

How Far Should Salt Intake Be Reduced?

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Abstract—The current public health recommendations are to reduce salt intake from 9 to 12 g/d to 5 to 6 g/d. However, these values are based on what is feasible rather than the maximum effect of salt reduction. In a meta-analysis of longer-term trials, we looked at the dose response between salt reduction and fall in blood pressure and compared this with 2 well-controlled studies of 3 different salt intakes. All 3 studies demonstrated a consistent dose response to salt reduction within the range of 12 to 3 g/d. A reduction of 3 g/d predicts a fall in blood pressure of 3.6 to 5.6/1.9 to 3.2 mm Hg (systolic/diastolic) in hypertensives and 1.8 to 3.5/0.8 to 1.8 mm Hg in normotensives. The effect would be doubled with a 6 g/d reduction and tripled with a 9 g/d reduction. A conservative estimate indicates that a reduction of 3 g/d would reduce strokes by 13% and ischemic heart disease (IHD) by 10%. The effects would be almost doubled with a 6 g/d reduction and tripled with a 9 g/d reduction. Reducing salt intake by 9 g/d (eg, from 12 to 3 g/d) would reduce strokes by approximately one third and IHD by one quarter, and this would prevent ≈20 500 stroke deaths and 31 400 IHD deaths a year in the United Kingdom. The current recommendations to reduce salt intake from 9 to 12 g/d to 5 to 6 g/d will have a major effect on blood pressure and cardiovascular disease but are not ideal. A further reduction to 3 g/d will have a much greater effect and should now become the long-term target for population salt intake worldwide. (*Hypertension*. 2003;42:1093-1099.)

Key Words: sodium, dietary ■ blood pressure ■ dose response ■ cardiovascular diseases

High blood pressure is the most important risk factor for cardiovascular disease.¹ A recent World Health Organization report states that elevated blood pressure alone causes ≈50% of cardiovascular disease worldwide.¹ A more recent article in *The Lancet* demonstrates that nonpersonal health interventions, including government action to stimulate a reduction in the salt (sodium chloride) content of processed foods, are cost-effective ways to limit cardiovascular disease and could avert >21 million disability-adjusted life years per year worldwide.² A recent meta-analysis of 1 million adults in 61 prospective studies demonstrates that the relation between blood pressure and cardiovascular risk is much stronger than previously estimated.³ Much evidence from epidemiologic,⁴ migration,⁵ intervention,⁶ genetic,⁷ and animal⁸ studies suggests that salt intake plays an important role in regulating blood pressure. Several meta-analyses of randomized, salt reduction trials have been published in the last few years.⁹⁻¹² However, most of the previous meta-analyses included trials of very short duration (eg, 5 days) and included trials with a short-term salt loading followed by salt deprivation (eg, from 20 to less than 1 g/d) for only a few days.^{9,11} These short-term salt loading and salt depletion experiments are not appropriate for helping to inform public health policy, which is for a more modest reduction in salt intake over a more prolonged period of time. A more recent meta-analysis by Hooper et al¹² is an important attempt to look at whether long-term salt reduction (ie, >6 months) in randomized trials causes a fall in

blood pressure. However, most trials included in this meta-analysis only achieved a very small reduction in salt intake, and on average, salt intake was reduced by only 2 g/d. It is therefore not surprising that there was only a small, but still highly significant, fall in blood pressure. Furthermore, an important point as to whether there is a dose response to salt reduction has been overlooked in this meta-analysis.

Salt intake in many countries is between 9 and 12 g/d. The current World Health Organization recommendations for adults are to reduce salt intake to 5 g/d or less,¹³ and the UK¹⁴ and US recommendations¹⁵ are 6 g/d or less. However, these recommendations are based on what is feasible and not on what might have been the maximum impact on blood pressure and cardiovascular disease. Recent evidence suggests that these levels, although they might be feasible, are too high.

Studies in experimental animals have shown a clear dose response between salt intake and blood pressure: the higher the salt intake, the higher the blood pressure.¹⁶ A recent study in chimpanzees, the animal species closest to humans with 98.4% genetic identity, demonstrated a dose response when salt was increased from their usual intake of 0.5 g/d to 5, 10, and 15 g/d.⁸ In humans, it is difficult to conduct such trials, particularly to keep individuals on a low-salt diet long term because of the widespread presence of salt in nearly all processed, restaurant, canteen, and fast foods. However, 2 well-controlled trials have studied 3 salt intakes (ie, from

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11.2, 6.4, to 2.9 g/d in 1 trial¹⁷ and from 8.3, 6.2, to 3.8 g/d in the other¹⁸), and both showed a clear dose response to salt reduction.

