

Salt and high blood pressure

Sailesh MOHAN and Norm R. C. CAMPBELL

Departments of Medicine and Community Health Sciences, University of Calgary, 3330 Hospital Drive NW, Calgary, Alberta, Canada T2N 4N1

ABSTRACT

HBP (high blood pressure) is the leading risk of death in the world. Unfortunately around the world, blood pressure levels are predicted to become even higher, especially in developing countries. High dietary salt is an important contributor to increased blood pressure. The present review evaluates the association between excess dietary salt intake and the importance of a population-based strategy to lower dietary salt, and also highlights some salt-reduction strategies from selected countries. Evidence from diverse sources spanning animal, epidemiology and human intervention studies demonstrate the association between salt intake and HBP. Furthermore, animal studies indicate that short-term interventions in humans may underestimate the health risks associated with high dietary sodium. Recent intervention studies have found decreases in cardiovascular events following reductions in dietary sodium. Salt intake is high in most countries and, therefore, strategies to lower salt intake could be an effective means to reduce the increasing burden of HBP and the associated cardiovascular disease. Effective collaborative partnerships between governments, the food industry, scientific organizations and healthcare organizations are essential to achieve the WHO (World Health Organization)-recommended population-wide decrease in salt consumption to less than 5 g/day. In the milieu of increasing cardiovascular disease worldwide, particularly in resource-constrained low- and middle-income countries, salt reduction is one of the most cost-effective strategies to combat the epidemic of HBP, associated cardiovascular disease and improve population health.

INTRODUCTION

Cardiovascular disease is the single largest risk for mortality both in developed and developing countries, killing an estimated 17 million people each year [1]. HBP [high BP (blood pressure)] is one of the key cardiovascular disease risk factors accounting for nearly two-thirds of all strokes and a half of all ischaemic heart disease [2]. In addition, it is a major risk for dementia, chronic kidney disease and heart failure [3,4]. The most recent estimate indicates that globally 7.6 million premature deaths (13.5% of total global mortality) and 92 million disability-adjusted life years (6.0% of the global total) were attributable to HBP. Of note, approx. 80% of the HBP-related disease burden occurred in low-

and middle-income countries, and over half occurred in people aged 45–69 years [5]. Furthermore, it has been estimated that 90% of the population aged between 55–65 years with normal BP in developed countries will develop HBP during their lifetime (Figure 1) [6].

Reducing BP with drug therapy is associated with significant decreases in cardiovascular morbidity and mortality; however, it requires extensive healthcare resources and, in clinical practice, most with hypertension are either undiagnosed, untreated or sub-optimally treated [7–9]. Lack of diagnosis and treatment of hypertension is a particular concern for developing countries, where the treatment and control rate is typically much less than 10% [10]. Preventing hypertension through population interventions is a highly attractive cost-effective

Key words: cardiovascular disease, high blood pressure, hypertension, salt, salt reduction, sodium.

Abbreviations: BP, blood pressure; CHD, coronary heart disease; DASH, Dietary Approaches to Stop Hypertension; DBP, diastolic BP; HBP, high BP; SBP, systolic BP; TOHP, Trials of Hypertension Prevention; WHO, World Health Organization.

Correspondence: Dr Norm R. C. Campbell (email ncampbel@ucalgary.ca).

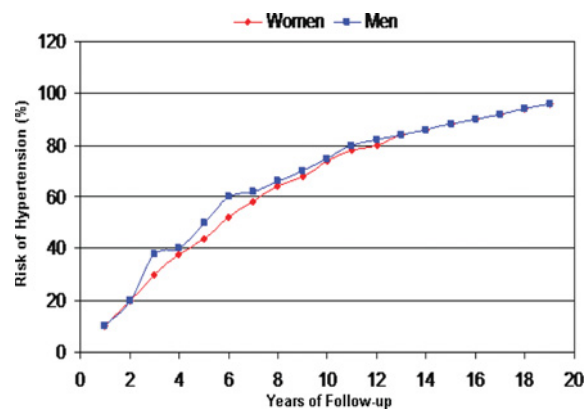


Figure 1 Lifetime risk of developing hypertension in White normotensive women or men aged 65 years

This Figure has been re-drawn with data taken from [6].

companion strategy to programmes to improve the treatment of hypertension [11]. There are a variety of lifestyle changes that are effective in preventing hypertension [12,13].

In the present review, we will assess the evidence regarding the association between excess dietary salt intake and HBP, describe the importance of salt reduction as a population-based strategy to prevent and control hypertension, and highlight some successful examples of salt-reduction strategies from selected countries.

