The association between blood pressure, age, and dietary sodium and potassium: a population study

KAY-TEE KHAW, M.SC., M.R.C.P., AND ELIZABETH BARRETT-CONNOR, M.D.

ABSTRACT We examined the relationship between blood pressure and dietary sodium and potassium intake estimated from 24 hr diet recall in a population of 584 men and 718 women 30 to 79 years old in Southern California. In men, but not women, age-adjusted systolic and diastolic blood pressure correlated significantly with dietary sodium intake. In both men and women, age-adjusted diastolic blood pressure significantly inversely correlated with dietary potassium intake. Age-adjusted systolic and diastolic blood pressure correlated significantly with the dietary sodium/potassium ratio in each sex; correlations were better for the ratio than for either sodium or potassium alone. The relationship was apparent over the whole range of blood pressure and dietary intake. A marked age gradient was apparent in men, the regression slope for blood pressure vs sodium/potassium ratio increasing with increasing age, suggesting increasing sensitivity to dietary sodium/potassium ratio with age. Adjusting for intake of other dietary variables, including calories, protein, carbohydrate, saturated fat, alcohol, calcium, and fiber, did not alter the relationships; adjusting for body mass index reduced the strength of the association in women but not in men. These results support the hypothesis that dietary sodium and potassium are related to blood pressure within a population.


THE DIETARY sodium-hypertension hypothesis continues to generate controversy. Abundant clinical, experimental, and epidemiologic evidence suggests that high sodium intake is causally related to high blood pressure, at least under some circumstances.1, 2 In man, correlations between mean blood pressures and/or prevalence of hypertension and mean sodium intake have been demonstrated between populations3, 3-5 and several clinical trials have reported lowering of blood pressure with reduction of sodium intake.6-11 However, the relevance of dietary sodium intake to high blood pressure in human populations is still hotly debated.12-20 Recommendations to reduce dietary sodium in the general population have been criticized on several grounds: only a small proportion of the population may be salt sensitive, the effect of moderate sodium reduction on blood pressure is absent or trivial, and little is known of long-term effects.11, 12, 17-20

One focus of argument is the lack of consistent relationships between blood pressure and dietary sodium intake within a population. As Blackburn and Jacobs have stated: “Traditionally,. . . science gives more weight and greater inferential credence to individual than to population correlations.”21 Most studies have used urinary sodium excretion as a surrogate for dietary sodium intake.22 Although several23-33 have found correlations between blood pressure and urinary sodium, others report no such significant associations within a population.34-40 Very few studies have used sodium intake directly estimated from dietary data: none of three population studies41-47 found significant correlations of blood pressure with dietary sodium alone (although two reanalyses of the National Health and Nutrition Examination Survey of the National Centre for Health Statistics [NHANES I] data have reported a significant positive relationship44, 45). The failure to show a consistent sodium–blood pressure association within populations is often cited as evidence that sodium intake is not related to blood pressure in any clinically important way within the general population.36

We report here a positive relationship between the
dietary sodium-potassium ratio and blood pressure within an older geographically defined population, and examine the effects of age and other covariates on this relationship.

Methods

Eighty-two percent of all adult residents of a geographically defined upper middle class Caucasian community in Rancho Bernardo, CA, participated in a survey of heart disease risk factors (visit 1) between 1972 and 1974. A 30% subsample of this cohort (15% random sample plus all subjects identified as hyperlipidemic at this first visit) participated in a second, more extensive evaluation (visit 2). At visit 2, demographic, behavioral, and medical history were obtained by a standard interviewer-administered questionnaire, which included questions on use of medication and personal history of hypertension, heart disease, or stroke.

Height and weight were measured in light clothing without shoes: obesity was estimated as body mass index (weight in kg/height in m$^2$). Blood pressure was measured by trained observers using a standard mercury sphygmomanometer after the subject had been seated resting at least 5 min; the mean of two readings was used for analysis.