To study this dose-response relation further, we reanalyzed a recent meta-analysis of randomized, longer-term, salt reduction trials¹⁹ and looked at the dose-response relation and compared this with the 2 well-controlled studies of 3 salt intakes to try to determine the optimal salt intake that would have the maximum impact on blood pressure, strokes, and ischemic heart disease (IHD).

Methods

The methods of the meta-analysis were reported in detail elsewhere¹⁹ and are summarized here. The inclusion criteria were as follows:

1. Random allocation to either a reduced salt intake or usual salt intake (ie, control).
2. No concomitant interventions in either group.
3. Net reduction in 24-hour urinary sodium ≥ 40 mmol (2.4 g of salt per day). Net reduction in 24-hour urinary sodium was calculated as UNa (Post)–UNa (Pre) for crossover trials, where UNa (Post) designates urinary sodium on the reduced salt intake and UNa (Pre) designates urinary sodium on the usual salt intake. In parallel trials, the net change in urinary sodium was calculated as $\{[UNa (Post)–UNa (Pre)]_{\text{reduced salt group}}\} - \{[UNa (Post)–UNa (Pre)]_{\text{control group}}\}$.
4. Duration of salt reduction must have been for 4 or more weeks.
5. Study participants were not children or pregnant.

Mean effect sizes were calculated by weighting each trial by the inverse of the variance. Weighted linear regression was used to examine the dose-response relation between the change in 24-hour urinary sodium and the change in blood pressure.²⁰ From the regression line, we calculated the predicted falls in blood pressure with a reduction of 3, 6, and 9 g/d in salt intake.

Results

Trials in Hypertensive Individuals

The characteristics of individual trials included in the meta-analysis were reported in detail elsewhere¹⁹ and are summarized here. Seven hundred thirty-four hypertensive individuals were studied in 17 trials.^{17,18,21–35} Median age was 50 years (ranging from 24 to 73 years). The study duration varied from 4 weeks to 1 year (median, 6 weeks). The median 24-hour urinary sodium on the usual salt intake was 161 mmol (9.5 g of salt per day), ranging from 125 to 191 mmol (7.4 to 11.2 g of salt per day), and on the reduced salt intake it was 87 mmol (5.1 g of salt per day), ranging from 57 to 117 mmol (3.4 to 6.9 g of salt per day). The median net change in 24-hour urinary sodium was -78 mmol (4.6 g of salt per day), ranging from -53 to -117 mmol (3.1 to 6.9 g of salt per day).

The pooled estimates of changes in blood pressure were -5.0 ± 0.4 mm Hg (mean \pm SEM, $P < 0.001$; 95% confidence interval [CI], -5.8 to -4.2 mm Hg) for systolic and -2.7 ± 0.2 mm Hg ($P < 0.001$; 95% CI, -3.2 to -2.3 mm Hg) for diastolic pressure.

To examine whether there was a dose-response relation between the changes in 24-hour urinary sodium and the changes in blood pressure, we performed weighted linear regression and assumed a zero intercept. The assumption for using this model was that the absence of a change in urinary sodium would be associated with no change in blood pres-