SALT AND HBP

History

Prior to refrigeration, salt was the major mechanism for food preservation and, thus, has played a critical role in the evolution of civilization [14]. Until recently, there was limited access to salt and, therefore, diets typically contained <0.25 g of salt/day. However, with widespread inexpensive commercial access to salt, most diets now have almost 10 g daily. It is likely that the rapid increase in dietary sodium is contributing to the current epidemic of cardiovascular disease [15].

Salt consumption and its sources

In the INTERSALT study of salt consumption and BP, the median intake in 32 countries was 9.9 g/day [16]. Salt consumption varied from 0.1 g/day in Yanamano, Brazil to 15 g/day in Tianjin, China. In developed countries, salt intake is usually between 9–12 g/day [17], and up to 80% of salt comes from processed foods. In Canada, for example, over half of total salt intake is from ten commonly consumed groups of processed foods/beverages (Figure 2). In Asia and other developing nations, most salt is added during cooking or contained in sauce and seasonings [11,18]. Sodium is naturally

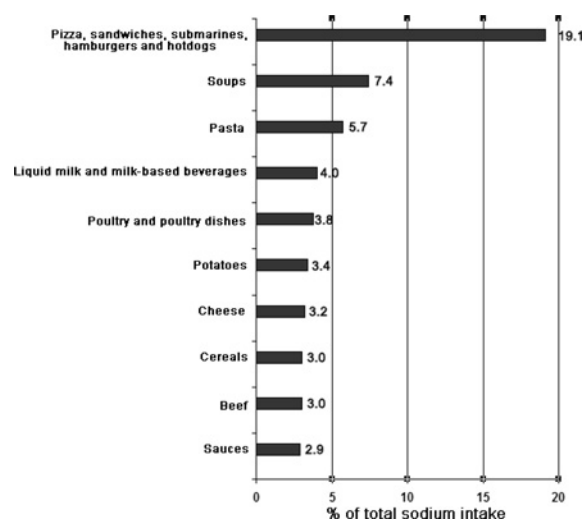


Figure 2 Main sources of sodium intake from processed foods in Canada

Values have been obtained from Blood Pressure Canada.

Table 1 Equivalent amounts of sodium (in mg and mmol) and salt (in g)

A teaspoon of salt contains approx. 6 g of salt.

Sodium (mg)	Sodium (mmol)	Salt (g)
1200	51	3.0
2000	87	5.0
2400	104	6.0
4000	174	10

present in small quantities in unprocessed foods, but is also added to foods either during processing, cooking or at the table. The main reasons for addition of salt in food processing are for flavour, texture, preservation and to increase thirst to sell more beverages [19,20].

Salt and sodium

The main evidence for the association between high intakes of salt and BP relates to sodium. The major source of sodium in the diet is from salt (NaCl). The terms salt and sodium are often used synonymously, although, on a weight basis, salt comprises 40% sodium and 60% chloride; 1 g of sodium is equivalent to 2.55 g of salt; 1 mmol of sodium is equivalent to 23 mg of sodium; and 1 g of salt is equivalent to 17 mmol of sodium [19]. Table 1 provides an overview of the different units. For simplicity, in the present review we have converted all units, including 24 h urinary sodium excretion, into g of salt/day.

Physiology

Sodium is a major cation in the extracellular fluid, with a key role in maintaining fluid balance in the body. The kidneys regulate sodium and water homeostasis,

and the sodium composition is regulated largely by renal excretion and conservation. Sodium is needed for maintaining extracellular fluid, acid–base balance and oncotic pressure, as well as muscle and nerve activity. Furthermore, it helps generate transmembrane gradients which permit the uptake of nutrients by cells of the intestinal mucosa and renal tubules. Most functions of sodium are interdependent with potassium. Although any decrease in extracellular fluid volume, due to falling plasma volume, lowers BP, any rise in extracellular fluid volume increases BP by increasing plasma volume [4,19].

Hypertensive mechanisms

Even though the physiology of salt intake in the development of hypertension is complex, animal studies indicate that BP increases with increasing dietary salt. Multiple species of animals, such as mice, rats, rabbits, dogs, pigs and chimpanzees, have an increase in BP when provided high-sodium diets [21]. The lack of the ability of the human kidneys to fully excrete excess salt is one of the major mechanisms in the association between salt intake and BP [22]. Owing to aging, this excretory capability declines and even small increases in salt intake may increase BP. Some recent studies report that potassium also plays an important role in the development of hypertension [4]. Sodium excess and potassium deficits have an impact on VSMCs (vascular smooth muscle cells), and high-sodium/low-potassium diets can increase BP [4]. Furthermore, a high-potassium diet has been found to blunt the increase in BP caused by high-sodium intake in animal studies [23]. Notably, an increase in potassium intake may also reduce sodium sensitivity in humans [4].