A 24 hr dietary recall was obtained by a certified Lipid Research Clinic dietician. This included all nutrient and alcohol intake, with quantities assessed with the use of containers and food models. When possible, the brand names of commercial manufactured food products and recipes for home cooked foods were obtained. No information on added table salt, salt substitutes, or low-salt diets per se was obtained. In 1985 the raw 24 hr dietary recall data were coded for nutrient intake by the Nutrition Coordinating Center, University of Minnesota, using their 1983 computerized data base. This version estimates sodium content of food intake by taking into consideration not only sodium naturally occurring in food sources and sodium from commercial processed foods, but also includes an estimate of average sodium added during cooking with standard American recipes. Total 24 hr intake of sodium and potassium from food sources was obtained and converted to SI units for comparability with other studies. The 24 hr dietary sodium/potassium ratio was calculated as sodium (in mmol)/potassium (in mmol).

Partial correlation coefficients of blood pressure with 24 hr intake of sodium, potassium, and sodium/potassium ratio were calculated separately for men and women with the use of the Statistical Package for Social Sciences, adjusting for age, obesity, and dietary variables including calories, protein, carbohydrate, dietary fiber, calcium, saturated fat, and alcohol. We also examined correlation coefficients using sodium, potassium, and sodium/potassium ratio as a percentage of calories. Mean sodium/potassium ratio was calculated for clinically determined preassigned 20 and 10 mm Hg increments in systolic and diastolic blood pressure, respectively, adjusting for age with the use of analysis of variance. Regression coefficients for blood pressure with the sodium/potassium ratio were calculated for men and women, before and after adjusting for age and dietary variables. To examine the effect of age on this relationship, we calculated coefficients in three sex-specific age groups. We also examined regression slopes after stratifying for blood pressure and body mass index using preassigned clinically defined cutpoints, by estrogen use in women, and after stratifying for calcium intake.

Results

We report cross-sectional visit 2 results on the 584 men and 718 women who were 30 to 79 years old. Table 1 shows mean blood pressure, body mass index, 24 hr dietary sodium, potassium, sodium/potassium ratio, and caloric intake by sex and age group. In both sexes, mean systolic blood pressures increased with age; diastolic blood pressures showed less consistent changes with age. Body mass index changed little with age, although total caloric intake decreased in both sexes. In men, mean sodium and potassium intake decreased

| TABLE 1 |
| Mean blood pressure, body mass index, and dietary variables by age and sex for Rancho Bernardo men and women 30 to 79 years old |

<table>
<thead>
<tr>
<th></th>
<th>30–49 years</th>
<th>50–64 years</th>
<th>65–79 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men</td>
<td>n = 158</td>
<td>n = 173</td>
<td>n = 253</td>
</tr>
<tr>
<td>Systolic BP (mm Hg)</td>
<td>122.4 (11.9)</td>
<td>134.7 (16.0)</td>
<td>145.2 (20.9)</td>
</tr>
<tr>
<td>Diastolic BP (mm Hg)</td>
<td>82.2 (9.2)</td>
<td>84.1 (9.3)</td>
<td>83.7 (10.2)</td>
</tr>
<tr>
<td>Body mass index (kg/m$^2$)</td>
<td>26.4 (3.0)</td>
<td>26.6 (2.9)</td>
<td>25.6 (2.8)</td>
</tr>
<tr>
<td>Sodium (mmol)</td>
<td>171 (66)</td>
<td>147 (81)</td>
<td>132 (50)</td>
</tr>
<tr>
<td>Potassium (mmol)</td>
<td>77 (31)</td>
<td>71 (23)</td>
<td>68 (21)</td>
</tr>
<tr>
<td>Sodium/potassium ratio</td>
<td>2.4 (0.9)</td>
<td>2.2 (1.3)</td>
<td>2.1 (0.8)</td>
</tr>
<tr>
<td>Calories</td>
<td>2437 (901)</td>
<td>2056 (543)</td>
<td>2019 (572)</td>
</tr>
<tr>
<td>Women</td>
<td>n = 170</td>
<td>n = 239</td>
<td>n = 309</td>
</tr>
<tr>
<td>Systolic BP (mm Hg)</td>
<td>115.7 (15.8)</td>
<td>132.0 (19.2)</td>
<td>142.1 (22.2)</td>
</tr>
<tr>
<td>Diastolic BP (mm Hg)</td>
<td>77.1 (9.7)</td>
<td>81.7 (9.9)</td>
<td>81.8 (10.4)</td>
</tr>
<tr>
<td>Body mass index (kg/m$^2$)</td>
<td>23.3 (3.5)</td>
<td>24.3 (3.9)</td>
<td>24.5 (3.5)</td>
</tr>
<tr>
<td>Sodium (mmol)</td>
<td>112 (50)</td>
<td>114 (42)</td>
<td>101 (48)</td>
</tr>
<tr>
<td>Potassium (mmol)</td>
<td>57 (23)</td>
<td>63 (19)</td>
<td>57 (19)</td>
</tr>
<tr>
<td>Sodium/potassium ratio</td>
<td>2.1 (0.8)</td>
<td>1.9 (0.8)</td>
<td>1.9 (0.9)</td>
</tr>
<tr>
<td>Calories</td>
<td>1666 (703)</td>
<td>1656 (483)</td>
<td>1521 (491)</td>
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Values are mean (± SD).

