other.
consumption of beer. Family history of hypertension was defined positive if either father, mother, or any sibling had hypertension detected under the age of 60 years. Corpulence was described by body-mass index (BMI): weight (kg) over the square of height (m). The mean arterial pressure (MAP), the sum of the diastolic blood pressure and one-third of the pulse pressure, was used as the measure of blood pressure.

The average MAP was computed in strata of reported salt intake with simultaneous break-down first by sex and intake of salt and saturated fats and second by sex and intake of salt, saturated fats, and alcohol. A covariance correction for four variables most strongly correlated with MAP was used. The variation of means was tested for significance by analysis of covariance (ANCOVA).

The partial association of mean arterial pressure with the intake of salt as well as saturated and polyunsaturated fats and serum cholesterol was estimated with the multivariate least-squares regression technique. Age, body-mass index, and heart rate were entered as additional independent variables for the purpose of adjustment. The missing observations in some of the independent variables were replaced by grand means.

**Table 1** Correlations between mean arterial pressure and selected variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>MAP</th>
<th>BMI</th>
<th>AGE</th>
<th>HR</th>
<th>CHOL</th>
<th>FAM</th>
<th>ALC</th>
<th>FAT</th>
<th>MEA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean arterial pressure</td>
<td>(MAP)</td>
<td>-0.40</td>
<td>-0.35</td>
<td>-0.27</td>
<td>0.06</td>
<td>-0.05</td>
<td>-0.27</td>
<td>0.02</td>
<td>0.08</td>
</tr>
<tr>
<td>Body-mass index</td>
<td>(BMI)</td>
<td>-0.04</td>
<td>0.13</td>
<td>0.11</td>
<td>0.01</td>
<td>-0.03</td>
<td>0.03</td>
<td>-0.11</td>
<td>0.07</td>
</tr>
<tr>
<td>Age</td>
<td>(AGE)</td>
<td>-0.05</td>
<td>0.37</td>
<td>0.12</td>
<td>0.06</td>
<td>-0.11</td>
<td>0.03</td>
<td>-0.03</td>
<td>0.11</td>
</tr>
<tr>
<td>Heart rate</td>
<td>(HR)</td>
<td>-0.07</td>
<td>-0.01</td>
<td>-0.01</td>
<td>0.02</td>
<td>-0.04</td>
<td>0.02</td>
<td>-0.05</td>
<td>0.05</td>
</tr>
<tr>
<td>Serum cholesterol</td>
<td>(CHOL)</td>
<td>-0.27</td>
<td>-0.05</td>
<td>-0.01</td>
<td>0.03</td>
<td>-0.06</td>
<td>0.04</td>
<td>-0.02</td>
<td>0.06</td>
</tr>
<tr>
<td>Family history of hypertension</td>
<td>(FAM)</td>
<td>-0.11</td>
<td>0.00</td>
<td>0.00</td>
<td>0.07</td>
<td>0.07</td>
<td>0.03</td>
<td>-0.07</td>
<td>-0.13</td>
</tr>
<tr>
<td>Intake of alcohol</td>
<td>(ALC)</td>
<td>0.04</td>
<td>0.01</td>
<td>0.03</td>
<td>0.09</td>
<td>0.06</td>
<td>0.03</td>
<td>0.03</td>
<td>0.07</td>
</tr>
<tr>
<td>Intake of saturated fats</td>
<td>(FAT)</td>
<td>0.17</td>
<td>0.07</td>
<td>0.08</td>
<td>0.08</td>
<td>0.04</td>
<td>0.08</td>
<td>0.04</td>
<td>0.13</td>
</tr>
<tr>
<td>No of BP measurements</td>
<td>(MEA)</td>
<td>0.03</td>
<td>0.03</td>
<td>0.07</td>
<td>0.06</td>
<td>0.03</td>
<td>0.07</td>
<td>0.04</td>
<td>0.00</td>
</tr>
<tr>
<td>Intake of salt</td>
<td>(SALT)</td>
<td>0.05</td>
<td>0.06</td>
<td>-0.04</td>
<td>0.00</td>
<td>-0.02</td>
<td>0.04</td>
<td>-0.05</td>
<td>-0.03</td>
</tr>
</tbody>
</table>

