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Reducing Population Salt Intake Worldwide: From Evidence to Implementation

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Abstract Raised blood pressure is a major cause of cardiovascular disease, responsible for 62% of stroke and 49% of coronary heart disease. There is overwhelming evidence that dietary salt is the major cause of raised blood pressure and that a reduction in salt intake lowers blood pressure, thereby, reducing blood pressure-related diseases. Several lines of evidence including ecological, population, and prospective cohort studies, as well as outcome trials, demonstrate that a reduction in salt intake is related to a lower risk of cardiovascular disease. Increasing evidence also suggests that a high salt intake may directly increase the risk of stroke, left ventricular hypertrophy, and renal disease; is associated with obesity through soft drink consumption; is related to renal stones and osteoporosis; is linked to the severity of asthma; and is probably a major cause of stomach cancer. In most developed countries, a reduction in salt intake can be achieved by a gradual and sustained reduction in the amount of salt added to foods by the food industry. In other countries where most of the salt consumed comes from salt added during cooking or from sauces, a public health campaign is needed to encourage consumers to use less salt. Several countries have already reduced salt intake. The challenge now is to spread this out to all other countries. A modest reduction in population salt intake worldwide will result in a major improvement in public health. (Prog Cardiovasc Dis 2010;52:363-382)

Keywords: Dietary salt; Blood pressure; Cardiovascular disease; Renal disease; Obesity; Stomach cancer; Salt reduction programs

Humans, like all other mammals, consumed less than 0.25 g of salt per day during several million years of evolution. About 5000 years ago, the Chinese discovered that salt could be used to preserve foods. Salt then became of great economic importance as it was possible to preserve foods during the winter and allowed the development of settled communities. Salt was the most taxed and traded commodity in the world, with intake reaching a peak around the 1870s. However, with the invention of the deep freezer and the refrigerator, salt was no longer required as a preservative. Salt intake had been declining, but with the recent large increase in the consumption of highly salted processed foods, salt intake is now increasing again. The average salt intake in most countries around the world is approximately 9 to 12 g/d, with many Asian countries having mean intakes more than 12 g/d.[1] Salt intake is commonly more than 6 g/d in children older than 5 years and increases with age.[1]

Humans are genetically programmed to a salt intake of less than 0.25 g/d. The recent changes (in evolutionary terms) to a high salt intake present a major challenge to the physiologic systems to excrete these large amounts of salt through the kidneys. The consequence is that the high salt intake causes a rise in blood pressure (BP),[2,3] thereby, increasing the risk of cardiovascular disease (CVD),[4,5] and renal disease.[6-8] Furthermore, a high salt intake may

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Abbreviations and Acronyms

BP = blood pressure

CHD = coronary heart disease

CVD = cardiovascular disease

DASH = Dietary Approaches to Stop Hypertension

eNOS = endothelial nitric oxide synthase

TOHP = Trial of Hypertension Prevention

have direct effects on stroke,[9,10] left ventricular hypertrophy,[11] progression of renal disease, and proteinuria,[6] independent of but additive to the effect of salt on BP. There is also increasing evidence that salt intake is indirectly related to obesity through soft drink consumption,[12,13] associated with an increased risk of renal stones and osteoporosis,[14] linked to the severity of asthma,[15] and is probably a major cause of stomach cancer.[16]

In this article, we provide a comprehensive review on the evidence for the harmful effects of a high salt intake and the beneficial effects of reducing salt consumption. In addition, we provide a brief update on the current salt reduction programs that have been successfully carried out in several countries.

Salt and sodium

The terms salt and sodium are often used synonymously. However, on a weight basis, salt comprises 40% sodium and 60% chloride. The conversion of different units for sodium and salt is as follows: 1 g sodium = 2.5 g salt; 1 mmol sodium = 23 mg sodium; 1 g salt = 0.4 g sodium; and 1 g salt = 17 mmol sodium. Salt is the major source of sodium in the diet (approximately 90%). Throughout this review, we use the term *salt* for simplicity.

Salt and BP

Raised BP is a major cause of CVD, responsible for 62% of stroke and 49% of coronary heart disease. Importantly, the risk of CVD increases throughout the range of BP, starting at 115/75 mm Hg.[17] It has been shown that a high salt intake, a low consumption of fruit and vegetables (ie, low potassium intake), obesity, excess alcohol intake, and lack of physical exercise all contribute to the development of high BP. However, the diversity and strength of the evidence is much greater for salt than for other factors.

Animal studies

Studies in different species of animals, for example, rat, dog, chicken, rabbit, baboon, and chimpanzee have shown that salt intake plays an important role in regulating BP.[18,19] Furthermore, in all forms of experimental hypertension, whatever the animal model, a high salt intake is essential for BP to rise. A study in chimpanzees (98.8% genetic homology with man) demonstrated that a gradual increase in salt intake from 0.5 g/d that is close to humans' evolutionary intake, to 10 to 15 g/d that is similar to our current salt intake, caused progressive and large increases in BP (Fig 1).[18] At the end of the 20-month study, the salt supplements were stopped and BP declined to that of the control group.

Human genetic studies

Molecular genetic studies in humans have identified mutations in several genes that cause Mendelian forms of hypertension or hypotension.[20] All of these affect the kidney's ability to excrete sodium, and salt intake has dramatic effects on BP in these individuals. Although such genetic disorders are very rare, these studies clearly indicate the importance of salt intake in regulating BP.

Epidemiological studies

A number of studies in primitive societies that did not have access to salt have shown a lower BP compared to developed societies. Although several factors may contribute to the lower BP, numerous studies have demonstrated the profound importance of salt intake. For instance, a study in the Pacific Islands where one undeveloped community used seawater in their foods and the other did not, showed that the community using seawater had higher BP.[21] Another study of 2 rural communities in Nigeria, one of which had access to salt from a salt lake and the other did not, showed differences in salt intake and differences in BP, and yet in all other aspects of lifestyle and diet, the 2 communities were similar.[22] The Qash'qai, an undeveloped tribe living in Iran who had access to salt deposits on the

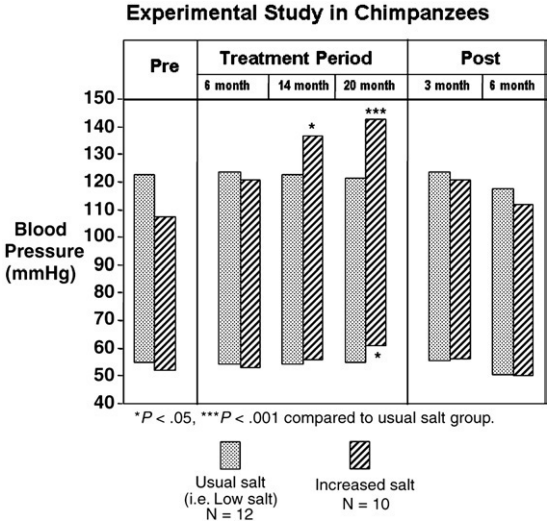

Fig 1. Blood pressure in chimpanzees that either continued on their usual low-salt diet or were given an increased salt intake. At the end of the 20-month study, the salt supplements were stopped and blood pressure declined to that of the control group. Adapted from Denton et al.[18]

ground, developed high BP and a rise in BP with age similar to that which occurred in developed communities, but in all aspects, they lived a lifestyle similar to undeveloped communities who did not have access to salt.[23]

In spite of this evidence, it was felt necessary to set up a large international study on salt and BP (INTERSALT)[2] using standardized method for measuring BP and 24-hour urinary sodium. The intention was to study communities with a wide range of salt intake, for example, from 0.5 to 25 g/d. However, among the 52 communities recruited into the study, only 4 had a low salt intake (ie, ≤3 g/d) and most lay between 6 and 12 g/d and none had the high salt intake as originally envisaged. Nevertheless, the study demonstrated a significant positive relationship between salt intake and BP. There was also a highly significant positive relationship between salt intake and the increase in BP with age (Fig 2). It was estimated that an increase of 6 g/d in salt intake over 30 years would lead to an increase in systolic BP by 9 mm Hg.[2]

One criticism of the INTERSALT study made by the Salt Institute (a public relations company defending the interests of salt extractors and manufacturers worldwide) was that when the 4 communities consuming lower salt were excluded, there was no overall relationship remaining between salt intake and BP in the 48 communities. The INTERSALT's investigators reanalyzed their data and showed that the highly significant within-population association between salt intake and BP across all 52 centers was virtually unchanged when the 4 low-salt populations were excluded, and the association between salt intake and the rise in BP with age persisted across 48 centers.[2,24,25]

More recent epidemiological studies, for example, the international study of macro- and micronutrients and BP[26] and the Norfolk Cohort of the European Prospective Investigation into Cancer,[27] have lent further support for the important role of salt intake in determining the levels of population BP.