*Estimated from 24 hr diet recall.
with age, as did the sodium-potassium ratio; trends were less apparent in women. For the whole group, mean sodium and potassium intake from food sources was 125 (range 7 to 959) and 64 (range 8 to 199) mmol/24 hr, respectively; mean sodium/potassium ratio was 2.0 (range 0.3 to 11.5).

Table 2 shows age-adjusted sex-specific partial correlation coefficients of dietary sodium, potassium, and sodium/potassium ratio with blood pressure. In men, age-adjusted systolic and diastolic blood pressure correlated significantly positively with sodium alone and sodium/potassium ratio and negatively with potassium intake. In women, age-adjusted systolic and diastolic blood pressure correlated significantly negatively with potassium intake and positively with the sodium/potassium ratio; there was no significant relationship with sodium intake alone. For both men and women, the correlations were stronger for the sodium/potassium ratio than for either sodium or potassium alone.

Adjusting for body mass index reduced these correlations more in women than in men. The relationships were similar when the sodium/calorie, potassium/calorie and sodium/potassium/calorie ratios were used in place of the absolute quantities (not shown). Excluding

| TABLE 2 | Age-adjusted partial correlation coefficients of blood pressure with dietary sodium, potassium, and sodium/potassium ratio for Rancho Bernardo men and women 30 to 79 years old |
|-----------------|-----------------|-----------------|-----------------|
| Systolic blood pressure | n | Sodium | Potassium | Sodium/potassium ratio |
| Men | | | | |
| All (age adjusted) | 584 | +.09E | -.06D | +.14F |
| Age and obesity adjusted | +.09E | -.06D | +.13F |
| Adjusted for age and dietary variablesA | +.12E | -.04 | +.13F |
| Excluding hypertensivesB | +.05 | -.05D | +.09E |
| Excluding those with personal history of cardiovascular diseaseC | 511 | +.08E | -.09E | +.16F |
| Women | | | | |
| All (age adjusted) | 718 | +.03 | -.06D | +.10E |
| Age and obesity adjusted | +.01 | -.05 | +.07E |
| Adjusted for age and dietary variablesA | +.06D | -.06E | +.10E |
| Excluding hypertensivesB | -.01 | -.08E | +.07E |
| Excluding those with personal history of cardiovascular diseaseC | 666 | +.01 | -.06D | +.09E |
| Those not taking hormones | 469 | +.01 | -.07E | +.08E |
| Those taking hormones | 249 | +.08E | -.02E | +.12E |
| Diastolic blood pressure | | | | |
| Men | | | | |
| All (age adjusted) | +.11F | -.09E | +.18F |
| Age and obesity adjusted | +.11F | -.09E | +.17F |
| Adjusted for age and dietary variablesA | +.16F | -.06D | +.17F |
| Excluding hypertensivesB | +.01 | -.11F | +.10E |
| Excluding those with personal history of cardiovascular diseaseC | +.10F | -.12F | +.18F |
| Women | | | | |
| All (age adjusted) | +.02 | -.07E | +.08E |
| Age and obesity adjusted | +.00 | -.06D | +.05 |
| Adjusted for age and dietary variablesA | +.06D | -.04 | +.08E |
| Excluding hypertensivesB | -.02 | -.08E | +.05 |
| Excluding those with personal history of cardiovascular diseaseC | -.06F | +.07D |
| Those not taking hormones | +.01 | -.10F | +.09E |
| Those taking hormones | +.04 | -.02 | +.08E |