The increases in BP are dose-dependent. Different animal models of hypertension have been developed to be more or less sensitive to dietary sodium. Genetically salt-sensitive rats given a low-salt diet had only a small rise in BP with age in comparison with those on a high-salt diet [24]. In monkeys, chimpanzees and baboons, increases in salt intake in the range consumed by humans caused progressive and very large increases in BP that can be reversed by salt withdrawal [25].

Other animal models demonstrate that not all of the hypertensive response to dietary salt occurs quickly or is reversible [21,25–27]. There can be delays in the increase in BP on exposure to high dietary sodium, indicating that acute testing of ‘sodium sensitivity’ to assess the BP response may not predict long-term increases in BP. Furthermore, increases in BP are not fully reversible in some animal models, indicating the extent of hypertension caused by high dietary sodium may be significantly underestimated by studies that reduce sodium intake in humans [21].

Animal models not only demonstrate that the increases in BP caused by high dietary sodium are strongly associated with adverse cardiovascular events, but also that

high sodium in the diet causes significant direct vascular and cardiac damage independent of BP [21,28–31].

EVIDENCE

Epidemiological investigations

Many large observational epidemiological investigations conducted worldwide link high salt intake and hypertension. In one of the first major global studies on sodium intake (INTERSALT), 24 h urinary sodium was significantly associated with BP as well as the increase in BP with age [16]. Lowering salt intake by 5.8 g was associated with a 3.1 mmHg decrease in SBP (systolic BP) [32]. Furthermore, it also found that populations with low average daily salt intakes had low BP and very little or no increase in BP with age [16]. More recent studies have confirmed these findings and provided more evidence on the salt–BP relationship. INTERMAP (International Study on Micronutrient and Blood Pressure) showed that a lower salt intake and smaller sodium/potassium ratio resulted in lower population BP [33]. Another large study, EPIC-Norfolk (the Norfolk Cohort of the European Prospective Investigation into Cancer) also found sodium to be an important determinant of population BP levels [34]. In the WHO-CARDIAC (World Health Organization Cardiovascular Diseases and Alimentary Comparison) study, among post-menopausal women aged 48–56 years in 17 countries, 24 h sodium excretion was positively associated with BP [35].

A few studies from a single group of investigators have concluded neutral or even reverse associations between salt and cardiovascular disease; however, they had methodological limitations leading to potential bias. Importantly, these studies controlled for BP and hypertension, a major mechanism for sodium-induced harm and, hence, are biased to find a lack of harm associated with high-sodium diets [36–38].

Migration investigations

Studies of population groups that migrated from areas with lower salt intakes to areas with higher salt intake have reported increases in BP. In Kenya, the mean urinary sodium/potassium ratio of migrants was higher than that of non-immigrants, and the immigrants also had higher SBPs [39]. In China, Yi farmers living in remote villages were compared with Yi migrants in the county [40]. The Yi migrants consumed more sodium and less potassium, had lower serum potassium levels and a greater urinary sodium/potassium ratio. Urinary excretion of sodium was greater in Yi migrants than in Yi farmers, suggesting that changes in lifestyle, including dietary changes, contribute importantly to the higher BP among Yi migrants. Notably, BP rose very little with age after puberty in Yi farmers, whereas it increased with age in Yi migrants [40].

Human genetic investigations

Currently known genetic causes of human hypertension and hypotension affect the sodium excretion ability of the kidney. Although these are rare, the genetic causes clearly indicate the importance of salt in regulating human BP and suggest the potential for genetic and acquired variation in renal handling of sodium to cause hypertension [41].

Intervention trials

Results from many clinical trials of salt reduction in humans generally confirm increases in BP with increases in dietary salt. The exception is trials where interventions to lower dietary salt had little effect on dietary sodium, or where the interventions were of very short duration or had a small sample size. Meta-analyses of clinically relevant trials provide strong evidence that salt reduction decreases BP. The largest treatment effect was found in trials lasting more than 4 weeks. Findings from a few of the major trials and meta-analysis are summarized below.