Migration studies

Several studies showed that migration from isolated low-salt societies to an urban environment with an increased salt intake was associated with a rise in BP.[28,29] For example, a well-controlled migration study of a rural tribe in Kenya demonstrated that on migration to Nairobi, there was an increase in salt intake and a reduction in potassium intake, and BP rose compared to those in a similar control group who remained in the rural environment.[29]

Population-based intervention studies

Population-based intervention studies have shown that when salt intake was successfully decreased, there was a reduction in population BP.[30,31] However, as some of the study populations failed to achieve a reduction in salt intake, there was no change in BP.[32,33] The most successful intervention study is the one conducted in 2 similar villages in Portugal[30] that achieved a difference of approximately 50% in salt intake between the 2 villages. After 2 years' intervention, there was a difference of 13/6 mm Hg in BP (Fig 3). A recent randomized community-based intervention trial in 2 rural villages in northeastern Japan demonstrated that dietary counseling for 1 year reduced salt intake by 2.3 g/d as measured by 24-hour urinary sodium, and this was associated with a decrease of 3.1 mm Hg in systolic BP.[34]

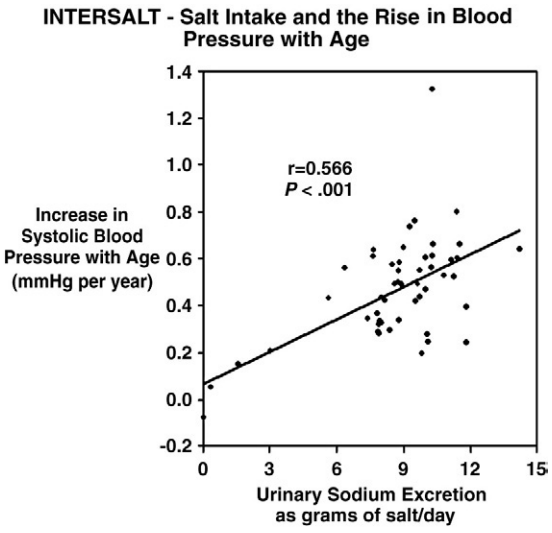

Fig 2. Relationship between salt intake and the slope of the rise in systolic BP with age in 52 centers in the INTERSALT study. Adapted from INTERSALT.[2]

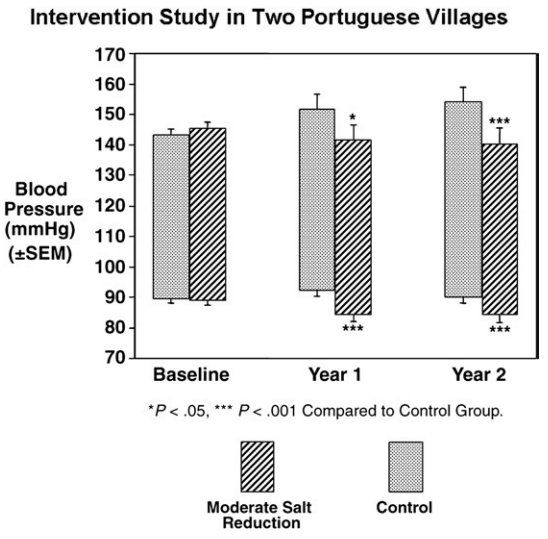

Fig 3. Changes in BP in 2 Portuguese villages in an intervention study. With the intervention, there was a difference of 42% in salt intake between the 2 villages at 1 year and 47% at 2 years. This was associated with a significant difference in BP. Adapted from Forte et al.[30]

Treatment trials

Ambard and Beaujard, in 1904, were the first to show that a large reduction in salt intake lowered BP. These results were confirmed over the next 30 years by several workers, but it was not until Kempner resuscitated the idea of severe salt restriction that it became widely used in the treatment of hypertension.[35] More recently, randomized trials have studied the effects of modest reductions in salt intake, that is, from the current intake of approximately 9 to 12 g/d to approximately 5 to 6 g/d and have shown that the falls in BP were equivalent to single drug therapy in hypertensive individuals,[36] and there were also significant falls in BP in those with normal BP.

Several meta-analyses of salt reduction trials have been performed.[37-41] In 2 meta-analyses,[38,40] it was claimed that salt reduction had very little effects on BP in individuals with normal BP and a reduction in population salt intake was not warranted. However, these 2 meta-analyses are flawed. Both included trials of very short duration with many comparing the effects of short-term salt loading to abrupt and severe salt restriction for only a few days. It is known that such acute and large reductions in salt intake increase sympathetic activity, plasma renin activity, and angiotensin II,[42] which would counteract the effects on BP. Furthermore, most BP-lowering drugs do not exert their maximal effects within a few days. This is particularly true with diuretics that are likely to work by a similar mechanism to that of salt reduction. It is therefore inappropriate to include the short-term salt restriction trials in a meta-analysis that attempts to apply them to public health recommendations for a longer term modest reduction in salt intake. A meta-analysis by Hooper et al[41] attempted to look at whether salt reduction for 6 months or more caused a fall in BP. However, most trials included in this meta-analysis achieved only a very small reduction in salt intake. It is, therefore, not surprising that there was only a small but still significant fall in BP. A more recent meta-analysis of randomized trials of 1 month or longer demonstrated that a modest reduction in salt intake caused significant and important falls in BP in both hypertensive and normotensive individuals.[43] Furthermore, there was a dose response to salt reduction. A reduction of 6 g/d would lower BP by 7/4 mm Hg in hypertensive individuals and 4/2 mm Hg in normotensive individuals (Fig 4).[43]

Two well-controlled trials have studied 3 salt intakes (ie, 12, 6, and 3 g/d in one trial and 8, 6, and 4 g/d in the other), each for 4 weeks.[44,45] Both showed a clear dose response, that is, the lower the salt intake achieved, the lower the BP. From the dose-response relationship, it is clear that the current recommendations to reduce salt from 9 to 12 g/d to 5 to 6 g/d will have a major effect on BP but are not ideal. A further reduction to 3 g/d will have a much greater effect. The question is whether it is feasible to reduce salt intake to 3 g/d. Clinical studies have shown that, with appropriate advice, this level of salt intake could be achieved and maintained in highly motivated individuals.[44] However, to achieve this for the entire population would be very difficult unless the food industry cooperates (see "the role of the food industry in salt reduction).

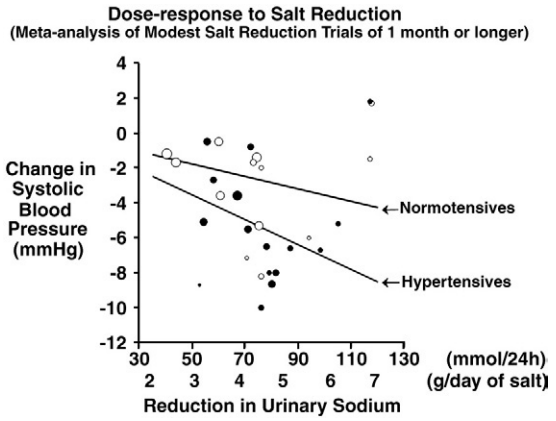

Fig 4. Relationship between the reduction in 24-hour urinary sodium and the change in blood pressure in a meta-analysis of modest salt reduction trials. The open circles represent normotensive subjects and the solid circles represent hypertensive subjects. The slope is weighted by the inverse of the variance of the net change in BP. The size of the circle is in proportion to the weight of the trial.

Salt and BP in specific groups

Infants

A meta-analysis of 3 controlled trials with 551 infants showed that a 54% reduction in salt intake for an average duration of 20 weeks reduced systolic BP by 2 mm Hg ($P <$ .01). Among the 3 trials included in the meta-analysis, 2 were carried out in the early 1970s and 1980s, and at that time, salt concentrations in formula milk were approximately 3 times higher than in human milk.[46] Currently, in most developed countries, salt is no longer added to formula milk or baby foods, and salt concentrations in formula milk are very similar to those in human milk. However, salt intake in infants and toddlers is dramatically increased when table foods are introduced, which usually begins at about 6 to 9 months of age. The introduction of cow's milk at about 12 months increases salt intake further. A recent study in the United States showed that almost all 12- to 24-month-old toddlers had salt intake above the "adequate level" of the Dietary Reference Intake established by the Food and Nutrition Board of the Institute of Medicine, and the mean salt intake was 4.1 g/d.[47] This is obviously far too high, and a reduction in salt intake will be beneficial for those toddlers' BP.

There is evidence suggesting that salt intake in early life may have a long-lasting effect on BP. A double-blind trial carried out in 476 Dutch newborn babies in the 1980s showed that, when salt intake was reduced by approximately

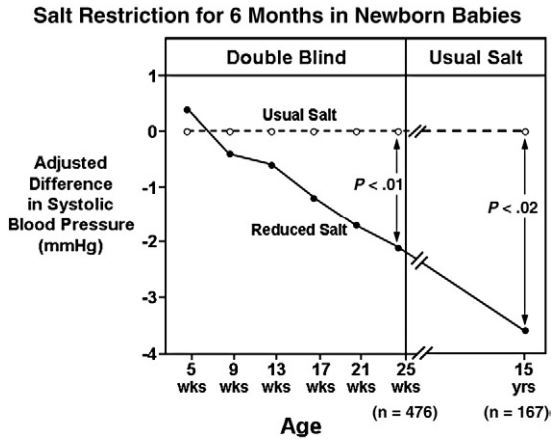

Fig 5. Difference in systolic BP in newborn babies, randomized to either a usual salt intake or a reduced salt intake over the first 6 months of life. At 6 months, the study was discontinued, with all participants resuming their usual salt intake. Fifteen years later, a subgroup had BP remeasured. Adapted from Hofman et al[46] and Geleijnse et al.[48]

30%, there was a progressive difference in systolic BP between babies in the reduced salt and those in the usual salt group during the 6 months study (Fig 5).[46] The study was discontinued at 6 months, and all infants went back to their usual salt intake that was generally high because low salt baby foods were not commercially available in the Netherlands at that time. Fifteen years later, 35% of these babies were followed up.[48] There remained a significant difference in systolic BP, when adjusted for potential confounding factors, between those babies who in the first 6 months of life had had a reduced salt intake compared to those who had not, despite a similar urinary sodium and potassium excretion between the 2 groups during follow-up. These results are consistent with those from animal experiments.