A Dietary variables are calories, protein, fiber, calcium, saturated fat, and alcohol.
B History of hypertension and/or taking antihypertensive medication.
C Personal History of hypertension and/or taking antihypertensive medication.
Dp > .05 < .10; Gp < .05; Hp < .01.
all subjects with a personal history of hypertension or those using antihypertensive medication reduced the correlations with sodium level, but tended to increase them for potassium, overall reducing the blood pressure correlations with sodium/potassium ratios only slightly. Although both systolic and diastolic pressure correlated with the sodium/potassium ratio in all women, when women were stratified by hormone use (mainly unopposed postmenopausal estrogen), a positive correlation of systolic blood pressure with sodium was apparent only in those taking hormones while a negative correlation of potassium with diastolic pressure was apparent only in those not taking hormones.

After adjusting for age, neither systolic nor diastolic blood pressure correlated significantly with 24 hr protein, fat, carbohydrate, or total caloric intake in men or women. Adjusting for dietary calcium, alcohol, fiber, saturated fat, and calories did not alter the relationship of blood pressure with sodium and potassium intake.

Figure 1 shows mean age-adjusted sodium/potassium ratio by increments in blood pressure. The mean ratio of sodium/potassium intake increased with increasing levels of systolic blood pressure even at levels below 140 mm Hg in both men and women.

Table 3 shows age-adjusted regression slopes for systolic and diastolic blood pressure on 24 hr sodium/potassium ratio. In men and women, respectively, every unit increase in sodium/potassium ratio was associated with a 2.4 and 2.2 mm Hg increase in systolic blood pressure and a 1.7 and 1.0 mm Hg increase in diastolic blood pressure. Regression slopes did not differ significantly between the random sample and hyperlipidemic groups. Regression slopes were similar after including calories, calcium, alcohol, carbohydrate, calcium dietary fiber, protein, and fat simultaneously in the multiple regression. In men there was a marked age gradient for the relationship of both systolic and diastolic blood pressure with sodium/potassium ratio: for men under 50 years, the regression slope for the relationships of both systolic and diastolic blood

![Graph](image-url)

**FIGURE 1.** Age-adjusted mean sodium (mmol)/potassium (mmol) ratio by systolic and diastolic blood pressure category. Rancho Bernardo men and women 30 to 79 years old.

### Table 3

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>SBP &lt;160 mm Hg and DBP &lt;90 mm Hg</th>
<th>DBP ≥90 mm Hg or SBP ≥160 mm Hg</th>
<th>BMI &lt;27 kg/m²</th>
<th>≥27 kg/m²</th>
<th>Calcium</th>
<th>Hypertension users (diet adjusted)</th>
<th>Regression slope (SE) for blood pressure (mm Hg) vs sodium/potassium ratio</th>
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SBP = systolic blood pressure; DBP = diastolic blood pressure; BMI = body mass index.

Adjusted for age, calories, protein, carbohydrate, saturated fat, alcohol, fiber, and calcium.