Well-conducted dose–response clinical trials provide the most robust evidence about the impact of salt on human BP. The largest among them, the DASH (Dietary Approaches to Stop Hypertension)-Sodium trial tested the effects of three different sodium intakes (low, intermediate and high) on BP in two separate diets: the DASH diet (rich in fruits, vegetables and low-fat dairy products) and the control diet (Figure 3). Both the type of diet and sodium reduction were effective in lowering BP. Comparing the high- with low-sodium groups, the differences in SBP were 6.7 mmHg among those on the control diet and 3.0 mmHg among those on the DASH diet, with stronger effects among hypertensive participants. The corresponding differences in DBP (diastolic BP) were 3.5 and 1.6 mmHg respectively [42]. Subsequent analyses reported that the effect of sodium reduction remained among clinically relevant subgroups (race, weight and baseline urinary sodium excretion) [43]. Notably, sodium reduction was reported to reduce BP in non-hypertensive subjects on both of the diets. The DASH trial is of particular importance because the food was provided to participants improving adherence to the diet in contrast with many trials where the intervention to reduce dietary sodium was ineffective at reducing dietary sodium.

Findings from the few major clinical trials that lasted 1 or more years and investigated the impact of sodium reduction on BP are summarized below. In Phase I of the TOHP (Trials of Hypertension Prevention), those randomized to the sodium-reduction group had reduced sodium excretion by 2.6 g/day and had a 1.7/0.9 mmHg decrease in SBP/DBP at 18 months [44]. Subsequently, in Phase II of TOHP, the reduction in sodium excretion was 2.4 g/day with a 1.2/0.7 mmHg lowering of SBP/DBP, resulting in an 18% decrease in hypertension incidence at 36 months [45]. In the Hypertension Prevention Trial, which randomized healthy men and women aged 25–

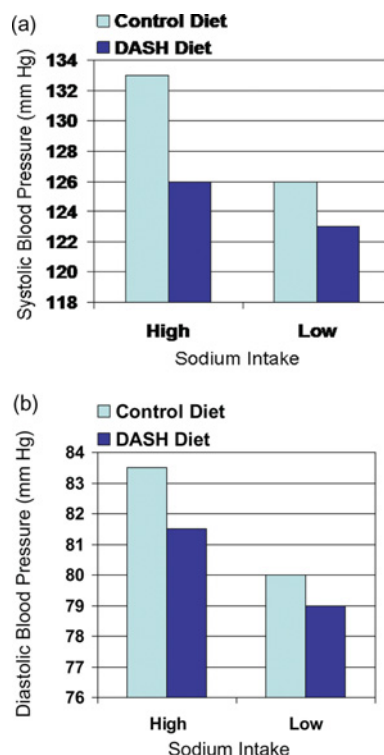


Figure 3 Changes in mean SBP (a) and DBP (b) in the DASH-Sodium trial

The DASH diet is high in fresh fruit, fresh vegetables and low-fat dairy products, and is low in saturated fat. In addition, the DASH study examined high-, intermediate- and low-sodium diets. Reductions in BP were seen with the DASH diet compared with the control diet and with low-dietary sodium as indicated. This Figure has been re-drawn with data taken from [42].

49 years to one of four lifestyle interventions based on dietary counselling treatment, a 13% decrease in sodium excretion at 6 months and a mean SBP decrease of 1.7 mmHg was observed in the sodium-reduction group. These decreases were not observed at 3 years [46]. TONE (Trial of Nonpharmacologic Interventions in the Elderly) randomized 975 elderly men and women on hypertension treatment to weight loss or sodium reduction, with the attempted withdrawal from antihypertensive medication after 3 months of intervention. After 29 months, a 31% reduction in hypertension or cardiovascular event was observed in the sodium-reduction group, and they had a 50% decrease in the return to antihypertensive medication compared with those on usual care [47].

Multiple meta-analyses of salt reduction trials have also been reported (Table 2). Meta-analysis of 40 sodium-reduction trials with a minimum duration of 2 weeks reported that a median salt reduction of 4.4 g/24 h (calculated from 24 h urinary sodium excretion) was associated with a 2.5/1.9 mmHg reduction in SBP/DBP. The average decrease in SBP/DBP among hypertensive subjects was 5.2/3.7 mmHg and 1.3/1.1 mmHg in trials

Table 2 Average salt and BP reduction in selected meta-analysis of salt reduction trials
CI, confidence interval.