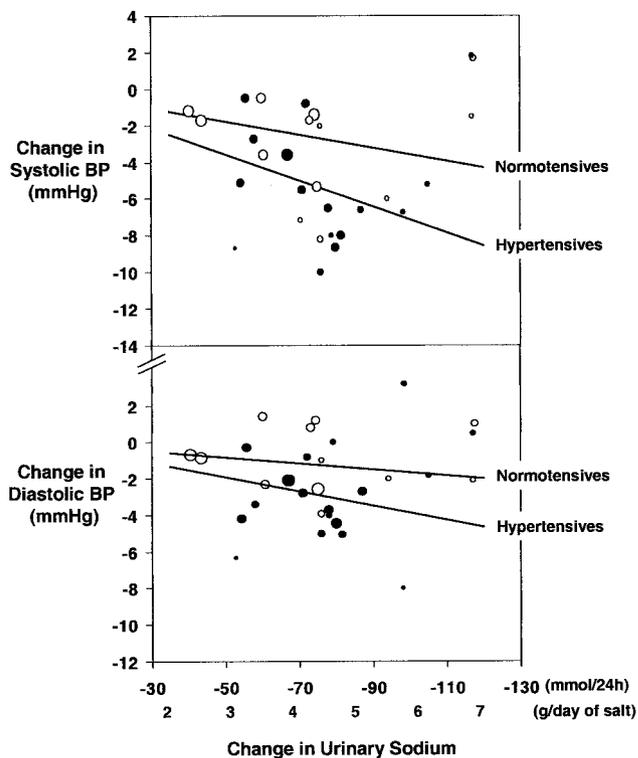


Figure 1. Relation between the net change in 24-hour urinary sodium excretion and blood pressure in the meta-analysis. Open circles represent normotensives and solid circles represent hypertensives. The slope is weighted by the inverse of the variance of the net change in blood pressure. The size of the circle is proportional to the weight of the trial.

sure, ie, all other factors being equal between 2 randomized treatments. The results showed a significant dose response to salt reduction ($P < 0.001$ for both systolic and diastolic pressures; Figure 1). A reduction of 50 mmol/d (3 g/d) in salt intake predicts a fall in blood pressure of 3.6/1.9 mm Hg. A reduction of 100 mmol/d (6 g/d) predicts a fall in blood pressure of 7.1/3.9 mm Hg. A reduction of 150 mmol/d (9 g/d) in salt intake would cause a fall in blood pressure of 10.7/5.8 mm Hg.

Trials in Normotensive Individuals

Two thousand two hundred twenty normotensive individuals were studied in 11 trials.^{18,35–43} Median age was 47 years (ranging from 22 to 67 years). The study duration varied from 4 weeks to 3 years (median, 4 weeks). The median 24-hour urinary sodium on the usual salt intake was 154 mmol (9.1 g of salt per day), ranging from 128 to 200 mmol (7.5 to 11.8 g of salt per day), and on the reduced salt intake it was 82 mmol (4.8 g of salt per day), ranging from 56 to 135 mmol (3.3 to 7.9 g of salt per day). The median net change in 24-hour urinary sodium was -74 mmol (4.4 g of salt per day), ranging from -40 to -118 mmol (2.4 to 6.9 g of salt per day).

The pooled estimates of changes in blood pressure were -2.0 ± 0.3 mm Hg ($P < 0.001$; 95% CI, -2.6 to -1.5 mm Hg) for systolic and -1.0 ± 0.2 mm Hg ($P < 0.001$; 95% CI, -1.4 to -0.6 mm Hg) for diastolic pressure. The dose-response analysis with fixing the intercept showed a

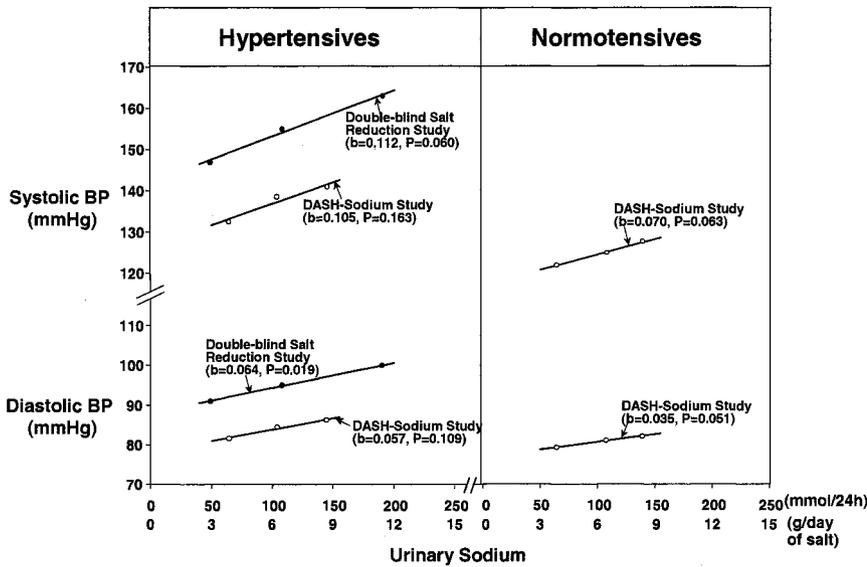


Figure 2. Dose-response relation between 24-hour urinary sodium and blood pressure in the double-blind salt reduction study and the DASH-Sodium study.

significant dose response to salt reduction ($P < 0.001$ for systolic and $P < 0.05$ for diastolic pressures; Figure 1). A reduction of 3, 6, and 9 g/d in salt intake predicts a fall in blood pressure of 1.8/0.8, 3.6/1.7, and 5.4/2.5 mm Hg, respectively.