\( ^{a} p > .05 < .10; ^{b} p < .05; ^{c} p < .01. \)
pressure with increasing sodium/potassium ratio was not significant; the regression slope was greater in the 50 to 64 age group and steepest in the over 65 age group, with a 3.7 increase in systolic and 3.2 increase in diastolic pressure per unit increase in sodium/potassium ratio. No consistent trends with age were apparent for women.

When the blood pressure–sodium/potassium regression was examined by blood pressure category, the slopes were steeper for those with a systolic pressure over 160 mm Hg or diastolic pressure over 90 mm Hg in both men and women. Men with a body mass index greater than 27 kg/m² (i.e., at least 20% greater than Metropolitan ideal weight) showed a steeper regression slope for the relationship of blood pressure with sodium/potassium ratio than men with a body mass index less than 27 kg/m². However, the reverse was seen in women, in whom the regression slope for blood pressure vs sodium/potassium ratio was steeper in those with a body mass index less than 27 kg/m². Stratifying by calcium intake also revealed sex differences: men with a calcium intake less than 10 mmol a day had a steeper regression slope for the relationship of blood pressure with sodium/potassium. The converse was so in women.

Discussion

The difficulty of characterizing individual dietary sodium intake is particularly pronounced given its ubiquitous discretionary use as well as large intraindividual variability. Most population studies have used urinary sodium, usually 24 hr collections, to determine dietary sodium intake. Only some of these have found a significant positive relationship between urinary sodium and blood pressure. As reviewed by Watt and Foy, the absence of a correlation between urinary sodium and blood pressure may be due to a lack of statistical power in most studies, and hence does not negate a real relationship.

Very few population studies have used quantitative dietary sodium data. Of three identified, two estimated sodium by use of food frequency histories, and only the NHANES I survey used the more quantitative 24 hr diet recall method. The Connecticut study, which was a probability sample of 3566 subjects without known hypertension, compared blood pressures between high and low salt use groups with the use of semiquantitative food frequency estimates. Mean blood pressures were significantly higher only for diastolic blood pressure in women. A Netherlands study of 2391 men and women reported no significant association of blood pressure with dietary sodium estimated from a 1 week food frequency recall. The NHANES I is the only large population study we know of that has reported quantitative data on dietary sodium intake calculated from food sources using specific 24 hr diet recalls and blood pressure and these data have had conflicting interpretations. One widely publicized analysis reported that hypertensive subjects had significantly lower sodium intake; however, an alternate analysis, adjusting for age, found a positive relationship between sodium intake and blood pressure. In three other analyses of the same data set, one reported an association between dietary sodium/potassium ratio only in men 25 to 54 years old; another reported that sodium/potassium ratio correlated with blood pressure only at low levels of calcium intake, and yet another reported no significant association between sodium/potassium ratio–blood pressure, but did note a significant sodium/calcium ratio–blood pressure correlation.

There are several reasons why we may have been more successful than others in demonstrating a dietary sodium–blood pressure relationship. In the Rancho Bernardo population, a geographically defined, socioeconomically homogenous Caucasian cohort, confounding by socioeconomic, ethnic, and geographic factors was less than that likely in NHANES I, which was a weighted probability sample of the U.S. population. Furthermore, in Rancho Bernado, quantitative measures of exact foods eaten were obtained from the 24 hr diet recalls with the use of representative food models and by highly trained dieticians, in contrast to NHANES I, and rather than food frequency estimates used in the two negative studies. Also, the recent 1983 data base included sodium added in cooking, which may have increased the accuracy of sodium estimates. Although our finding of a relationship between blood pressure and dietary sodium was estimated from a single 24 hr diet recall, with no record of discretionary sodium intake, in populations such as this in which much processed food, which is high in sodium, is eaten, over 80% of the sodium intake is likely to be nondiscretionary. It is notable that mean sodium intake estimated only from food sources was already well over 100 mmol/day. It is also possible that in this retirement population intraindividual daily variation in diet may have been less than in other, younger populations. Finally, the older age of the cohort may have played a role, since the strongest association of sodium intake and blood pressure was seen in men over 65 years old.