Study	Number of participants		Salt reduction (g/day)	BP change in hypertensive subjects (95% CI)		BP change in normotensive subjects (95% CI)	
	HBP	Normal		SBP	DBP	SBP	DBP
Cutler et al. [96]	873	760	4.5	-4.9 (-6.2 to -3.6)	-2.6 (-3.4 to -1.8)	-1.7 (-2.7 to -0.7)	-1.0 (-1.7 to -0.3)
Midgley et al. [51]	1131	2374	5.6-7.3	-3.7 (-5.1 to -2.4)	-0.9 (-1.9 to +0.1)	-1.0 (-1.6 to -0.5)	-0.1 (-0.5 to +0.3)
Cutler et al. [97]	1043	1689	4.5	-4.8 (-5.8 to -3.8)	-2.5 (-3.2 to -1.8)	-1.9 (-2.6 to -1.2)	-1.1 (-1.6 to -0.6)
Graudal et al. [98]	2161	2581	6.9	-3.9 (-4.8 to -3.0)	-1.9 (-2.5 to -1.3)	-1.2 (-1.8 to -0.6)	-0.3 (-0.9 to 0.3)
He and MacGregor [99]	734	2220	4.6	-5.0 (-5.8 to -4.2)	-2.7 (-3.2 to -2.3)	-2.0 (-2.6 to -1.5)	-1.0 (-1.4 to -0.6)
Hooper et al. [50] (at 13-60 months)	1188	2326	2.0-2.4	-1.5 (-12.6 to +9.6)	-7.0 (-12.5 to -1.5)	-1.1 (-1.9 to -0.3)	-0.5 (-1.1 to 0.0)

of normotensive subjects [48]. The most recent Cochrane review included trials (20 in hypertensive subjects and 11 in normotensive subjects) lasting more than 4 weeks with a reduction in salt of at least 2.3 g/day [49]. It found reductions of 5.0/2.7 mmHg in SBP/DBP among hypertensive patients with a median reduction in salt of 4.6 g/day, and 2.0/1.0 mmHg in SBP/DBP among normotensive subjects with a median reduction of 4.4 g of salt/day. Furthermore, a dose-response relationship was observed, with a BP reduction of 7.1/3.9 mmHg among hypertensive patients and 3.6/1.7 mmHg among normotensive participants in SBP/DBP per 6 g decrease in urinary sodium [49]. Another meta-analysis by Hooper et al. [50] reviewed results of three trials in normotensive participants, five in untreated hypertensive patients and three in those being treated for hypertension, with follow-up between 6 months to 7 years. As most of the trials included had relatively ineffective dietary advice interventions, there was a small decrease in sodium intake (2 g/day), SBP (by 1.1 mmHg) and DBP (by 0.6 mmHg). The meta-analysis by Hooper et al. [50] may be most useful in demonstrating that dietary advice on its own is not a very effective strategy for the long-term lowering of dietary sodium. In contrast, an older meta-analysis found that salt reduction had no or very little effect on BP in normotensive individuals [51]; however, the meta-analysis which was funded by a food processor included many trials of very short duration with the median duration of salt reduction of 14 days in the normotensive participants [51]. In addition, many trials were included comparing the effects of acute salt loading to abrupt and severe salt restriction for only a few days. Another meta-analysis that included studies of similar duration and intensity of sodium reduction found increased lipid and glucose levels with sodium reduction [52]. Such large acute changes in salt intake increase sympathetic activity, plasma renin activity and AngII (angiotensin II), counteracting the effects of sodium reduction on BP and are not likely to be relevant to long-term sodium reduction or public health.

Some successful population intervention studies

Population decreases in BP, even if modest, could yield substantial reductions in cardiovascular disease morbidity and mortality. A population-wide 2 mmHg decrease in DBP is estimated to reduce hypertension prevalence by 17%, and the risk of CHD (coronary heart disease) and stroke by 6 and 15% respectively [53]. Another estimate indicated that a 5 mmHg population-wide reduction in SBP would decrease CHD and stroke mortality by 9 and 14% respectively (Figure 4). Many population-based intervention studies have been carried out. Some did not achieve a reduction in salt intake and, consequently, there was no difference in BP [54,55]; however, studies in which salt intake was successfully decreased show