Children and adolescents

There have been more than 20 observational epidemiological studies on salt and BP in children and adolescents.[49] Many of these studies did not show a significant association. This is not surprising given the large day-to-day intraindividual variations of salt intake. In addition, many studies had methodological problems, for example, the methods used to assess salt intake were unreliable. Among the observational studies that were methodologically stronger (eg, multiple measurements of salt intake were made, urinary sodium was measured, and confounding factors were controlled for), most showed a significant positive association between salt intake and BP.[49,50] For instance, in a carefully conducted study where 7 consecutive 24-hour urine samples were collected, Cooper et al[51] demonstrated a significant linear relationship between urinary sodium and systolic BP in 73 children aged 11 to 14 years. The relationship remained significant after controlling for age, sex, race, pulse rate, height, and body weight. A recent meta-analysis of 10 salt reduction trials with 966 participants demonstrated that a modest reduction in salt intake had a significant effect on BP in children and adolescents.[52] A 42% reduction in salt intake for an average duration of 4 weeks reduced systolic BP by 1.2 mm Hg ($P < .001$) and diastolic by 1.3 mm Hg ($P < .001$).

It has been shown that BP patterns in children reflect those in adulthood, that is, the higher the BP during childhood, the higher the BP in adulthood.[53] A lower salt diet, starting from childhood, may well lessen the subsequent rise in BP with age, which would have major public health implications in preventing the development of hypertension and CVD later in life.

Current salt intake in children is very high. Even in 1984, a study in the United Kingdom where 2 consecutive 24-hour urine samples were collected in 34 school children aged 4 to 5 years showed that the average sodium excretion was 4 g of salt per day.[54] If this is expressed for adults on a weight basis, it is equivalent to approximately 15 to 20 g/d. This was at a time when consumption of processed foods by children was not high. Since then, salt intake in children in developed countries has increased due to the increasing consumption of processed foods that now account for approximately 80% of total salt intake. Surveys in the United States showed that the proportion of foods that children consumed from restaurants and fast-food outlets increased by nearly 300% between 1977 and 1996,[55] and it is very likely to have increased even further in more recent years. Snack food consumption showed a similar trend. The processed, restaurant, fast foods, and snacks are generally very high in salt, fat, and sugar. It is possible that children from the age of 3 to 4 onward now consume as much salt as adults.

Older people

The fall in BP with a reduction in salt intake has been shown to be related to age, that is, the older the individual, the greater that fall in BP with salt reduction.[56] A double-blind study in individuals aged 60 to 78 years demonstrated a large reduction in BP (7.2/3.2 mm Hg in hypertensive individuals and 8.3/2.9 mm Hg in normotensive individuals) when salt intake was reduced from 10 to 5 g/d.[57] A meta-analysis of all salt reduction trials in older people including 5 trials with participants 60 years or older and 6 with a mean age close to 60 years confirmed these findings.[58] In view of the fast-growing proportion of elderly in the population, the very high incidence of stroke and heart failure, and the great benefits of BP-lowering in older people, a reduction in salt intake in this group is therefore particularly important in reducing the BP-related disease burden.

Variations in BP response to salt reduction

Randomized trials have shown that, for a given reduction in salt intake, the falls in BP were larger in

individuals of African origin, in older people, and in those with raised BP compared to whites, young people, and those with normal BP, respectively.[59] The greater decreases in BP in these individuals were, at least in part, due to the diminished responsiveness of their renin-angiotensin system.[56,60]

The term "salt sensitivity" has been commonly used to describe the variations of BP response to salt reduction. However, almost all of the studies on salt sensitivity have used a protocol of very large and sudden changes in salt intake. As described previously, these studies are irrelevant to the public health recommendations of more modest reduction in salt intake for a prolonged period. There is strong evidence that a modest reduction in salt intake should be carried out universally in the entire population. A reduction in population salt intake lowers population BP. Even a small reduction of BP across the whole population would have a large impact on reducing the appalling burden of CVD.[61]

Salt and other dietary and lifestyle changes for lowering BP

There is evidence that a reduction in salt intake is additive to other dietary and lifestyle changes for lowering BP.[45,62] The DASH (Dietary Approaches to Stop Hypertension)-Sodium trial,[45] a well-controlled feeding trial, studied 3 levels of salt intake (8, 6, and 4 g/d) on 2 different diets, that is, the normal American diet and the DASH diet, which is rich in fruits, vegetables, and low-fat dairy products. The study demonstrated that a reduction in salt intake lowered BP both on the normal American diet and on the DASH diet. The combination of a low salt and the DASH diet had a greater effect on BP than either intervention alone, though the combined effects were not as great as the simple addition of each separate intervention (Fig 6).[45] The Trial of Nonpharmacologic Interventions in the Elderly demonstrated that a combination of salt reduction and weight loss were more successful than either intervention alone in maintaining satisfactory BP control after withdrawal of antihypertensive medication in older people who were obese and had hypertension.[62] The Trial of Hypertension Prevention (TOHP) II also showed a greater effect of a combined salt and weight reduction on the incidence of hypertension during the first 6 months of the study in overweight people with high normal BP. However, this effect did not sustain over the following 30 months due to the failure of maintaining lower salt and weight.[63]

Salt and antihypertensive treatments

Randomized trials have demonstrated that a reduction in salt intake causes further decreases in BP in individuals who are already on antihypertensive drug treatments. It also enhances BP control and reduces the need for antihypertensive drug therapy.[62,64] Salt restriction is particularly effective in lowering BP when the renin-angiotensin system is blocked by an angiotensin-converting enzyme inhibitor or angiotensin receptor blocker because the reactive increase in plasma renin activity and thereby angiotensin II that occurs with salt reduction offsets the falls in BP. A randomized double-blind trial showed that a reduction of 5.8 g/d in salt intake reduced BP by 13/9 mm Hg in hypertensive individuals who were already on captopril.[64] These falls in BP were greater than those observed in similar salt reduction trials in hypertensive individuals who were not on any treatments.[36,44] The Trial of Nonpharmacologic Interventions in the Elderly study demonstrated that, in elderly hypertensive individuals after withdrawal of medication, a very modest reduction in salt intake of only 2.4 g/d reduced the occurrence of high BP, resumption of medication, and cardiovascular events by 32% ($P < .001$) during 28-month follow-up.[65]

A high salt intake contributes to resistant hypertension. Recently, Pimenta et al[66] studied 12 patients whose average BP was 146/84 mm Hg while taking 3 or more

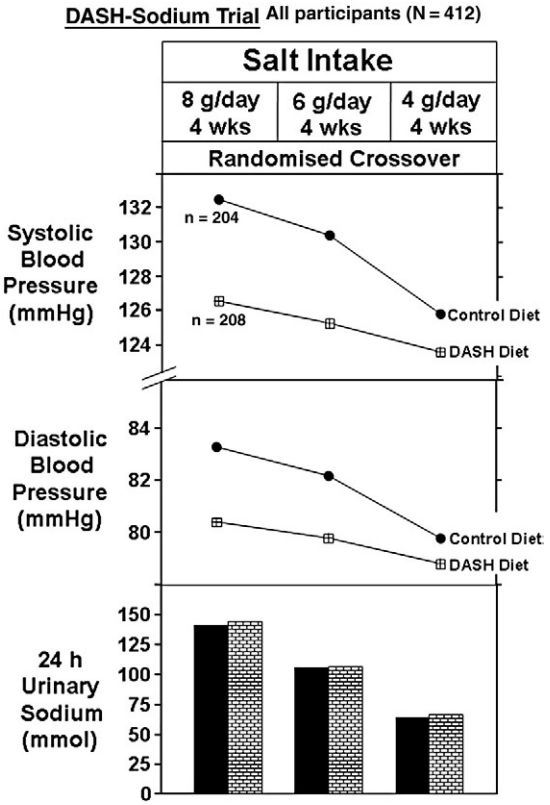

Fig 6. Changes in BP and 24-hour urinary sodium excretion with the reduction in salt intake in all participants (hypertensive subjects: n = 169; normotensive subjects: n = 243) on the normal American diet (ie, control diet) and on DASH diet. Redrawn from Sacks et al.[45]

different types of antihypertensive drugs in a randomized crossover trial and showed that, when salt intake was reduced from 14.8 to 2.7 g/d for 1 week, there were large decreases in office BP of 23/9 mm Hg. The reductions in 24-hour ambulatory and daytime and nighttime BPs were almost identical to the decreases in office BP.[66] Clearly, replication of this study, particularly with a larger sample size and longer duration, is warranted.