**Table 2** Mean adjusted arterial pressure (mm Hg) according to sex and intake of alcohol, saturated fats, and salt

<table>
<thead>
<tr>
<th>Sex</th>
<th>Low alcohol intake</th>
<th>High alcohol intake</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Low fat intake</td>
<td>High fat intake</td>
<td>Low fat intake</td>
<td>High fat intake</td>
<td>Low fat intake</td>
<td>High fat intake</td>
<td>Low fat intake</td>
<td>High fat intake</td>
<td>Low fat intake</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Low fat intake</td>
<td>High fat intake</td>
<td>Low fat intake</td>
<td>High fat intake</td>
<td>Low fat intake</td>
<td>High fat intake</td>
<td>Low fat intake</td>
</tr>
<tr>
<td>Low salt intake</td>
<td>106-5</td>
<td>106-2</td>
<td>108-8</td>
<td>109-5</td>
<td>101-6</td>
<td>103-3</td>
<td>102-7</td>
<td>97-3</td>
<td>104-3</td>
</tr>
<tr>
<td></td>
<td>(1823)</td>
<td>(1421)</td>
<td>(187)</td>
<td>(147)</td>
<td>(2892)</td>
<td>(821)</td>
<td>(39)</td>
<td>(16)</td>
<td>(7346)</td>
</tr>
<tr>
<td>High salt intake</td>
<td>106-9</td>
<td>106-9</td>
<td>109-1</td>
<td>111-3</td>
<td>101-3</td>
<td>103-5</td>
<td>103-0</td>
<td>122-6</td>
<td>104-6</td>
</tr>
<tr>
<td></td>
<td>(235)</td>
<td>(249)</td>
<td>(62)</td>
<td>(75)</td>
<td>(362)</td>
<td>(134)</td>
<td>(14)</td>
<td>(2)</td>
<td>(1133)</td>
</tr>
<tr>
<td>Total</td>
<td>106-6</td>
<td>106-4</td>
<td>108-9</td>
<td>110-3</td>
<td>101-5</td>
<td>103-4</td>
<td>102-8</td>
<td>100-1</td>
<td>104-3</td>
</tr>
<tr>
<td></td>
<td>(2058)</td>
<td>(1670)</td>
<td>(249)</td>
<td>(222)</td>
<td>(3254)</td>
<td>(955)</td>
<td>(33)</td>
<td>(18)</td>
<td>(8479)</td>
</tr>
</tbody>
</table>

Four-way analysis of covariance: Main effects: sex p<0.001, alcohol intake p<0.001, fat intake p = 0.009, salt intake not significant.
Interactions: sex-fat intake p = 0.001, sex-alcohol intake p = 0.11, sex-alcohol intake−fat intake−salt intake p = 0.011.
Covariates: BMI p<0.001, age p<0.001, heart rate p<0.001, family history of hypertension p<0.001.

**Results**

The reported intake of salt correlated weakly with the intake of alcohol (r = 0.15) and saturated fats (r = 0.13) whereas the intake of saturated fats and alcohol were almost independent of each other (table 1). The intake of saturated fats correlated moderately with age. The mean arterial pressure had notable crude positive correlations with body-mass intake, age serum cholesterol, heart rate, and saturated fat intake.

When adjusting for body-mass intake, age, heart rate, and family history of hypertension, the average MAP rose almost linearly over the categories of reported salt intake both in men and in women (fig 1). The linear component of the relationship was highly significant in one-way analysis of variance with no covariance correction (p<0.001).

Both salt intake (p = 0.044) and saturated fat intake (p = 0.015) were positively associated with MAP in the simultaneous break-down by these factors and sex in a three-way analysis of covariance (fig 2). In addition, the intake of saturated fats was more strongly associated with MAP in women than in men (p<0.001 for interaction). In the four-way analysis of covariance (table 2) allowing in addition...
with MAP more strongly in men than in women (for interaction). In both sexes combined the adjusted MAP was 8% (8.0 mm Hg) higher in those who used a lot of salt, saturated fats, and alcohol (111.4 mm Hg) compared with those who did not (103.4 mm Hg).

The linear regression model including 10 independent variables explained 31% of the variation of MAP. MAP had a significant positive partial regression on both alcohol intake and saturated fat intake. These variables jointly accounted, however, for only 3% of the explained variation of MAP.

Table 3 Partial regression of mean arterial pressure on selected variables

<table>
<thead>
<tr>
<th>Increase in $R^2$</th>
<th>t value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body-mass index</td>
<td>0.077</td>
</tr>
<tr>
<td>Age</td>
<td>0.045</td>
</tr>
<tr>
<td>Heart rate</td>
<td>0.043</td>
</tr>
<tr>
<td>Sex (0 if male; 1 if female)</td>
<td>0.020</td>
</tr>
<tr>
<td>Family history of hypertension</td>
<td>0.006</td>
</tr>
<tr>
<td>Serum cholesterol</td>
<td>0.006</td>
</tr>
<tr>
<td>Intake of alcohol (g/wk)</td>
<td>0.002</td>
</tr>
<tr>
<td>Intake of saturated fats</td>
<td>0.001</td>
</tr>
<tr>
<td>No of BP measurements</td>
<td>0.001</td>
</tr>
<tr>
<td>Intake of salt</td>
<td>0.000</td>
</tr>
<tr>
<td>Entire model</td>
<td>0.308</td>
</tr>
</tbody>
</table>

1F-ratio for entire model.