Trials in All Individuals

When all individuals were grouped together, there were 2954 subjects (25% were hypertensives). Median age was 49 years (ranging from 22 to 73 years). The study duration varied from 4 weeks to 3 years (median, 5 weeks). The pooled estimates of changes in blood pressure were -2.9 ± 0.2 mm Hg ($P < 0.001$; 95% CI, -3.4 to -2.5 mm Hg) for systolic and -1.7 ± 0.2 mm Hg ($P < 0.001$; 95% CI, -2.0 to -1.4 mm Hg) for diastolic pressure. The dose-response analysis with fixing the intercept showed a significant dose response to salt reduction ($P < 0.001$ for systolic and $P < 0.001$ for diastolic pressures). A reduction of 3, 6, and 9 g/d in salt intake predicts a fall in blood pressure of 2.5/1.4, 5.0/2.8, and 7.5/4.2 mm Hg, respectively.

Dose Response Between Salt Intake and Blood Pressure in Our Previous Double-Blind Study and the DASH-Sodium Study

The best way to study the dose-response relation between salt intake and blood pressure is to look at the blood pressure responses to several levels of salt intake for a long term. So far, there are only 2 well-controlled trials that studied 3 salt intakes, each for 4 weeks. One is our double-blind study in 19 patients with untreated essential hypertension,¹⁷ and the other is the Dietary Approaches to Stop Hypertension (DASH)-Sodium study,¹⁸ in which 79 untreated hypertensives and 116 normotensives were studied on the normal American diet, and 81 untreated hypertensives and 121 normotensives were studied on the DASH diet.

In our double-blind study with 3 salt intakes, the blood pressure decreased by 8/5 mm Hg when salt intake, as judged by 24-hour urinary sodium, changed from 190 to 108 mmol/24 h

(11.2 to 6.4 g/d), and the blood pressure decreased by 8/4 mm Hg when salt intake changed from 108 to 49 mmol/24 h (6.4 to 2.9 g/d).¹⁷ The DASH-Sodium study showed that in all individuals (ie, both hypertensives and normotensives) who were studied on the normal American diet, the blood pressure decreased by 2.1/1.1 mm Hg when salt intake changed from 141 to 106 mmol/24 h (8.3 to 6.2 g/d) and by 4.6/2.4 mm Hg when salt intake changed from 106 to 64 mmol/24 h (6.2 to 3.8 g/d).¹⁸ In those who were studied on the DASH diet, the blood pressure decreased by 1.3/0.6 mm Hg when salt intake changed from 144 to 107 mmol/24 h (8.5 to 6.3 g/d) and by 1.7/1.0 mm Hg when salt intake changed from 107 to 67 mmol/24 h (6.3 to 3.9 g/d).¹⁸

To study the dose-response relation between salt intake and blood pressure, we performed linear regression analysis on these 2 studies by using the mean 24-hour urinary sodium and mean blood pressure at 3 salt intakes. For the purpose of comparison, we included only a subgroup of hypertensive and normotensive individuals who were studied on the normal American diet in the DASH-Sodium study. As shown in Figure 2, the 2 studies showed a very similar dose-response relation between salt intake and blood pressure. Within the range of 11.2 to 2.9 g of salt per day, the lower the salt intake, the lower the blood pressure. In patients with essential hypertension, a reduction of 3, 6, and 9 g/d in salt intake predicts a fall in blood pressure of 5.6/3.2, 11.2/6.4, and 16.8/9.6 mm Hg, respectively, in the double-blind, salt reduction study. In the DASH-Sodium study, the same reductions in salt intake predict a fall in blood pressure of 5.3/2.9, 10.5/5.7, and 15.8/8.6 mm Hg, correspondingly. The DASH-Sodium study also showed that in normotensive individuals, there was a dose response to salt reduction (Figure 2). A reduction of 3, 6, and 9 g/d in salt intake predicts a fall in blood pressure of 3.5/1.8, 7.0/3.5, and 10.5/5.3 mm Hg, respectively, in normotensives.

