Salt and blood pressure in Scotland

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Summary and conclusions

Dietary salt intake and urinary sodium excretion were compared in normotensive and hypertensive subjects in Renfrew, Scotland. All groups had high 24-hour urinary salt excretions, and hypertensive subjects did not eat or excrete more salt than normotensive subjects. The only significant relations found were a lower sodium excretion in hypertensive women than in normotensive women ($p < 0.02$) and a lower urinary sodium concentration in hypertensive men than in normotensive men ($p < 0.05$).

These data provide no support for the hypothesis that dietary salt is a major cause of hypertension.

Introduction

The hypothesis that excessive salt ingestion may be an important factor in the development of hypertension is based on much indirect evidence but little direct proof. In primitive societies where salt intake is very low raised blood pressure is rarely seen, and blood pressure does not rise with advancing age.¹⁻³ In patients with hypertension moderate or severe salt restriction leads to a fall in blood pressure.^{4,5} Few studies within a single society, however, have shown higher salt intakes in hypertensive than in normotensive subjects, yet the lack of evidence despite the "salt hypothesis" is widely held.

We have assessed salt intake and urinary sodium excretion in an area of Scotland where the prevalence of hypertension is high⁶ and where there is a correspondingly high incidence of coronary heart disease and stroke.⁷

who at the time of screening in 1972 had diastolic pressures of 105 mm Hg or more or had pressures below this value while receiving treatment for hypertension, having had recorded pressures above 105 mm Hg. After excluding subjects whose pressures were below 90 mm Hg on rescreening 227 patients were left. Of these, 49 either defaulted or died before this study began, and in a further 17 cases it was not considered safe to stop giving diuretics, so these were excluded from the analysis.

Thus 161 hypertensive subjects were eligible for investigation, and 24-hour urine collections were obtained from 110 (68.3%). The normotensive control group comprised 92 subjects representing a 5% random sample of untreated subjects with diastolic pressures below 90 mm Hg at screening. All blood pressures were measured with the London School of Hygiene sphygmomanometer.

All subjects were asked to provide a single 24-hour collection of urine, while taking their normal diet. A simple written instruction sheet was provided. Only those hypertensive subjects who had not taken diuretics for at least 28 days were included. All respondents were later asked to complete a simple questionnaire about their dietary salt,⁹ in which they stated whether they always, sometimes, or never added salt to their food before tasting and whether they preferred salty foods.

Replies to the questionnaire were subdivided into four grades of dietary salt intake.

Results

Table I shows the 24-hour urinary excretion of sodium, potassium, and creatinine in the hypertensive and normotensive subjects. Hypertensive subjects did not have higher urinary sodium excretions than normotensive subjects, and women with hypertension excreted significantly less sodium than their normotensive controls ($p < 0.02$).

As 24-hour urine collections are often incomplete, urinary sodium

TABLE I—Mean ($\pm$ SD) 24-hour urinary, sodium, potassium, and creatinine excretions, 24-hour urinary volume, urinary sodium concentration, and urinary sodium/g creatinine in male and female normotensive and hypertensive subjects

	Men		Women	
	Normotensive (n = 45)	Hypertensive (n = 51)	Normotensive (n = 47)	Hypertensive (n = 59)
24-Hour urinary sodium excretion (mmol/day) ..	196.1 $\pm$ 62.0	182.1 $\pm$ 67.2	173.4 $\pm$ 53.6**	144.4 $\pm$ 59.4**
Urinary sodium/g creatinine (mmol) ..	147.9 $\pm$ 48.9	136.9 $\pm$ 69.1	160.4 $\pm$ 44.3	145.6 $\pm$ 63.2
Urinary sodium concentration (mmol/l) ..	134.4 $\pm$ 43.6*	115.4 $\pm$ 40.3*	113.2 $\pm$ 33.9	103.0 $\pm$ 41.7
24-Hour urinary volume (l/day) ..	1.69 $\pm$ 0.55	1.54 $\pm$ 0.49	1.63 $\pm$ 0.53	1.43 $\pm$ 0.61
24-Hour urinary potassium excretion (mmol/day) ..	46.9 $\pm$ 20.0	47.0 $\pm$ 21.5	46.1 $\pm$ 18.8	41.1 $\pm$ 17.6
24-Hour urinary creatinine excretion (mmol/day) ..	12.1 $\pm$ 3.4	12.9 $\pm$ 4.0	9.7 $\pm$ 2.5	9.2 $\pm$ 2.8

Differences between means significant: * $t = 2.19$, $p < 0.05$; ** $t = 2.63$, $p < 0.02$.

Conversion: SI to traditional units—Urinary creatinine: 1 mmol/24 h $\approx$ 0.1 g/24 h. Urinary sodium and potassium: 1 mmol $\approx$ 1 mEq.

Subjects and methods

The epidemiological study of the 45-64-year-old population of Renfrew, Scotland, has been described elsewhere⁶; subsequently a follow-up clinic was established to study the hypertensive subjects and a random sample of normotensive subjects.⁸

In this analysis the hypertensive group was defined as all subjects

excretions were "corrected" to 1 g of creatinine. After this adjustment no significant differences were observed in urinary sodium excretion; urinary sodium concentrations in hypertensive subjects were if anything lower than that in normotensive subjects. No significant differences were found in urinary potassium concentrations or urinary volumes between men and women, or between hypertensive and normotensive subjects. Important differences between the two sexes were found: men tended to excrete more creatinine and sodium. We therefore could not consider the two sexes together in this analysis.