Mechanisms by which salt raises BP

Role of the kidneys

The mechanisms whereby salt raises BP are not fully understood. However, there is much evidence that individuals who develop high BP have an underlying defect in the kidneys' ability to excrete sodium. The kidney cross-transplantation experiments clearly demonstrated the important role of the kidneys in BP regulation.[67,68] When a kidney from a normotensive rat was inserted into a young bilaterally nephrectomized spontaneously hypertensive rat, the BP of the hypertensive rat did not rise, and conversely, when a kidney from a young hypertensive rat (before it developed hypertension) was inserted into a bilaterally nephrectomized normotensive rat, the BP of the normotensive rat rose. Similarly, the high BP of patients with essential hypertension who developed kidney failure became normal (over a mean follow-up of 4.5 years) after bilateral nephrectomy and transplantation with a kidney from a normotensive donor.[69] These findings clearly indicate that whatever functional abnormalities may occur at other sites, the primary disturbance that initiates the rise in BP resides in the kidneys.

Role of extracellular volume

The traditional concepts regarding the mechanisms for salt-induced high BP are that the impaired ability of the kidneys to excrete sodium causes sodium and water retention, particularly on a high salt intake, leading to volume expansion and the stimulation of various compensatory mechanisms. The persistent presence of some of the compensatory mechanisms eventually causes BP to rise that in turn helps overcome the kidneys' difficulties in excreting sodium. On the basis of experiments in 70% nephrectomized dogs given large amounts of saline intravenously daily for 2 weeks, Guyton[70] suggested that volume expansion raises BP by the autoregulatory effect on resistance vessels.

Direct role of plasma sodium

There is now increasing evidence that small changes in plasma sodium may be an important mechanism for the changes in BP with changing salt intake. A number of studies have shown that an increase or decrease in salt intake causes parallel changes in plasma sodium in both hypertensive and normotensive individuals, for example, a decrease of approximately 3 mmol/L ($P < .001$) in plasma sodium when salt intake was reduced from 20 to 1 g/d for 5 days. In a well-controlled double-blind trial of 1 month, plasma sodium was reduced by 0.4 mmol/L ($P < .05$) when salt intake was decreased from approximately 10 to 5 g/d in 118 hypertensive individuals. The decrease in plasma sodium was weakly but significantly correlated with the fall in systolic BP.[71]

Several epidemiological studies have shown a significant positive association between plasma sodium and blood pressure.[72-74] In a study of 3578 London civil servants, a 1 mmol/L increase in plasma sodium was associated with a 1 mm Hg increase in systolic BP after adjusting for confounding factors.[72] In another study of a Japanese population (3222 normotensive subjects and 741 patients with essential hypertension), serum sodium distribution was shifted by approximately 2 mmol/L toward higher values in the hypertensive subjects.[73] However, the Framingham Heart Study showed that serum sodium was not associated with BP cross-sectionally or with the development of hypertension during 4 years of follow-up.[75]

Plasma sodium is a major determinant of extracellular volume, thereby, influencing BP. At the same time, small changes in plasma sodium may have a direct effect on BP, independent of extracellular volume.[76] Using peritoneal dialysis in rats, Friedman et al[77] were able to change plasma sodium in an opposite direction to extracellular volume by altering sodium concentration of the dialysis fluid. When plasma sodium was increased by 10 to 15 mmol/L, there was a rapid increase in BP despite a reduction in extracellular volume. When plasma sodium was decreased, there was a fall in BP despite an increase in extracellular volume. The changes in BP were directly related to the changes of intracellular sodium. Friedman et al[77,78] suggested that increases in intracellular sodium may affect vascular smooth muscle tension and thereby BP. There is also evidence to suggest that small changes in plasma sodium may directly affect the hypothalamus' control of BP through the local renin-angiotensin system.[76]

Tissue culture experiments demonstrated that increasing bath sodium concentration within the physiologic range caused marked cellular hypertrophy in both arterial smooth muscle and cardiac myocytes.[79] In cultured bovine endothelial cells, when bath sodium concentration was increased from 137 to 142 mmol/L, endothelial nitric oxide synthase (eNOS) activity was reduced by 25%. The decrease in eNOS activity was in a sodium concentration-dependent manner within the range studied (137-157 mmol/L) (Fig 7).[80] Using cultured human endothelial cells, Oberleithner et al[81] demonstrated that an increase in the sodium concentration of the culture medium from 135

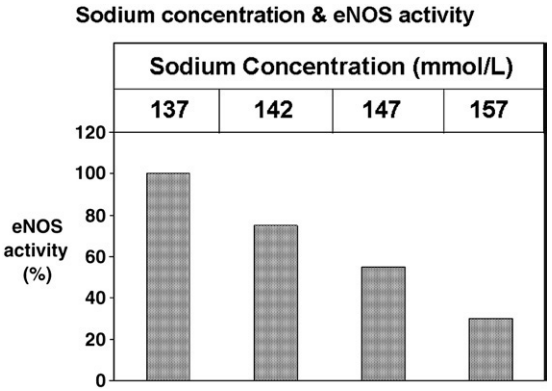

Fig 7. Effects of sodium concentration in the culture medium on eNOS activity in cultured bovine aortic endothelial cells. Adapted from Li et al.[80]

to 145 mmol/L stiffened endothelium and reduced nitric oxide release.

Recent studies in humans have shown that salt intake does affect endothelial function. A randomized trial in 29 overweight and obese normotensive individuals showed that, when salt intake was reduced from 9.2 to 3.8 g/d for 2 weeks, there was a significant improvement in endothelial function as measured by brachial artery flow-mediated dilatation.[82] A study in healthy volunteers showed that an increase in salt intake was associated with a blunted endothelial response to acetylcholine.[83]

Salt and CVD

A reduction in salt intake lowers BP, and as raised BP throughout its range is a major risk factor for CVD, this would be predicted to reduce CVD. Based on the falls in BP from a meta-analysis of randomized salt reduction trials,[43] it was estimated that a reduction of 6 g/d in salt intake would reduce stroke by 24% and coronary heart disease (CHD) by 18%. This would prevent approximately 35,000 stroke and CHD deaths a year in the United Kingdom[84] and approximately 2.5 million deaths worldwide.

In addition to its effects on BP, a reduction in salt intake may have beneficial effects on the cardiovascular system independent of BP,[85] for example, a direct effect on stroke,[9] left ventricular hypertrophy.[11] Therefore, the total effect of salt reduction on cardiovascular outcomes may be larger than those estimated from BP falls alone.

Population studies

In the late 1950s, deaths from stroke in Japan were among the highest in the world, and salt intake was also very high. The number of stroke in different parts of Japan was directly related to the amount of salt consumed. The Japanese Government initiated a campaign to reduce salt intake. Over the following decade, the national salt intake was reduced from an average of 13.5 to 12.1 g/d, and in the north, salt intake fell from 18 to 14 g/d. Paralleling this reduction in salt intake, there were falls in BP both in adults and children and an 80% reduction in stroke mortality[86] despite large increases in fat intake, cigarette smoking, alcohol consumption, and obesity. It would appear that the Western influence that was rapidly overtaking Japan at that time had little effect on BP, provided salt intake was reduced, and overall the reduction in salt intake appeared to be associated with the falls in deaths from stroke.

Since the 1970s, Finland has aimed to reduce salt intake in the entire population.[87,88] This has been carried out in part through collaboration with the food industry to develop reduced salt food products and raising the general awareness among consumers of the harmful effects of salt on health. Over the following 30 years, salt intake was reduced by one third. This was accompanied by a fall of more than 10 mm Hg in both systolic and diastolic BP, a pronounced decrease of 75% to 80% in both stroke and CHD mortality, and a remarkable increase of 5 to 6 years in life expectancy.[87] The reduction in salt intake was a major contributory factor for these results, particularly, the fall in BP as both body mass index and alcohol consumption had increased during that period. An increase in potassium intake via the use of reduced sodium, potassium- and magnesium-enriched salt, an increased consumption of fruit and vegetables, a reduction in fat intake, and a decrease in smoking rate in men also played a part in the fall in CVD.

Prospective cohort studies

Twelve prospective cohort studies have looked at the relationship between salt intake and cardiovascular outcomes.[5,10,89-99] Among these studies, 7 used dietary assessment methods, 1 used overnight urinary sodium, and 4 used 24-hour urinary sodium to estimate dietary salt intake. The dietary methods, for example, 24-hour dietary recall, are unreliable in estimating a person's salt consumption, particularly as no account is taken of discretionary salt. Many of these prospective studies had baseline salt intake measured in the 1970s, a time when discretionary salt would have contributed substantially to salt intake. These dietary assessment methods have been criticized previously. For instance, Karppanen and Mervalaala[100] pointed out that, in the National Health and Nutrition Examination Survey follow-up study, many women in the lowest quartile of salt intake who had an energy intake near starvation level had survived for 20 years and they actually weighed 4 kg more than those in the highest quartile of salt intake who apparently also had a much higher energy intake. Owing to the methodological flaws, the results from these studies should be interpreted with great caution.