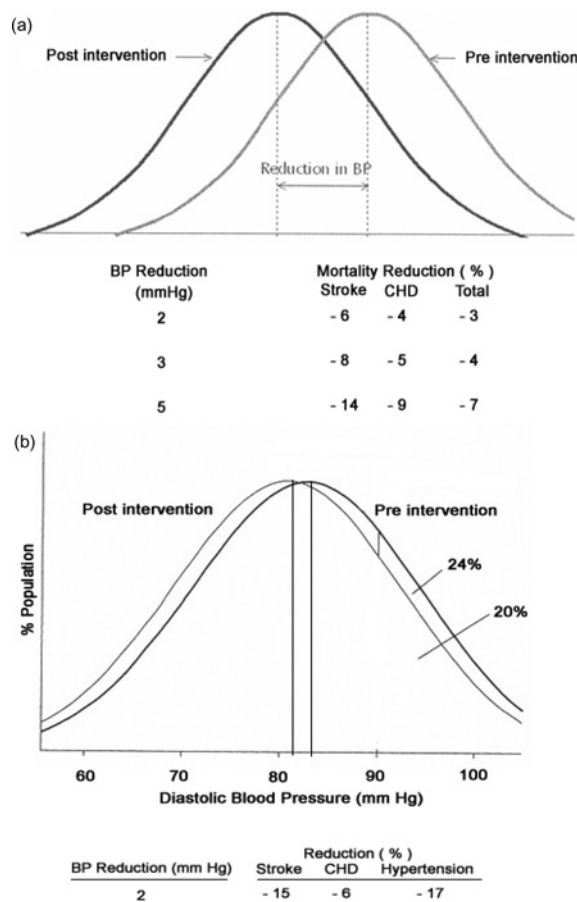


Figure 4 Potential impact of population-wide SBP shifts on mortality and DBP shifts on morbidity

(a) Theoretical reduction in population SBP, and indicates the estimated reduction in stroke, CHD and total mortality with the reduced BP. The figure can be used to estimate the benefits of reducing dietary salt based solely on BP effects. For example, a 4.5 g reduction in dietary salt would decrease SBP by approx. 5 mmHg in hypertensive patients and be estimated to reduced total mortality by 7%. The same reduction in dietary salt would reduce SBP by 2 mmHg in a normotensive population and be expected to reduce total mortality by 3%. Reproduced from [100] with permission. © (2006) Lippincott Williams & Wilkins (<http://lww.com>). Adapted from [95] with permission. © (1991) Lippincott Williams & Wilkins (<http://lww.com>). (b) Theoretical reduction in population DBP, and indicates the estimated reduction in stroke, CHD and hypertension prevalence. Reproduced from *Archives of Internal Medicine*, April 10, volume 155, pp. 701–709 with permission. Copyright © (1995) American Medical Association. All rights reserved.

a reduction in population BP. In Japan, a national campaign reduced salt intake between 1.5–4 g/day, resulting in a population BP reduction and an 80% reduction in stroke mortality notwithstanding an increase in other cardiovascular risk factors [56].

Finland provides one of the best examples of a population-based salt reduction. From the 1970s, it had a population salt-reduction policy based on regulation and education [57]. By 2002, salt intake had decreased by 40%

(Figure 5). Notably, there were remarkable decreases in population BP (>10 mmHg), and stroke and CHD mortality (>70%). The reduction in salt intake was estimated to be a major contributory factor [57]. In a community-based educational programme in China, the mean salt intake was reduced by 1.3 g/day in men and 0.6 g/day in women, and the mean SBP decreased 3 mmHg for the total population and 2 mmHg for normotensive subjects [58]. A very substantial 50% reduction in dietary sodium was achieved in an intervention study in Portugal, such that after 2 years there was a 13/6 mmHg difference in SBP/DBP between the intervention and control communities [59]. An intervention trial among 550 healthy rural Japanese showed that tailored dietary education for 1 year reduced salt intake by 2.3 g/day and lowered SBP by 2.7 mmHg [60].

Similar studies from developing countries are few; however, in one of the first salt reduction trials in Africa, where hypertension and cardiovascular disease is increasing, a community-based cluster randomized trial in Ghana among 1013 participants found that at 6 months the intervention group achieved a 2.5/3.9 mmHg reduction in SBP/DBP [61]. A recent community-based sodium reduction trial in Pakistan reported a significant 6 mmHg reduction in SBP among those with high normal BP [62]. These studies provide evidence of the impact of community-based and context-specific salt-reduction programmes in developing countries where most salt is still discretionary rather than from processed foods.

OTHER HEALTH EFFECTS OF SALT

High intake of salt has also been reported to be associated with non-cardiovascular outcomes, such as higher rates of obesity, stomach cancer, urinary calcium excretion that may lead to osteoporosis and formation of kidney stones, and an increased severity of asthma symptoms [31,63–69]. Although many of these health risks remain unproven in randomized controlled trials, they have biological plausibility and underline the significant safety issues with the addition of high quantities of sodium to food.

OTHER BENEFITS OF SALT REDUCTION

Decreased salt intake not only reduces BP and related cardiovascular disease risk, but has other beneficial cardiovascular effects that are independent of and additive to its effect on BP [70]. It has been reported to have a direct effect reducing stroke [71], left ventricular hypertrophy [72], aortic stiffness [73], and chronic kidney disease and proteinuria [74,75]. For that reason, it is reasonable to infer that the total impact of reducing salt intake on cardiovascular outcomes could be greater than those expected from BP reduction only.