Discussion

In our data based on a large cross-sectional population sample blood pressure associated consistently with the reported intake of alcohol and saturated fats. There was also a weak association between blood pressure and reported dietary salt intake, but this association was in these data explained by the correlation of salt intake with alcohol and saturated fats. Since increased intakes of salt, saturated fats, and alcohol tended to cluster in the same people, it was not possible to separate any independent effect of salt intake on blood pressure. When analysed separately without taking fats and alcohol into account, those reporting high intake of salt had slightly higher blood pressures than those using less salt.

For several methodological reasons the power of the cross sectional design in detecting associations between blood pressure and various components of diet is likely to be low. Firstly, blood pressure is a parameter with a notable random and biological variation within individuals over time. This tends to increase the within-group variance of blood pressure and subsequently increase the probability of type I error. The stability of the blood pressure values can be improved by stabilising the conditions in which the measurements are done and increasing the number of

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Fig 1 Adjusted mean arterial pressure according to dietary intake of salt in men and women. Two-way ANCOVA main effects: sex $p<0.001$, salt intake $p=0.031$; interaction: not significant; covariates: BMI $p<0.001$, age $p<0.001$, heart rate $p<0.001$, family history of hypertension $p<0.001$.

Fig 2 Adjusted mean arterial pressure according to sex, intake of saturated fats, and intake of salt. Three-way ANCOVA: main effects: sex $p<0.001$, fat intake $p=0.015$, salt intake $p=0.044$; interactions: sex-fat intake $p<0.001$, others not significant; covariates: BMI $p<0.001$, age $p<0.001$, heart rate $p<0.001$, family history of hypertension $p<0.001$.  

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Jukka T Salonen, Jaakko Tuomilehto, and Antti Tanskanen
Relation of blood pressure to reported intake of salt, saturated fats, and alcohol

measurements for each subject. Nevertheless, even one measurement of blood pressure has been shown to predict the occurrence of later coronary heart disease as well as an estimate based on several measurements.21 Some random variability is included also in the estimates of intake of nutrients based on questionnaires. The assessment of dietary salt intake by questionnaires has resulted in varying success. Hill et al.22 did not succeed in developing a questionnaire that would associate well with urinary sodium excretion. In a recent study, however, a sizeable correlation was shown between the 24-hour urinary sodium excretion and certain salting habits assessed by questionnaire with the same questions as in the present study.24

Secondly, a certain latency period seems likely in the hypothetical causal effect of sodium intake and probably also of fat content of the diet and alcohol consumption on blood pressure, since hypertension in general develops gradually over time.24 Also, only a part of the people are probably genetically “salt-sensitive.” According to this theory, among the people who consume excess salt, only those who are salt sensitive develop hypertension and those who are “salt-resistant” remain normotensive.25 26 More generally, since blood pressure is partly determined by environmental factors—for instance, diet—and partly by genetic factors,27 associations between blood pressure and dietary components are expectedly more easily detectable between populations than within populations. Finally, excluding the persons with antihypertensive or hypolipidemic medication and chronic cardiovascular disease may weaken the observed associations between blood pressure and dietary components by reducing the inter-individual variation in blood pressure and by excluding those who are most susceptible for raised blood pressure. Those who are treated or who have already developed a disease might also be those who are most sensitive to environmental exposures. For these reasons the actual associations may be stronger than those observed here. The restriction of the sample was, however, made to increase the homogeneity of the subjects with regard to other potential confounding factors, even though some of the study’s statistical power to detect associations was lost.

Despite several shortcomings, the assessment of salt intake of an individual by reported salting habits has an advantage compared with urinary sodium excretion measurements. It is unlikely that cross sectional urinary measurements can give the correct information of the salt intake during the preceding years of life,28 whereas people’s habits are probably more stable.

In several epidemiological studies from widely different parts of the world an association between excessive salt intake and high blood pressure or hypertension has been observed29-36 whereas in other surveys no such relationship was found.37-42

The results concerning the relationship between blood pressure and alcohol consumption are also conflicting. A positive association has been observed in several studies.11-16 These results, however, concern mostly individuals designated as chronic alcoholics or heavy drinkers.43-46

Using the questionnaire data, the absolute amount of alcohol remains unknown and most probably too low.46 In the study, however, hypertension was found to be associated with raised serum-glutamyl transpeptidase levels.47

Despite the limitations of the cross sectional design, we found a consistent association between blood pressure and the intake of saturated fats and alcohol, even when allowing for several potential confounding factors. Also salt intake tended to associate with blood pressure. Given the low power of design, the observed associations, though rather weak, support the hypothesis that blood pressure is influenced by the diet. A large enough, multifactorial clinical trial is needed to test the hypothesis of the causal link between dietary salt intake, fat composition, and alcohol consumption and blood pressure.

References

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