Twenty-four-hour urinary sodium is the most accurate method to measure salt intake. Among the 12 prospective studies, 4 had 24-hour urinary sodium measured. However,

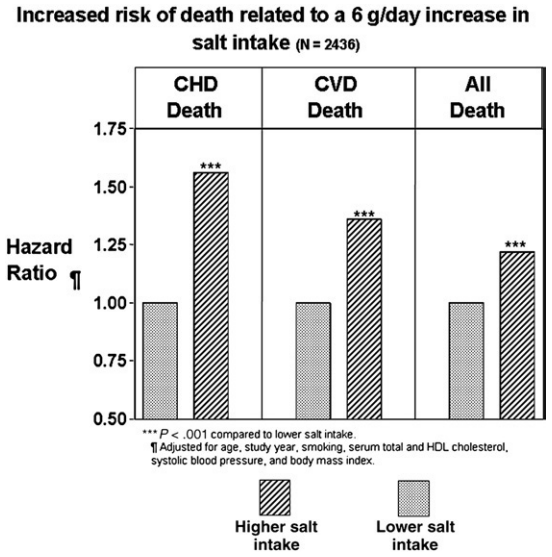

Fig 8. The hazards ratios for CHD, CVD, and all-cause mortality associated with a 6 g/d increase in salt intake as judged by 24-hour urinary sodium excretion. Adapted from Tuomilehto et al.[5]

in the NY Worksite Study,[90] 24-hour urinary sodium was measured after all hypertensive individuals had their salt intake restricted for 5 days, and no measurement was made on the participants' usual diet. Furthermore, 24-hour urinary analysis revealed severe methodological problems as individuals in the lowest quartile of salt intake had a much lower urinary creatinine level[101] indicating incomplete collection of 24-hour urine. The results from this study therefore cannot be used to look at the effects of salt reduction.

The Scottish Heart Health Study[92] that enrolled a random sample of 11,629 individuals aged 40 to 59 years had 24-hour urinary sodium measured while on individuals' usual diet. The follow-up data showed that a higher salt intake was associated with a higher risk of coronary events in women, but the association was not significant in men. Another prospective cohort study[5] that measured 24-hour urinary sodium on usual salt intake in a random sample of 2436 Finnish men and women aged 25 to 64 years showed that an increase of 6 g/d in salt intake was related to an increase of 56% in CHD deaths, 36% in CVD deaths, and 22% in all deaths (Fig 8).[5] A recent follow-up study of individuals who were allocated to the control group of TOHP I and II showed that baseline urinary sodium-to-potassium ratio was significantly associated with CVD risk during 10 to 15 years of follow-up.[99]

Outcome trials

Cook et al[4] reported the long-term effects of salt reduction on CVD in individuals participating in 2 large randomized trials, TOHP I and II. More than 3000 participants with an average baseline blood pressure of 127/85 mm Hg were randomized to a reduced salt group (for 18 months in TOHP I and 36-48 months in TOHP II) or to a control group. Compared with the control group, individuals in the intervention group reduced their salt intake by 25% to 30% from an average of approximately 10 g/d. This resulted in a fall in BP of 1.7/0.9 mm Hg at 18

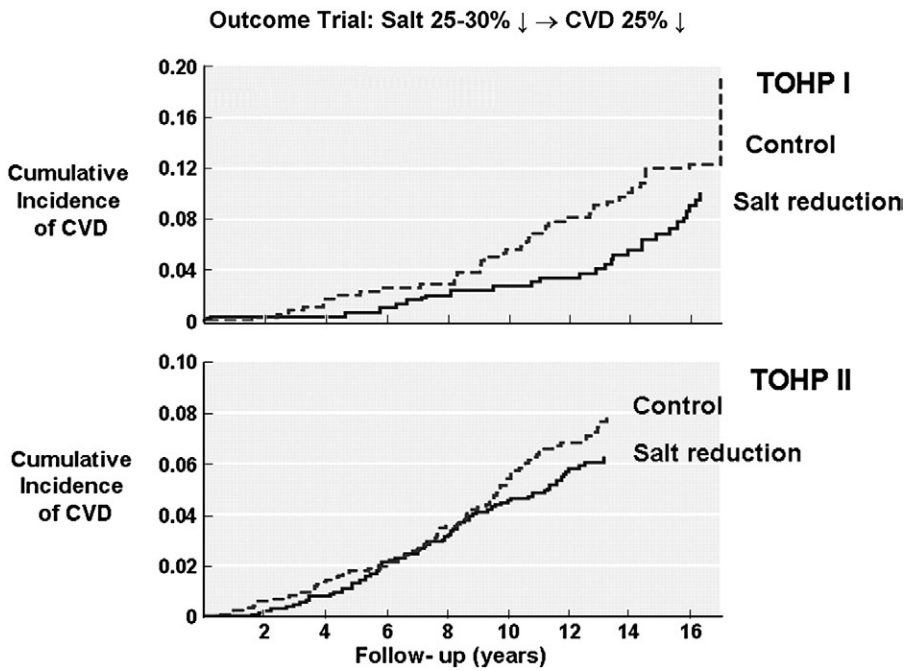

Fig 9. Cumulative incidence of CVD by salt intervention group in the TOHP I and II, adjusted for age, sex, and clinic. Adapted from Cook et al.[4]

months (TOHP I) and 1.2/0.7 mm Hg at 36 months (TOHP II). After the original trials were completed, participants were not given further dietary advice. A follow-up study at 10 to 15 years posttrial showed that individuals who were originally allocated to the reduced salt group had a 25% lower incidence of cardiovascular events after adjusting for confounding factors (Fig 9).[4] Another outcome trial of more than 2.5 years in elderly Taiwanese veterans (n = 1981) showed that switching from the usual salt to potassium-enriched salt (49% sodium chloride, 49% potassium chloride, 2% other additives) with a subsequent reduction of 17% in salt intake and an increase of 76% in potassium intake as measured by urinary sodium/creatinine ratio and potassium/creatinine ratio, resulted in a 40% decrease in CVD mortality.[102]

Other harmful effects of salt

There is increasing evidence that salt has other deleterious effects on health, independent of and sometimes additive to its effect on BP.

Salt and water retention

When humans go from a low to a high salt intake, there is retention of salt and, thereby, water, and this expands the extracellular volume. The increase in extracellular volume is a trigger for various compensatory mechanisms to allow an increase in urinary sodium excretion but at the expense of continued retention of salt and water. Approximately 1.5 L of extracellular fluid is retained, and this continues as long as a higher salt intake is consumed. The increase in extracellular fluid exacerbates all forms of salt and water retention, for example, heart failure[103] and is a major cause of edema in women, aggravating both cyclical and idiopathic edema.[104]

Direct effect on stroke

Experimental studies in animals[105] and epidemiological studies in humans[9,10,106] demonstrated that salt intake was directly related to the risk of stroke, independent of BP. In an ecological analysis, Perry and Beevers[9] found a significant positive correlation between 24-hour urinary sodium and stroke mortality (Fig 10), and this relationship was stronger than that found when urinary sodium was plotted against BP. A study from Japan confirmed a close relationship between salt intake and stroke mortality within a single country, and the relationship was independent of BP and other confounding factors.[10]

Direct effect on left ventricular mass

Left ventricular hypertrophy is an important independent predictor of cardiovascular morbidity and mortality.[107] Several cross-sectional studies showed a positive correlation between 24-hour urinary sodium and left ventricular mass, independent of BP (Fig 11).[11,108,109] A reduction in salt intake has been shown to decrease left ventricular mass in hypertensive individuals.[110-112]

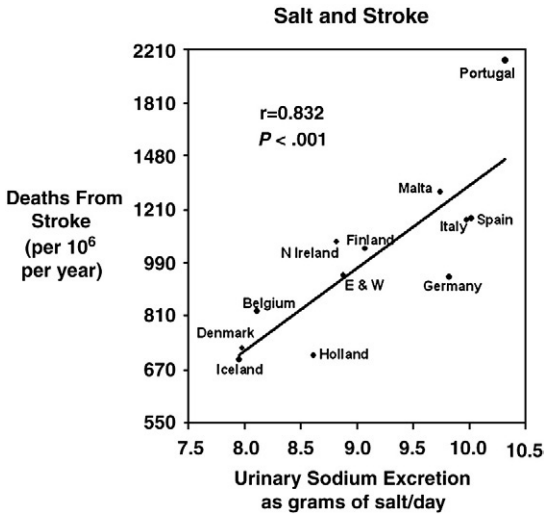

Fig 10. Relationship between salt intake and deaths from stroke in 12 European countries. Adapted from Perry and Beevers.[9]

Stomach cancer

An ecological analysis showed a significant direct association between salt intake (as measured by 24-hour urinary sodium) and deaths from stomach cancer among 39 populations from 24 countries (Fig 12).[113] A study in a Japanese population confirmed this finding.[16] Several studies have shown that chronic *Helicobacter pylori* infection, which causes both duodenal and gastric ulcers and stomach cancer, was also closely associated with salt intake.[114-116] Foods that contain high concentrations of salt are irritating to the delicate lining of the stomach. It is