The age gradient may also help explain discrepancies reported from different studies. For example, McCar-
ron’s report\textsuperscript{41} that sodium intake was significantly lower in hypertensive subjects in the NHANES I study would be expected if older subjects were more likely to have hypertension and if older subjects also consumed less sodium, as was in the case in NHANES I and in the Rancho Bernardo cohort.

Biological data support the hypothesis that increasing age is associated with increasing susceptibility to the effects of dietary sodium and potassium on blood pressure: the ability of the kidney to excrete a salt load decreases with age,\textsuperscript{53} so it is plausible that older people may respond to high salt load with a greater increase in blood pressure.\textsuperscript{54} Observational data also supports with this hypothesis: in the longitudinal Kenyan migration study,\textsuperscript{55} after 18 months of follow-up, the regression for blood pressure vs age was significantly positive for the urban group and still flat for the rural control subjects; older subjects had a greater rise in blood pressure than the young for the same duration of urbanization. A recent review of trials of sodium restriction also suggested that blood pressure reduction with sodium restriction is greater with increasing age.\textsuperscript{11} It thus seems likely that the aging process may be associated with increased sensitivity to environmental agents such as sodium that may raise blood pressure.

The effects of sodium and potassium on blood pressure are probably biologically interdependent, as postulated by Meneely and Battarbee.\textsuperscript{56} Three population-based studies, one of them in this cohort, have reported a negative association between dietary potassium intake and blood pressure.\textsuperscript{57–59} In the present analysis, although both sodium and potassium showed independent relationships to blood pressure, the correlations with the dietary sodium/potassium ratio were in general stronger than those with either sodium or potassium alone. A recent analysis\textsuperscript{45} of the NHANES I data also indicates that a relationship between blood pressure and sodium/potassium ratios is more readily demonstrable within this population than a relationship with either sodium or potassium alone. These results agree with reports of correlations between blood pressure and urinary sodium/potassium ratios estimated from 24 hr, overnight, or even casual samples.\textsuperscript{26–28, 60–63}

Although overall blood pressure was related to dietary sodium/potassium ratio in both sexes, sex differences were apparent. Blood pressure correlated with sodium intake alone only in men. However, when women were stratified by exogenous estrogen use, systolic blood pressure significantly correlated with dietary sodium intake only in hormone-using women. Conversely, although blood pressure negatively correlated with dietary potassium overall in both men and women, when stratified by hormone use, this negative relationship was only present in women not taking exogenous sex hormones. Other studies have noted sex differences in these relationships. For example, the Whitehall study,\textsuperscript{64} based on a younger cohort, reported that systolic and diastolic blood pressure inversely correlated with urinary potassium in men but not women. Hormonal mechanisms may affect sensitivity to dietary sodium or potassium. In a Belgian study,\textsuperscript{65} in which blood pressure overall in young women was not related to sodium excretion, a positive association was noted in women taking an oral contraceptive. The absence of a consistent age gradient in the blood pressure–sodium and potassium regression for women may reflect hormonal factors that affect this relationship.

The relationship of body mass index to blood pressure and sodium and potassium also differed between the sexes. Body mass index was more strongly related to blood pressure in women than men. Adjusting for body mass index reduced the associations of blood pressure with sodium and potassium more in women than in men. This finding is consistent with those of Watson et al.,\textsuperscript{26} which suggest that body mass is a more important determinant of blood pressure in women than men. Several studies have reported interactions between dietary salt and obesity affecting blood pressure interventions.\textsuperscript{10, 66, 67} In the Rancho Bernardo cohort of men, the regression of blood pressure on sodium/potassium ratio was greater for men with a body mass index over 27; the converse was so for women. The sex differences may be due to the imprecision of body mass estimates, which often reflect different fat and muscle mass distribution in men vs women.