Comparison of the Dose-Response Relation Among 3 Studies

To compare the dose-response relation among 3 studies, we superimposed the regression lines found in the 2 studies with

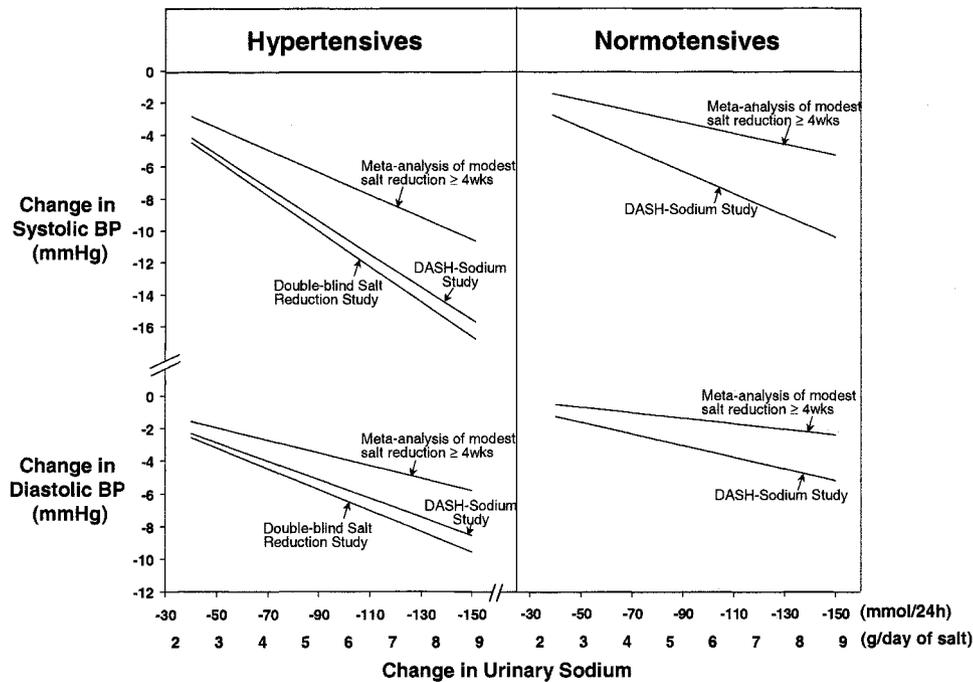


Figure 3. Comparison of the dose-response relation among 3 studies.

3 salt intakes on our meta-analysis (Figure 3). There was a similar dose response in all 3 studies. However, the regression line from our meta-analysis was flatter. This is not surprising, because some less well-controlled studies were included in the meta-analysis, and the diversity of patients recruited to different trials (eg, age, ethnic group, baseline blood pressure and potassium intake) might also have affected the blood pressure responses. Table 1 shows the predicted falls in blood pressure with reductions of 3, 6, and 9 g/d of salt intake in 3 studies. A reduction of 3 g/d in salt intake would have an effect on blood pressure, but the effect would be doubled with a 6 g/d reduction and tripled with a 9 g/d reduction in salt intake.

Discussion

Our meta-analysis of randomized, longer-term, salt reduction trials demonstrates a dose response to salt reduction. More

importantly, the dose response found in our meta-analysis is consistent with the dose response found in the 2 well-controlled trials with 3 salt intakes. Within the range of 12 to 3 g of salt per day, the lower the salt intake, the lower the blood pressure. The current public health recommendations to reduce salt intake from 9 to 12 g/d to 5 to 6 g/d will have a major effect on blood pressure but are no means ideal. A further reduction to 3 g of salt per day will have a much greater effect on blood pressure.

One important point is that it is not clear from the 3 studies whether the dose response to salt reduction is linear or nonlinear. In hypertensives, both the DASH-Sodium study and our double-blind study of 3 salt intakes showed a nonlinear dose response for systolic pressure, ie, a steeper dose response at a lower level of salt intake. In other words, for a given reduction in salt intake, the fall in systolic blood pressure is larger when salt intake is at a lower level.