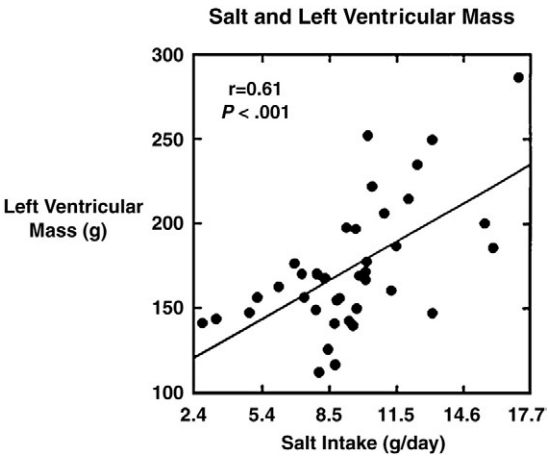

Fig 11. Relationship between salt intake and left ventricular mass in individuals with systolic BP > 121 mm Hg. Adapted from Kupari et al.[11]

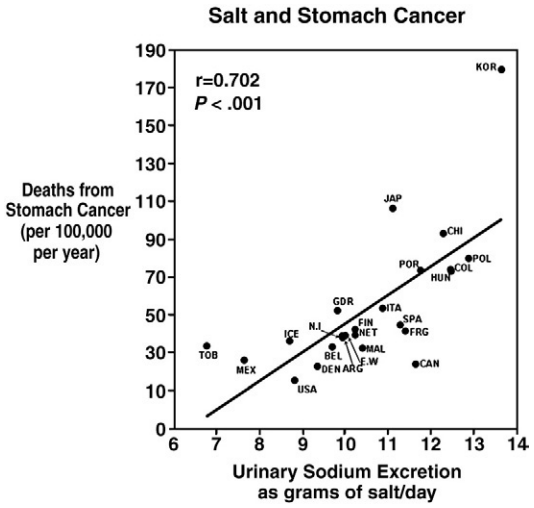

Fig 12. Relationship between salt intake and deaths from stomach cancer. Adapted from Joossens et al.[113]

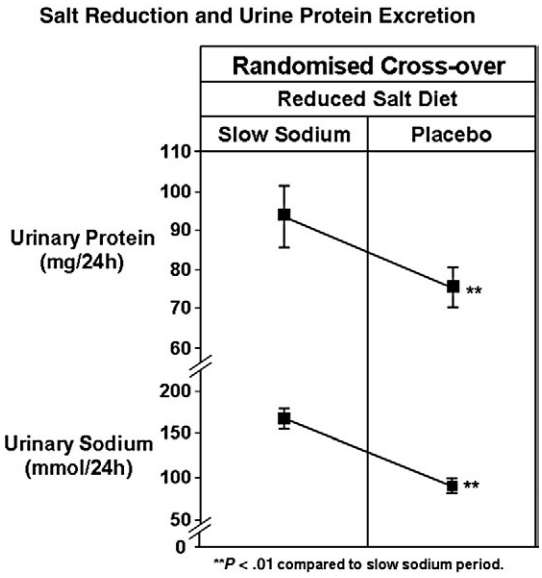

Fig 13. Change in urinary sodium and protein excretion with a modest reduction in salt intake from approximately 10 to 5 g/d in 40 hypertensive blacks.

possible that this makes *H. pylori* infection more likely or more severe and that the *H. pylori* infection then leads to stomach cancer. A reduction in salt intake may reduce *H. pylori* infection and therefore prevent stomach cancer.

Kidney disease

Urinary albumin excretion has been shown to be an important and independent risk factor for the development and progression of renal disease and also for cardiovascular disease in individuals with diabetes, chronic kidney disease, hypertension, and the general population.[117] Importantly, the risk increases throughout the range of albumin excretion, and there is no threshold. Several epidemiological studies have shown a direct association between salt intake and urinary albumin excretion, independent of BP.[118,119] A randomized double-blind trial in 40 black hypertensive individuals demonstrated that a reduction in salt intake from approximately 10 to 5 g/d reduced 24-hour urinary protein by 19% ($P < .01$) (Fig 13).[8] A more recent double-blind trial in a larger number of individuals including 71 whites, 69 blacks, and 29 Asians with mildly raised BP demonstrated that even a smaller reduction in salt intake, that is, from an average of 9.7 to 6.5 g/d reduced 24-hour urinary albumin excretion significantly in all 3 ethnic groups.[120] Other studies in patients with proteinuria or diabetes showed that the antiproteinuric effects of an angiotensin-converting enzyme inhibitor or angiotensin receptor blocker were abolished by increasing salt intake.[7,121] A retrospective analysis of 57 chronic kidney disease patients with an average observation period of 3 years showed that a lower salt intake reduced proteinuria and slowed down the progression of renal disease despite a similar BP control between the 2 groups on a high and low salt intake.[6]

In patients who are on dialysis, a reduction in salt intake reduces the amount of fluid that they drink between dialyses. This particularly applies to hemodialysis patients, in whom BP is a major problem. A lower salt intake decreases weight gain between dialyses, and BP is easier to control.

Renal stones and osteoporosis

Salt intake is one of the major dietary determinants of urinary calcium excretion. Both epidemiological studies and randomized trials showed that a decrease in salt intake reduced urinary calcium excretion.[14,57,122,123] As calcium is the main component of most urinary stones, salt intake is therefore an important cause of renal stones. Until recently, it was assumed that when salt intake was increased, the increase in calcium excretion was compensated for by an increase in intestinal calcium absorption. There is now evidence to suggest that when salt intake is increased, there is a negative calcium balance with stimulation of mechanisms not only to increase intestinal absorption of calcium, but also to mobilize calcium from bone. A 2-year longitudinal study in postmenopausal women showed that the loss of hip bone density was related to 24-hour urinary sodium at entry to the study and was as strong as that relating to calcium intake.[124]

Asthma

Although it is not thought that a high salt intake is a cause of asthma, epidemiological and clinical evidence suggested an association between salt intake and the severity of asthma.[15,125] This was supported by some randomized

trials.[126,127] For example, a double-blind study in 22 mild to moderate asthmatic men demonstrated that a decrease in salt intake reduced the severity of asthma attacks, the use of medication, and airway resistance.[126] However, a more recent double-blind trial showed that a lower salt intake as an adjunctive therapy to normal treatment had no additional therapeutic benefit in adults with asthma.[128] A recent population-based study in children aged 6 to 7 years demonstrated that adding salt to foods was strongly and independently associated with an increased risk of respiratory symptoms, that is, wheeze and asthma.[129]

Obesity

A high salt intake has been suggested as an indirect cause of obesity, through the effect it has on soft drink consumption. A carefully controlled metabolic study in adult humans showed that a reduction in salt intake caused a significant decrease in fluid consumption.[12] It was estimated that reducing salt from the current intake of approximately 10 g/d to the World Health Organization (WHO) recommended level of 5 g/d would reduce fluid consumption by approximately 350 mL/d. A study in 10,074 free living individuals across the world showed an almost identical relationship between usual salt and fluid intake.[12] As a considerable proportion of fluid intake is in the form of soft drinks and soft drink consumption is associated with obesity,[130] a reduction in salt intake could therefore play a role in helping to reduce obesity. Karppanen and Mervaala[87] analyzed the data on the sales of salt and carbonated beverages in the United States between 1985 and 2005 and showed a close link between the two. They were also in parallel with the trend of obesity prevalence.

A recent analysis of the dataset of the National Diet and Nutrition Survey for young people (aged 4-18 years) in Great Britain showed that, after adjusting for confounding factors, salt intake was significantly associated with total fluid intake and also with sugar-sweetened soft drink consumption.[13] A difference of 1 g/d in salt intake was associated with a difference of 100 and 27 g/d ($P < .001$) in total fluid and sugar-sweetened soft drink consumption, respectively. These results, in conjunction with the evidence from experimental studies where only salt intake was changed,[12] demonstrate that salt intake is an important determinant of total fluid and sugar-sweetened soft drink consumption in children. It has been shown that soft drink consumption is related to childhood obesity.[131,132] A reduction in salt intake could therefore play a part in helping to reverse the increasing trend of childhood obesity worldwide.