Other dietary factors, including low intakes of calcium fiber, and unsaturated fat, and high intakes of alcohol, protein, and saturated fat have been associated with higher blood pressure levels.\textsuperscript{41, 68–72} Both dietary sodium and potassium correlated with these other dietary variables and total caloric intake; however, adjusting for all these factors did not significantly change the correlation of blood pressure with dietary sodium/potassium ratio in either sex. When all these variables were simultaneously entered into the multiple regression analysis, only the dietary sodium/potassium ratio remained significant, and the regression slope was unchanged. It thus seems unlikely that the dietary sodium/potassium ratio was a surrogate measure for any of these other dietary factors.

A recent analysis from NHANES I\textsuperscript{45} suggesting a threshold effect of dietary calcium found correlations of blood pressure with dietary sodium/potassium ratio only in those consuming less than 400 mg (approxi-
mately 10 mmol) calcium daily. In the Rancho Bernardo cohort, results were not consistent between the sexes: for women, regression of blood pressure on sodium/potassium ratio was stronger in those consuming less than 10 mmol calcium/24 hr, but the reverse was true in men.

As pointed out by Watt and Foy, there are two main blood pressure-sodium hypotheses. The first is that hypertensive individuals consume more sodium than nonhypertensives, and the second is that hypertensive subjects are more sensitive to the effects of sodium than nonhypertensives. Although these are not mutually exclusive, the second does not necessarily require that the hypertensive individual consume more sodium than the nonhypertensive subject for sodium to cause hypertension. Our data support both possibilities: in both men and women, the regression coefficients for blood pressure vs sodium/potassium ratio were steeper for categorically defined hypertensive than for nonhypertensive subjects. Thus, in this cohort, hypertensive subjects appear to be more susceptible to the effects of increasing dietary sodium/potassium ratio. This finding is also in accord with results of trials of sodium restriction in which falls in blood pressure have been observed to be greater in those with higher initial blood pressure. However, although some individuals are undoubtedly more sensitive than others to dietary sodium and potassium, it seems likely that this physiologic attribute, like most others, is determined by a combination of genetic and environmental influences, and is continuously distributed within the population: as illustrated in figure 1, in our study the relationship between increasing dietary sodium/potassium intake and higher systolic blood pressure was apparent over the whole range of blood pressures, not just in hypertensive subjects.

This relationship between blood pressure and dietary sodium/potassium ratio, apparent over the whole range of blood pressures and dietary sodium and potassium intakes, suggests that reducing sodium or increasing potassium intake, even modestly, may have an effect on population mean blood pressures even at lower blood pressure levels. A 10% reduction in sodium intake of about 10 to 20 mmol/day or increase in potassium of 10 to 20 mmol/day is associated with an average 2 to 4 mm Hg decrease in blood pressure. While the magnitude of reduction in blood pressure may be trivial in terms of individual risk, the implications for a population may be sizeable because increased blood pressure is a major risk factor for cardiovascular disease, the leading cause of death in Western communities. Cardiovascular risk increases with every increment in blood pressure and the majority of cardiovascular deaths in the population attributable to increased blood pressure occur at blood pressure levels below treatment thresholds. A reduction of population mean blood pressure by 2 to 4 mm Hg could be estimated to reduce cardiovascular disease by 10% to 20%. Additionally, since mean blood pressure levels rise steeply with age, the population risk attributable to increased blood pressure increases sharply with age. The age gradient in men suggests that at least some of the age-related rise in blood pressure may be due to increased sensitivity to the effects of sodium and potassium on blood pressure with age, and that intervention to reduce sodium and/or increase potassium intake may be particularly appropriate or effective in the older patient.

These findings support the hypothesis that blood pressure within a population is related to dietary sodium and potassium intake and raise questions about other possible known and unknown factors such as age, body mass, index, hormonal status, and other dietary factors that might modify these associations and their mechanisms of action.

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