TABLE 1. Predicted Falls in Blood Pressure With Salt Reduction in 3 Studies

Study and Measure	Reduction in Salt Intake					
	3 g/d (50 mmol/d)		6 g/d (100 mmol/d)		9 g/d (150 mmol/d)	
	Hypertensive	Normotensive	Hypertensive	Normotensive	Hypertensive	Normotensive
Meta-analysis of modest salt reduction¹⁹						
Fall in systolic blood pressure, mm Hg	3.6	1.8	7.1	3.6	10.7	5.4
Fall in diastolic blood pressure, mm Hg	1.9	0.8	3.9	1.7	5.8	2.5
Double-blind salt reduction study¹⁷						
Fall in systolic blood pressure, mm Hg	5.6	...	11.2	...	16.8	...
Fall in diastolic blood pressure, mm Hg	3.2	...	6.4	...	9.6	...
DASH-Sodium study¹⁸						
Fall in systolic blood pressure, mm Hg	5.3	3.5	10.5	7	15.8	10.5
Fall in diastolic blood pressure, mm Hg	2.9	1.8	5.7	3.5	8.6	5.3

DASH indicates Dietary Approaches to Stop Hypertension.

TABLE 2. Predicted Reductions in Stroke and IHD Deaths With Reductions in Salt Intake

Measure	Reduction in Salt Intake					
	3 g/d (50 mmol/d)		6 g/d (100 mmol/d)		9 g/d (150 mmol/d)	
	SBP	DBP	SBP	DBP	SBP	DBP
Fall in BP in all participants, mm Hg (from the meta-analysis)	2.5	1.4	5	2.8	7.5	4.2
Reduction in stroke death, %	12	14	23	25	32	36
Stroke deaths prevented in UK, n/y	7300	8300	13,700	15,500	19,300	21,600
Reduction in IHD death, %	9	10	16	19	23	27
IHD deaths prevented in UK, n/y	10,600	12,400	20,300	23,600	29,100	33,700

Blood pressure fall taken from the meta-analysis. IHD indicates ischemic heart disease; SBP, systolic blood pressure; DBP, diastolic blood pressure; and BP, blood pressure.

However, for diastolic blood pressure in both the DASH-Sodium study and our double-blind study, the dose-response relation appears to be linear, and in the DASH-Sodium study in normotensives, there appears to be a linear dose response for both systolic and diastolic blood pressure.

For the purpose of comparison, only subgroup data of the DASH-Sodium study were included in our analyses. It is of note that the overall results of the DASH-Sodium study showed a nonlinear dose response to salt reduction both on the normal American diet and on the DASH diet, indicating that salt reductions have greater effects on blood pressure with salt intake at lower levels. In our meta-analysis, we assumed a linear relation between the change in 24-hour urinary sodium and the change in blood pressure. This, if anything, would underestimate the effect of salt reduction on blood pressure at lower levels of salt intake if the dose-response relation were nonlinear.

The recent UK National Diet and Nutrition Survey, which was carried out between 2000 and 2001 in a nationally representative sample of 1495 adults aged 19 to 64 years, showed that 24-hour urinary sodium was 187 mmol (11.0 g/d of salt) for men and 139 mmol (8.1 g/d of salt) for women.⁴⁴ However, 24-hour urinary sodium is an underestimate of dietary salt intake. A study by Pietinen⁴⁵ showed that the 24-hour urinary sodium was, on average, 93% of dietary salt intake. Therefore, the average salt intake in the United Kingdom is actually between 8.7 and 11.8 g/d. A conservative estimate of the impact on cardiovascular disease (ie, from the falls in blood pressure in all individuals in the meta-analysis) indicates that a reduction of 3 g/d in salt intake would result in a fall in blood pressure of 2.5/1.4 mm Hg, and this would reduce strokes by 12% (estimated from systolic) to 14% (from diastolic) and ischemic heart disease (IHD) by 9% to 10%.³ In the United Kingdom, the total number of stroke deaths is 60 666 per year, and the total number of IHD deaths is 124 037 per year.⁴⁶ Therefore, a reduction of 3 g/d in salt intake would prevent ≈7300 to 8300 stroke deaths and 10 600 to 12 400 IHD deaths per year. As shown in Table 2, the effects on strokes and IHD would be almost doubled if salt intake were reduced by 6 g/d and tripled with a 9 g/d reduction. A reduction of 9 g/d in salt intake (eg, from 12 to 3 g/d) would result in a fall in blood pressure of 7.5/4.2 mm Hg, and this would reduce strokes by approximately one third and IHD by one quarter. In the United Kingdom,

this would prevent ≈20 500 stroke deaths and 31 400 IHD deaths per year. These numbers of stroke and IHD deaths prevented are likely to be an underestimate, because the falls in blood pressure in the much better controlled DASH-Sodium study¹⁸ and our double-blind study¹⁷ are larger and would have an even greater impact.