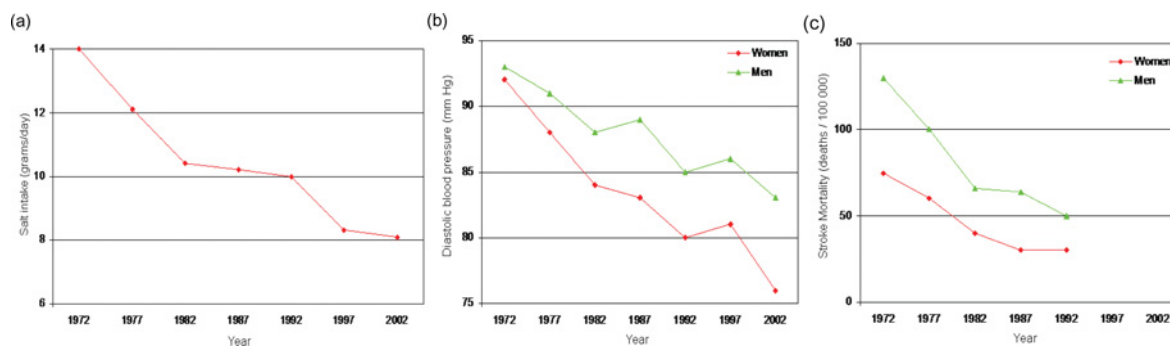


Figure 5 Decline in salt intake, BP and cardiovascular disease mortality in Finland

The observed reduction in dietary sodium (a), DBP (b) and mortality from stroke (c) in men and women. Panel (a) is reprinted from *Progress in Cardiovascular Disease*, vol. 49, Karppanen, H. and Mervaala, E., Sodium intake and hypertension, pp. 59–75, copyright (2006), with permission from Elsevier. Panels (b) and (c) are reprinted by permission from Macmillan Publishers Ltd: *Journal of Human Hypertension* (vol. 19, pp. S10–S19), copyright (2005).

SALT INTAKE AND CARDIOVASCULAR DISEASE MORTALITY

Several observational studies have examined salt intake and cardiovascular disease outcomes; however, large high-quality randomized trials examining morbidity and mortality have not been conducted. A few ecological studies have reported a direct association between higher salt intake or urinary sodium excretion and stroke mortality [76,77]. Prospective studies [78–80], with the exception of three studies undertaken by single group investigators [36–38], also report higher salt intake to predict the incidence of cardiovascular events. In addition, studies from Finland and Japan found an association between dietary sodium and increased risk of CHD or stroke [79,80]. The Finnish study estimated that a 6 g/day increase in salt intake was associated with a 56% increase in CHD deaths, 36% increase in cardiovascular disease deaths and 22% in all deaths [79].

A cluster randomized trial among elderly Taiwanese veterans, which substituted regular salt with potassium-enriched salt, found that the lower-sodium diet was associated with a markedly reduced cardiovascular event rate [81]. In a more recent long-term post-trial follow-up of patients in the TOHP trials, Cook et al. [82] reported a 30% reduction in cardiovascular disease events at 10–15 years in the reduced salt intervention group. These studies provide important evidence, consistent with animal studies, that salt reduction not only lowers BP, but also prevents adverse cardiovascular disease outcomes.

SALT MEASUREMENT AND MONITORING

Assessment of population salt intake is critical in monitoring the effectiveness of salt reduction initiatives. The primary methods are: (i) estimating salt intake by weighing ingested food, (ii) dietary recall, (iii) estimating salt content of food before ingestion, and (iv) measure-

ment by 24 h urinary sodium excretion. However, none of these methods is ideal, and there are several challenges that limit accuracy. The 24 h urinary sodium excretion is considered the gold standard method. However, this and other more reliable methods (method iii) are difficult to implement, whereas simpler ones (method ii) compromise reliability [11,16,83]. Furthermore, there is considerable intra-individual variability in intake such that a single day's measure does not adequately represent usual intake. Current methods when used to assess intake on a single day do not accurately predict usual salt intake on an individual basis. In addition, methods that rely on recall do not adequately quantify salt added while cooking or at the table and, thus, lead to underestimates, rendering them to be of little use in populations where much of the salt is added in cooking or at the table.