Not only did hypertensive subjects not excrete more sodium than normotensive subjects but also no correlations were found between blood pressure values and urinary sodium excretions in any hypertensive group studied (men: systolic blood pressure v 24-hour urinary sodium excretion, $\rho = 0.25$; diastolic blood pressure v urinary sodium excretion, $\rho = 0.25$; women: systolic blood pressure v 24-hour urinary sodium excretion, $\rho = 0.04$; diastolic blood pressure v urinary sodium excretion, $\rho = -0.06$). The correlations of blood pressure against urinary sodium in the hypertensive subjects were submitted to a

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Spearman's rank correlation analysis, as the data for blood pressure represented only the upper 10% of the distribution of blood pressure in the whole population. The normotensive data, representing a sample of cases with pressures below 90 mm Hg, were distributed more normally, so conventional regression analysis was possible. No significant relations were found between sodium excretion and blood pressure in the normotensive subjects (women: systolic blood pressure *v* 24-hour urinary sodium excretion, $r=0.0558$; diastolic blood pressure *v* 24-hour urinary sodium excretion, $r=0.0606$; men: systolic blood pressure *v* 24-hour urinary sodium excretion, $r=0.0502$; diastolic blood pressure *v* 24-hour urinary sodium excretion, $r=0.1298$).

The replies to the questionnaire were graded into four levels of sodium intake according to whether subjects said that they routinely added salt to food before tasting or preferred salty foods. This crude assessment failed to show any relation between dietary salt intake and urinary sodium excretion in normotensive subjects, though some trend was seen in hypertensive subjects (table II). Among men 43% of the hypertensive and 48% of the normotensive subjects had higher salt intakes (grades 3 and 4 on questionnaire), while among women 32% of hypertensive and 29% of normotensive subjects had higher intakes. This tendency for men to claim to eat more salt was reflected in their higher 24-hour urinary sodium excretions.

TABLE II—Dietary salt grade as assessed from responses to dietary salt questionnaires in male and female normotensive and hypertensive subjects. (Mean 24-hour urinary sodium excretions (mmol/day) are shown in parentheses)

Salt grade	Men		Women	
	Normotensive	Hypertensive	Normotensive	Hypertensive
1	8 (202)	16 (156)	25 (183)	28 (140)
2	12 (198)	8 (172)	8 (200)	8 (146)
3	13 (187)	11 (209)	8 (138)	11 (176)
4	5 (200)	9 (221)	4 (173)	7 (171)
No reply	7 (201)	7 (161)	2 (179)	5 (115)
Total	45 (196)	51 (182)	47 (173)	59 (144)

Discussion

Despite the high prevalence of hypertension in the Renfrew population (38.9% of this middle-aged population had diastolic pressures of 90 mm Hg or over), this study, like many others, failed to show excess salt ingestion or excretion in subjects with raised blood pressure within a single population.⁹⁻¹² Our study is similar to that of Berglund *et al*¹³ in finding slightly lower urinary sodium excretions in hypertensive subjects than in people with normal pressures.

The Gothenberg group, however, did find positive correlations between urinary salt excretion and blood pressure in normotensive subjects and negative correlations in hypertensive subjects. Although our study was larger than the Gothenberg study, we found no such correlations.

Some studies within a single population have shown greater urinary sodium excretion in hypertensive subjects,^{4,14} while others have found higher reported dietary salt intake.⁹ Using a similar questionnaire, however, neither we nor the Framingham group found such a relationship.

Our study, however, does not disprove the salt hypothesis, as we have detected relatively high salt excretions in a population with a high prevalence of hypertension. Generally, societies that take diets high in salt have higher blood pressures than those with low salt intakes. (If the present data were added to such international comparisons they would tend to confirm the salt hypothesis.) International comparisons, however, suffer from the obvious weakness that many other variables may be present to explain differences in blood pressure. Both hypertension and cardiovascular disease tend to be more common in north European countries, where salt and animal-fat consumption are higher, than in southern European countries such as Italy and Greece. Differences in diagnostic categorisation and other environmental and genetic factors may explain some of the reported differences in disease patterns. High salt intake can

only be one of many factors causing hypertension; the combination of high salt intake with other environmental influences and a genetic predisposition may cause raised blood pressure.

The collection of several 24-hour urine samples for each subject might have provided a more reliable estimate of salt intake, although the day-to-day variation in urinary sodium excretion is large even in subjects receiving a fixed dietary salt intake.¹⁵ The "correction" for creatinine excretion might overcome some of these problems. This technique was used by Parjis *et al*,⁴ and an impressive correlation was found between blood pressure and urinary sodium excretion.

The results of our study, however, do not confirm their observations.

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ONE HUNDRED YEARS AGO I was called to T E, who had attempted to commit suicide by firing a revolver into his mouth. On examining him, I found a very little blood in the mouth. On washing it out, I found the right side of the tongue charred, and an opening through the right tonsil about the size of a goosequill. On exploring this, my director took an upward and backward course to the transverse process of the first cervical vertebra, and then towards the left. I could detect the bullet firmly impacted in the body of that bone. The man seemed none the worse for the injury. I held a consultation, and we decided that it would be injudicious to attempt to remove the bullet. He was committed to prison, and thus I lost sight of him for ten weeks. He is now in perfect health, and in no way suffers from the bullet, which has never been removed. WALTER BUCHANAN, Chatham. (*British Medical Journal*, 1880.)