It is important to note that even in normotensives alone, salt reductions would have large effects on stroke and IHD. A reduction of 3 g/d in salt intake would lower blood pressure by 1.8/0.8 mm Hg, and this would reduce strokes by 9% and IHD by 6% in normotensives alone. A reduction of 6 g/d would reduce strokes by 17% and IHD by 12%, and a reduction of 9 g/d would reduce strokes and IHD by 24% and 18%, respectively, in normotensives.

In our article, we reported the reductions in blood pressure, stroke, and IHD death with reductions of 3, 6, and 9 g/d in salt intake. The long-term target should be to reduce salt intake to 3 g/d. Therefore, if individuals have a salt intake of 12 g/d, then they need to reduce salt intake by 9 g/d, and if individuals have a salt intake of 9 g/d, then they need to reduce salt intake by 6 g/d. The effects of these reductions in salt intake on blood pressure, stroke, and IHD deaths are shown in Table 1 and Table 2.

The levels of salt intake in many countries are similar to those in the United Kingdom. The percentage reduction in stroke and IHD applies to many countries. Therefore, the reductions in stroke and IHD worldwide, if salt intake were reduced from the current intake of 9 to 12 g/d to the now-recommended levels of 5 to 6 g/d, would be immense. The effects on stroke and IHD would be much greater if salt intake were reduced further, ie, to 3 g/d.

Although we calculated only the numbers of stroke and IHD deaths that could be prevented with reductions in salt intake, the percentage reductions in stroke and IHD apply to the incidence as well. Approximately 50% of patients who suffer stroke or heart attack survive; therefore, there would be a proportionate reduction in the numbers of these people. This would result in a reduction in disability and major cost savings both to individuals, their families, and the Health Service. Furthermore, high blood pressure is an important risk factor for heart failure. A reduction in salt intake would therefore have a major effect on heart failure.

From our studies, it is not clear whether reducing salt intake to <3 g/d has a greater effect on blood pressure,

because no randomized trials with a duration of 4 or more weeks have reduced salt intake to <3 g/d. However, the observational epidemiologic studies suggest that within the range of 14 to 0.01 g of salt per day, there is a dose response between salt intake and blood pressure, ie, the lower the salt intake, the lower the blood pressure.^{4,47} Despite the potential greater benefits of reducing salt intake to <3 g/d, it is currently impractical to achieve a reduction to <3 g/d, given the current amount of salt in processed foods that accounts for 75% to 80% of our salt intake.⁴⁸

In conclusion, our meta-analysis of longer-term salt reduction trials and the 2 well-controlled studies with 3 salt intakes demonstrate a consistent dose-response relation between salt intake and blood pressure within the range of 12 to 3 g of salt per day. Importantly, the dose-response relation exists in both hypertensive and normotensive subjects. The current public health recommendations to reduce salt intake from 9 to 12 g/d to 5 to 6 g/d will have a major effect on blood pressure and cardiovascular disease but are not ideal. A reduction to 3 g of salt per day will have a much greater effect and should now become the long-term target for population salt intake worldwide.

Perspectives

The totality of evidence that links salt intake to blood pressure is now overwhelming. Current recommendations are to reduce salt intake from 9 to 12 g/d to 5 to 6 g/d. Our article demonstrates that although these reductions will have a major effect on blood pressure and cardiovascular disease, reducing salt intake further to 3 g/d will have additional large effects. Therefore, the target of 5 to 6 g/d should be seen as an interim target, and the long-term target for population salt intake worldwide should now be 3 g/d. This will be difficult, particularly because in most developed countries, 75% to 80% of salt intake now comes from salt added to processed foods. In our view, the strategy should be that the food industry should gradually reduce the salt concentration of all processed foods, starting with a 10% to 25% reduction, which is not detectable by consumers, and continuing a sustained reduction over the course of the next decade. This strategy has now been adopted in the United Kingdom by both the Department of Health and Food Standards Agency, and several leading supermarkets and food manufacturers have already started to implement such changes. Of all the dietary changes to try and prevent cardiovascular disease, a reduction in salt intake is the easiest change to make, because it can be done without the consumers' knowledge but will require the cooperation of the food industry. Clearly, it would be helpful if individuals also reduced the amount of salt that they add to their own cooking or to their food. If this strategy were implemented and achieved and the 3 g/d target were reached throughout the world, there would be immense reductions in strokes, heart attacks, and heart failure.

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