GUIDELINES AND PUBLIC HEALTH POLICIES TO REDUCE SALT CONSUMPTION

Rising salt intakes coupled with escalating burden of hypertension and cardiovascular disease globally has prompted the WHO (World Health Organization) to recommend that salt intake be less than 5 g/day [11]. Many nations have developed their own nutritional/dietary guidelines on dietary sodium [84]. The U.K. guidelines recommend salt intake of 6 g/day or less for adults [19,85]. The US Institute of Medicine report set 3.75 g/day salt as an adequate intake, and 5.8 g/day as the upper tolerable intake level for most adults [86]. However, worldwide, most individuals continue to have intakes well in excess of this level. Groups such as the UK Consensus Action on Salt and Health [17] have calculated dose-responses that support a further reduction to 3 g/day, which it claims could achieve a one-third reduction in strokes and a one-quarter reduction in CHD and, thus, having greater population impact in

reducing cardiovascular disease than current guidelines would have (see <http://www.actiononsalt.org.uk/>). Despite these guidelines and the importance to health, most governments have been ineffectual in implementing these recommendations.

The WHO recommends that governments implement policies on food labelling, legislation and product reformulation in collaboration with the food industry [11]. Processed foods are the major source of salt in developed countries therefore collaboration with or regulation of the food industry to reduce salt content is critical. This strategy combined with public education is being employed successfully in the U.K. [87]. Major reductions in the salt content of foods have been achieved without having an impact upon product marketability.

In New Zealand, the National Heart Foundation's 'Pick the Tick' food-labelling programme influenced the food industry to make 23 product changes (in breads, breakfast cereals and margarine) that removed 33 tonnes of salt in a 1-year period [88]. The same programme in Australia resulted in the reformulation and salt reduction in 12 food products [89]. The Health Check programme of the Heart and Stroke Foundation of Canada also works with the food industry to reduce salt in processed foods. In an example of the WHO-recommended national multi-stakeholder involvement to reduce population salt consumption, 17 Canadian health organizations, including the Canadian Hypertension Society, have recently endorsed a national collaborative policy statement to advocate for reduced salt consumption by 2020 (see <http://www.hypertension.ca/bpc/>), and the Canadian Government has struck a work group to oversee the reduction in sodium additives to food and to educate the public on the health risks of high dietary sodium [90].

Regrettably, few developing countries which bear nearly 80% of the HBP-related disease burden have implemented a dietary guideline or a strategy to reduce population salt intake [5]. Furthermore, the food industry in many countries is poorly regulated with very little or no food-content labelling, making informed eating almost impossible. Unlike in developed countries, where the major source of salt consumption is through processed foods, in most developing countries it is through addition during cooking and in sauces/seasonings, emphasizing the importance of public education in these settings. However, in many developing countries, the middle class is rapidly increasing and aggressive marketing is leading to a marked increase in consumption of processed foods. This scenario offers a potential window of opportunity for effectively implementing strong preventive measures. The WHO report in 2006 provides a potential roadmap for governments in developing countries to initiate effective public health action to reduce population salt intake [11]. WASH (World Action on Salt and Health) was formed to assist in the dissemination of information

on the health risks of salt and mechanisms to reduce dietary salt (see <http://www.worldactiononsalt.com/>).

COST EFFECTIVENESS OF SALT REDUCTION

Studies have found that national programmes to reduce dietary salt consumption, including labelling changes and reformulation of products, are very cost effective [91]. Selmer et al. [92] estimated that effective implementation of salt-reduction interventions in Norway could reduce mortality by 1–2%, increase life expectancy and result in a 5% reduction in people requiring antihypertensive medication. This was projected to save \$ 270 million over 25 years. In Canada, where 30% of hypertension is estimated to be associated with excess dietary salt intake, an analysis indicated that reducing salt intake by half would eliminate hypertension in 1 million Canadians, double the number of Canadians with adequately controlled hypertension, and save the healthcare system at least \$430 million a year in hypertension treatment costs alone [93]. Most recently, a global analysis indicated that 8.5 million deaths could be avoided over 10 years (2006–2015) by salt-reducing initiatives alone, and the estimated cost/person of implementing this was reported to be between \$0.04–\$0.32 [94].

CONCLUSIONS

The association between salt intake and HBP is causal, and high dietary salt is estimated to contribute significantly to hypertension on a population basis. Currently, salt intake is far above recommended levels in most countries, resulting in a large and preventable burden of HBP and associated cardiovascular disease. Effective collaborative partnerships between governments, food industry and all other relevant stakeholders have been shown to be effective in reducing dietary salt. Nevertheless there are major challenges to achieve the WHO-recommended population target of less than 5 g/day; however, such a reduction would significantly contribute to shifting the BP distribution downwards in populations and yield substantial reductions in cardiovascular disease morbidity and mortality. In the milieu of increasing cardiovascular disease worldwide, particularly in resource-constrained low- and middle-income countries, salt reduction is one of the most cost effective strategies to combat the epidemic of HBP and cardiovascular disease, and to improve population health.

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