Salt and the renin-angiotensin system, the sympathetic nervous system, lipids, and insulin sensitivity

When salt intake is reduced, there is a physiologic stimulation of the renin-angiotensin system and the sympathetic nervous system. These compensatory responses are bigger with sudden and large decreases in salt intake and much smaller or minimal with a modest reduction in salt intake for a more prolonged period, which is the current public health recommendation on population salt intake. Randomized trials have demonstrated that, with a longer term modest reduction in salt intake, there was only a small increase in plasma renin activity[43] and no detectable change in the sympathetic nervous activity.[133]

Salt reduction lowers BP in a similar mechanism to that of thiazide diuretics. Both stimulate the renin-angiotensin system and, in the short-term, the sympathetic nervous system. However, outcome trials have demonstrated that long-term treatment with thiazide diuretics significantly reduced cardiovascular morbidity and mortality in hypertensive individuals.[134]

An acute and large reduction in salt intake causes a reduction in plasma volume and, thereby, a small increase in the concentration of plasma lipids. However, randomized trials of longer term modest salt reduction showed no significant changes in total cholesterol, triglyceride, or low- or high-density lipoprotein cholesterol.[43]

A number of studies have looked at the effects of changing salt intake on glucose tolerance and insulin sensitivity.[135,136] However, most of these studies involved a very large change in salt intake for only a few days. Randomized trials showed that a longer term modest reduction in salt intake had no significant effect on glucose tolerance or insulin sensitivity in hypertensive individuals.[137] A prospective study in 932 Finnish men and 1003 women with an average follow-up of 18 years demonstrated that a higher salt intake (measured by 24-hour urinary sodium) was associated with an increased risk of type 2 diabetes, independent of potential confounding factors including physical activity, obesity, and hypertension.[138]

Long-term treatment with thiazide diuretics may increase the risk of diabetes,[139] which is likely to be due to a lower level of serum potassium that occurred.[139,140] Concomitant treatment with potassium supplementation or potassium-sparing diuretics could lessen the glucose intolerance and possibly prevent the development of thiazide-induced diabetes.[139] The advantage of modest salt reduction over thiazide diuretics is that salt reduction does not have a significant effect on serum potassium but has a similar BP-lowering effect in hypertensive individuals as demonstrated by randomized trials.[141]

Cost-effectiveness of reducing population salt intake

Several studies have demonstrated that a reduction in population salt intake is very cost-effective.[142-145] For example, Murray et al[145] showed that nonpersonal health interventions, including government action to stimulate a reduction in the salt content of processed foods, were cost-

effective ways to limit CVD and could avert more than 21 million disability-adjusted life years per year worldwide. A study in Norwegian population documented that a 6 g/d reduction in salt intake with a very conservative estimate of 2 mm Hg fall in systolic BP could save costs to individuals and society by US$4.7 million per year.[142] It is very likely that this has considerably underestimated the true cost savings as randomized trials have shown that the fall in systolic BP with a 6 g/d decrease in salt intake is much greater than that projected in this study.[84] A study in Canada estimated that a reduction of 4.6 g/d in salt intake could save approximately $430 million per year from drugs, physician visits, and laboratory testing directly related to hypertension.[143]

In a more recent study, Asaria et al[144] estimated the effects and cost of strategies to reduce salt intake and control tobacco use for 23 low- and middle-income countries that account for 80% of chronic disease burden in the developing world. They demonstrated that, for 10 years (from 2006 to 2015), a 15% reduction in mean population salt intake could avert 8.5 million CVD deaths and a 20% reduction in smoking prevalence could avert 3.1 million CVD deaths. The modest reduction in salt intake could be achieved by a voluntary reduction in the salt content of processed foods and condiments by manufacturers, plus a sustained mass-media campaign aimed to encourage dietary change within households and communities. The cost for implementing such salt reduction programs was estimated to be US$0.09 per person per year. The cost for tobacco control including both price and nonprice measures was US$0.26 per person per year (Fig 14).[144] These figures clearly suggest that a reduction in salt intake is more or at the very least just as cost-effective as tobacco control in reducing CVD on its own, the leading cause of death and disability worldwide.

Worldwide salt reduction programs

Many countries have developed their own guidelines on dietary salt intake. The United Kingdom and US guidelines recommend salt intake of less than 6 g/d for adults.[146,147] The WHO set a worldwide target of a maximum intake of 5 g/d.[148] Through its regional directorates, the WHO is starting salt reduction strategies.[149] Eleven countries in the European Union have signed up to make a 16% reduction in salt intake over the next 4 years.[150] Several countries, for example, Finland, the United Kingdom, have already successfully carried out salt reduction programs.

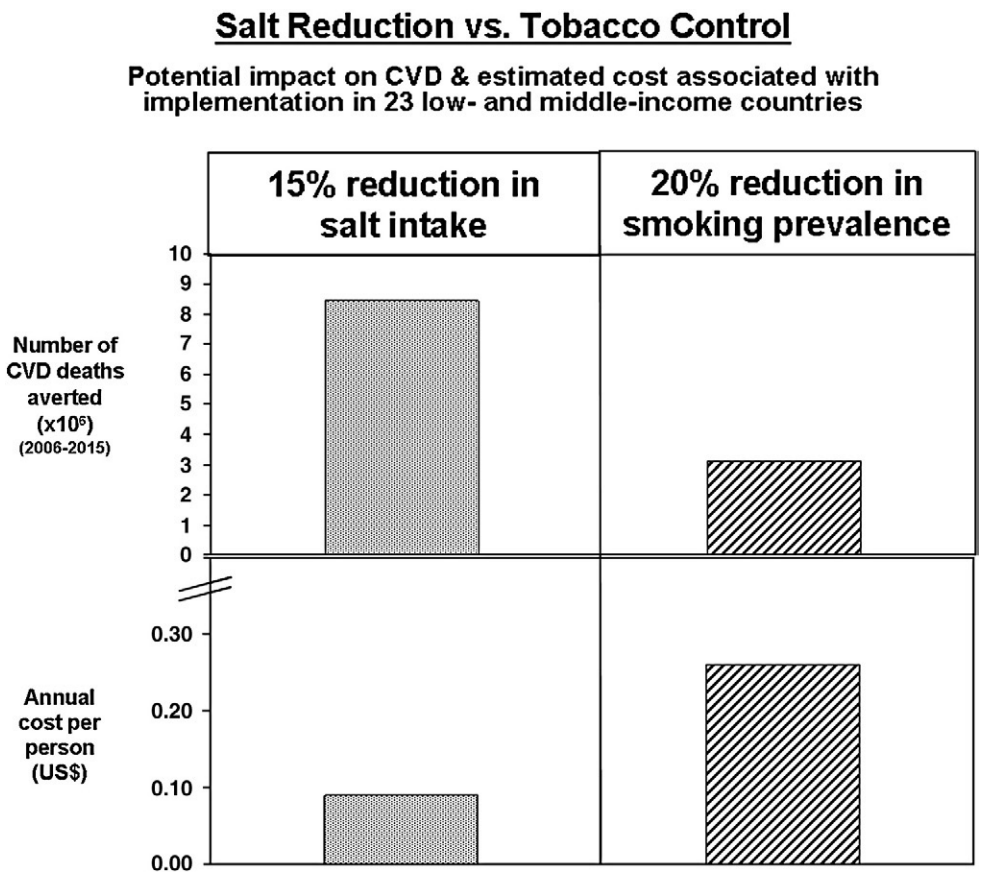

Fig 14. Number of CVD deaths averted and the financial costs associated with implementation of salt reduction and tobacco control in 23 low- and middle-income countries. Adapted from Asaria et al.[144]

The United Kingdom strategy—a model for other countries

The United Kingdom is one of the countries leading the way and setting an example for other countries in salt reduction strategies. In 1996, 22 experts on salt and BP set up an action group—Consensus Action on Salt and Health following the government's rejection of the recommendation to reduce salt intake by the Committee on Medical Aspects of Food and Nutrition Policy in 1994.[151,152] Consensus Action on Salt and Health has waged a highly successful public heath campaign to encourage the United Kingdom food industry to reduce the amount of salt added to foods, educate the public about the dangers of excess salt, and translate evidence into public health policy. Consensus Action on Salt and Health persuaded the United Kingdom Department of Health to change its stance on salt, finally resulting in the chief medical officer endorsing the original recommendations of the Committee on Medical Aspects of Food and Nutrition Policy to reduce salt intake to less than 6 g/d in adults, and also ensured that the United Kingdom Food Standards Agency took on the task of reducing salt intake.

A strategy to reduce population salt intake was developed based on the United Kingdom's average salt intake of 9.5 g/d[153] as measured by 24-hour urinary sodium (Table 1). It was estimated that approximately 15% of the salt consumed (ie, 1.4 g) was added either at the table or during cooking, 5% was naturally present in the foods (0.5 g), and the rest, 80% (7.6 g), was added by the food industry in processed, canteen, restaurant, and takeaway foods. To reach the target of 6 g, a reduction of 3.5 g (ie, 40%) was needed. Therefore, the food industry needs to reduce the amount of salt added to foods from 7.6 to 4.6 g (40% reduction), and the public also needs to reduce the amount of salt they add to foods themselves from 1.4 to 0.9 g (40% reduction).

It was estimated that, in the United Kingdom, only approximately 15% of foods was eaten outside the home, that is, restaurant, canteen, and others, and therefore, the main target in the initial phase of salt reduction should be on foods that were bought in supermarkets. These foods, where salt was added, were split into more than 80 categories. The Food Standards Agency set target levels of salt for each food category that the food industry needed to achieve within a certain period. These targets have been revised recently to ensure that salt intake will reach the target of 6 g/d by 2012.[154] The aim was to reduce salt added to foods by small amounts, that is, 10% to 20% and repeated at 1- to 2-year intervals. Such reductions cannot be detected by human salt taste receptors[155] and cause no technical or safety issues to the food in question.

Table 1
UK strategy for reducing salt

Salt intake		Reduction	Target
Source	g/d	needed	intake (g/d)
Table/cooking (15%)	1.4 g	40% reduction	0.9 g
Natural (5%)	0.5 g	No reduction	0.5 g
Food industry (80%)	7.6 g	40% reduction	4.6 g
Total	9.5 g	Target	6.0 g

Therefore, the food industry needs to reduce salt content of all foods where salt has been added by 40% over the next 5 years.

The United Kingdom salt reduction strategy started in 2003/2004 and salt intake has already fallen from 9.5 to 8.6 g/d by May 2008.[156] This may seem a small change, but it was on the back of an earlier increasing salt intake and it marks the beginning of a reversal of an increasing trend. Salt intake will fall further as increasing reductions in salt added to foods are made by the food industry.

Clear labeling of the salt content of food is essential. A front of pack signpost labeling system[157] has been developed, which is being implemented by many supermarkets where there is a color coding of green, amber, and red for low, medium, and high amounts of salt, fat, sugar, and calories, as well as the amount of salt per portion and per 100 g and the recommended intake for an adult for the whole day. This type of label is much preferred by consumers to others as they can see at a glance whether a product has a little or a lot of salt. It has already been shown to have a dramatic effect on the purchase of foods, particularly when they are in the red category.

Finland

Finland was one of the first countries to initiate a systematic approach to decrease salt intake in the population, in the late 1970s, through mass-media campaigns, cooperation with the food industry, and implementing salt labeling legislation.[87,158] Since the 1980s, many food companies have reduced the sodium content of their food products by replacing conventional table salt with a sodium-reduced, potassium-, and magnesium-enriched mineral salt known as Pansalt. In the early 1990s, the Ministry of Trade and Industry and the Ministry of Social Affairs and Health set salt labeling legislation for all food categories that made a substantial contribution to the salt intake of the Finnish population. Foods that are high in salt are required to carry a "high salt content" warning, and if a food product contains a low level of salt, the product is allowed to display a low-salt label. These different measures have resulted in a significant reduction in salt intake of the Finnish population, from an average of approximately 12 g/d in 1979 to less than 9 g/d in 2002.[88]

Other countries

Following the success of the United Kingdom campaign group—Consensus Action on Salt and Health, a global action group named World Action on Salt and Health (WASH) was established in 2005 to encourage

actions on salt reduction worldwide.[159] WASH works to reduce salt in the diet worldwide by exerting pressure on multinational food companies to reduce the salt content of their products. At the moment, there is a very large variation in the amount of salt added to the same branded products in different countries or regions of the world. This variation is entirely random. This illustrates once again how easy it would be for the food industry to reduce the amount of salt they add to foods, particularly as they could do this straightaway to their branded products.

WASH is supported by 375 members from 80 countries. WASH members in each country are encouraged to set up their own country division of WASH to work together on a localized level to lower salt intake specifically in their own population. For example, in 2007, an Australian Division of World Action on Salt and Health was established. They have launched a national campaign to lower salt intake of the Australian population to 6 g/d by 2012. In Canada, the Health Check program of the Heart and Stroke Foundation works with the food industry to reduce salt in processed foods.[160] Seventeen health organizations have recently endorsed a national collaborative policy statement to advocate for reduced salt intake, and the Canadian Government has struck a work group to oversee the salt reduction programs.

In the United States, there has been consistent advice to reduce salt intake to less than 6 g/d since the 1980s. However, little action has been taken until recently. In 2005, in a petition to the Food and Drug Administration, the Center for Science in the Public Interest called for tougher regulations on salt.[161] In 2007, the American Medical Association published a report calling for a major reduction in the salt content of processed and restaurant foods.[162] The American Medical Association also pressed the Food and Drug Administration to cease the rule that allows salt and its component sodium to be treated as "generally recognized as safe." In November 2008, Michael Bloomberg, the Mayor of New York City, announced his plan to cut salt levels in processed foods by 20% over the next 5 years.

Several other developed countries, for example, the Netherlands, Ireland, are also stepping up their activities to reduce salt intake. However, many other countries, particularly developing countries where approximately 80% of global BP-related disease burden occurs,[163] have not developed dietary guidelines or strategies to reduce salt intake. It is important that each country determines what its salt intake is and where the major sources of salt are in the diet and then implements a strategic approach to lowering salt intake in the population to the target level. In many developing countries, the major sources of salt consumption are additions during cooking and in sauces, seasonings, pickles, and others rather than prepackaged prepared foods. Public health campaigns are needed to encourage people to use less salt.

The role of the food industry in salt reduction

In most developed countries, approximately 80% of salt we eat is added to foods at the stage of manufacturing,[164]

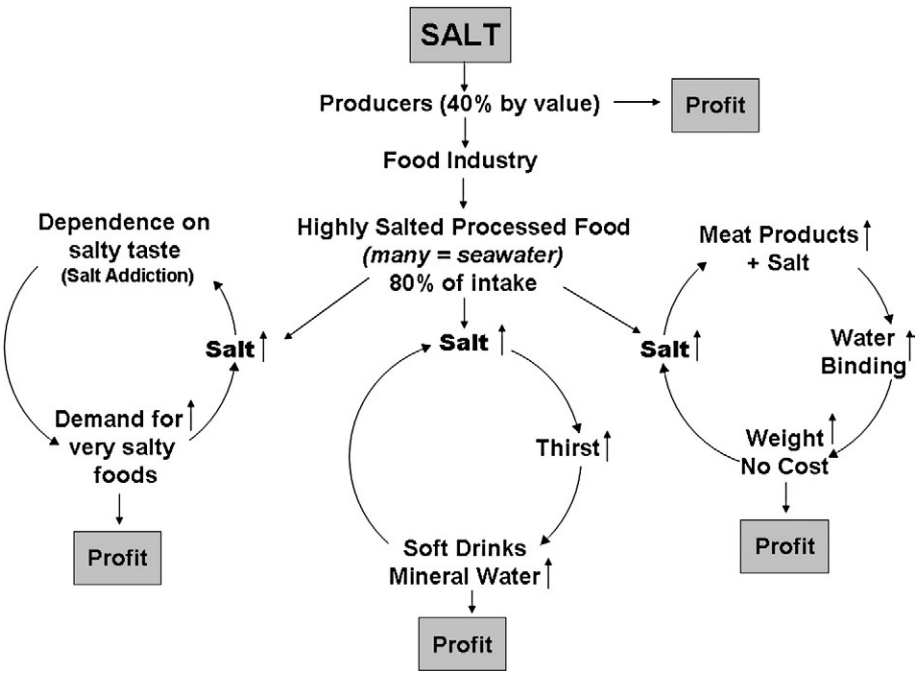

Fig 15. The commercial importance of salt in processed foods.

and the consumers have no say over how much salt is added. Therefore, to achieve a reduction in population salt intake, it is imperative that the food industry reduces the amount of salt they add to all foods. In view of the compelling evidence on the benefits of salt reduction, most food companies recognize that it is time to initiate salt reduction programs and start the process of reformulating their products. However, some members of the food industry are still reluctant to cooperate for commercial reasons (Fig 15). Salt makes cheap, unpalatable food edible at no cost. If highly salted foods are consistently consumed, the salt taste receptors are suppressed and habituation to salty foods occurs, with greater demand for profitable highly salted processed foods. In addition, in meat products, increasing salt concentration, combined with other water-binding chemicals, increases the amount of water that can be bound into the products, so the product weight can be increased by up to 20% with water. Salt is also a major determinant of thirst and any reduction in salt intake will reduce fluid consumption with a subsequent reduction in soft drink and mineral water sales.[12,13] Some of the largest snack companies in the world are part of companies selling soft drinks. It is therefore not surprising that the salt industry and some members of the food industry are very reluctant to see any reduction in salt intake and have been largely responsible for trying to make salt such a controversial issue relative to other dietary changes. Their strategies are identical to the techniques used by the tobacco industry and the tobacco manufacturers association. The commercial reasons for this opposition need to be acknowledged. However, they should not be allowed to stand in the way of a reduction in salt intake as this reduction will be of major benefit to the future health of the whole population, particularly if it is combined with other dietary and lifestyle changes, for example, increasing fruit and vegetable consumption, reducing saturated fat intake, and stopping smoking. The bulk of the food industry has nothing to fear for gradually reducing the very high salt content of many foods they produce. Indeed these lower salt foods are healthier. They lower BP and reduce the risk of CVD, renal disease, stomach cancer, and osteoporosis. The population will therefore live longer and there will be an increase in the number of consumers.

Conclusions

There is now overwhelming evidence for a reduction in salt intake in populations worldwide. Reducing salt from the current intake of 9 to 12 g/d to the recommended level of 5 to 6 g/d will have a major effect on BP and thereby CVD and may have other beneficial effects on health as outlined in this article.

All countries should adopt a coherent and workable strategy to reduce salt intake in the whole population. In most developed countries, approximately 80% of salt is hidden in foods, that is, added by the food industry.[164] It is therefore vital to persuade the food industry to make a gradual and sustained reduction in the amount of salt they add to foods. In many developing countries, where most of the salt consumed comes from salt either added during cooking or comes from sauces, public health campaigns are needed to encourage consumers to use less salt. In both settings, both approaches have important roles to play. In several countries, salt reduction programs have already been carried out successfully and salt intake has fallen.[87,156] Other countries should follow these examples and start taking action now. A modest reduction in population salt intake worldwide would result in a major improvement in public health—similar to the provision of clean water and drains in the late 19th century in Europe.

Statement of Conflict of Interest

All authors declare that there are no conflicts of interest.

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